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Case Report

Navigating Cardiovascular Risks: Peripartum Cardiomyopathy and Stroke

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Abstract

Background: Postpartum cardiomyopathy, also known as peripartum cardiomyopathy (PPCM), is a rare cause of stoke in young women. PPCM is defined as new-onset heart failure without an apparent cause between the last month of pregnancy and the fifth month post-partum. Risk factors include multiple parity, advanced maternal age, multiple gestation, preeclampsia, chronic hypertension, smoking, alcoholism, malnutrition, and long standing endometriosis.

Case Presentation: We describe the case of a 34-year- old woman, who was admitted to the hospital three months after the birth of her second child with dyspnea that rapidly progressed to cardiac asthma. One week later, was admitted to the Neurology Department with acute left hemiparesis and speech impairment.

Results: The MRI revealed a wedged-shaped hypodensity affecting the gray and white matter of the right anterior temporal and parietal lobes, a feature suggestive of hemodynamically right- sided acute ischemic stroke.

Conclusion: This case highlights the importance of a collaborative multidisciplinary team clinical approach to further evaluate and appropriately treat patients with PPCM secondary to stroke.

***Keywords:** Peripartum Cardiomyopathy, Women, Stroke, Interprofessional Team Members*

Introduction

Postpartum cardiomyopathy, also known as peripartum cardiomyopathy (PPCM), is defined as new-onset heart failure between the last month of pregnancy and the fifth month after delivery, without an identifiable cause. Postpartum cardiomyopathy is a rare cause of heart failure and stroke in young women. Heart failure in the peripartum period was first described in 1849. The overall incidence of PPCM ranges from 1 in 1,300 to 1 in 15,000 pregnancies. However, the incidence varies worldwide and is higher in developing countries. In 2010, the European Society of Cardiology (ESC) Working Group defined PPCM as an idiopathic cardiomyopathy with the following characteristics: 1. Heart failure (HF) occurs in the third trimester of pregnancy or within 5 months postpartum. 2. Lack of an identifiable cause for HF. 3. Left ventricular (LV) systolic dysfunction with a left ventricular ejection fraction (LVEF) of less than 45%. The LV may or may not be dilated.

The exact mechanism of the disease is unknown. However, various hypotheses regarding its etiology have been described, including viral myocarditis, nutritional deficiencies, autoimmunity, hemodynamic stress, vascular dysfunction, hormonal disorders, and underlying genetics. Alterations in prolactin processing and increased levels of soluble Fms-like tyrosine kinase 1 (Flt 1) have also been implicated in the development of PPCM. Prolactin is a hormone secreted by the pituitary gland in late pregnancy and after birth that stimulates milk production. However, prolactin can have negative effects on the heart muscle, restricting blood supply and causing cell death. Increased oxidative stress during pregnancy causes prolactin to be broken down by cathepsin D into an abnormal 16 kDa protein. This protein damages the heart and blood vessels. Availability Flt-1 is secreted from the placenta, which inhibits vascular endothelial growth factor signaling, leading to angiogenic imbalance and endothelial dysfunction. Relaxin-2, a hormone produced by the ovaries, breasts, and placenta, has a potentially beneficial effect on PPCM. It increases cardiac output and decreases vascular resistance. However, postpartum cardiomyopathy is a diagnosis of exclusion despite numerous attempts to establish its precise etiology and pathophysiology.

Risk factors include increased parity, increased maternal age, smoking, preeclampsia, eclampsia, chronic hypertension, alcoholism, use of tocolytics, and malnutrition.

Patients typically present with dyspnea, orthopnea, cough, hemoptysis, paroxysmal nocturnal dyspnea, and ankle edema. Tachycardia, increased jugular venous pressure, third heart sound (S3), and displaced apical stimulation are common findings. Approximately 6% of patients with PPCM have thromboembolic complications, including deep vein thrombosis, pulmonary embolism, stroke, and acute limb ischemia. Here, we report a rare case of a young woman with peripartum cardiomyopathy secondary to stroke.

Case Presentation

A 34-year-old man, a former smoker, presented to the emergency room 3 months after giving birth to his second child with 40 minutes of slurred speech, difficulty moving the left side of his body, and left hemiparesis. She had been admitted to the pulmonary ward a week earlier with severe fatigue, palpitations, unproductive cough, hypotension (90/50 mmHg), and progressive dyspnea with oxygen saturation of 80%. There was no history of fever, chest pain, or hemoptysis. The family history was positive for premature coronary artery disease and premature death. She reported that her mother and grandmother died of heart disease at the age of 50.

On examination, the patient's temperature was 36.6Â°C, heart rate 123 beats per minute, blood pressure 90/70 mmHg. Art., respiratory rate 22 breaths per minute, and oxygen saturation 93%. There was no peripheral edema. Examination of the chest and other systems revealed no abnormalities. All laboratory tests were normal except for an elevated NTproBNP level (4973.30) during the examination. (See Table 1 for details.)

On neurological examination, the patient had left upper motor nerve facial paralysis, left upper and lower limb strength of 4/5 and 5/5, respectively, according to the Medical Research Council (MRC) scale, and an ipsilateral Babinski sign.

A CT scan of the head and computed tomography angiography (CTA) of the supra-aortic artery were performed immediately, both of which were normal.

MRI showed wedge-shaped hypodensities in the gray and white matter of the right temporal and parietal lobes, features suggestive of a hemodynamically right-sided acute ischemic stroke [Figure 1]. An electrocardiogram showed sinus tachycardia. Transthoracic echocardiography (TTE) showed LVEF 25%, mild mitral regurgitation, and global left ventricular wall hypokinesis with left ventricular dilatation.

She was diagnosed with right ischemic stroke (hemodynamic) with peripartum cardiomyopathy.

The patient was treated with aspirin 100 mg (daily), furosemide 20 mg twice daily, spironolactone 25 mg (daily), metoprolol 12.5 mg, and prophylactic low-molecular-weight heparin (UFH) 4000 units subcutaneously once daily. She was prescribed Entresto (Sacubitil/Valsartan) 50 mg daily with strict monitoring of hemodynamic parameters. At discharge, her speech skills had improved significantly and she had no motor deficits. The patient was advised to avoid subsequent pregnancies. Because the ischemic stroke involved more than one-third of the right middle cerebral artery, anticoagulant therapy was initiated 2 weeks after the onset of the ischemic stroke to avoid the risk of bleeding. Anticoagulation for PPMC is required when LVEF is less than 30%.

Table 1. Laboratory parameters on admission.

Parameters	On admission	Reference range (adults)
Hematocrit (%)	48,4	42-52
Hemoglobin (g/dl)	15,0	13-17
White-cell count (per mm3)	9,5	4-10,5
Differential count (%)		
Neutrophils	52,9	40-72
Eosinophils	2,2	<5
Lymphocytes	35,7	25-45
Monocytes	8,99	3-9
Mean corpuscular volume (fL)	85,8	80-100
Prothrombin time (sec)	12,5	11-14
Creatinine (mg/dl)	0,79	0,72-1,25
Sodium (mmol/liter)	138	136-145
Potassium (mmol/liter)	4,2	3,5-5,1
Random blood sugar (mg/dl)	86	74-100
Urea (mg/dl)	32,7	19,1-44,1
Total bilirubin (mg/dl)	0,35	0,3-1,2
CRP (mg/dl)	0,13	< 0,5
Alanine transaminase (IU/L)	45	< 55
Aspartate transaminase (IU/L)	30	5-34
CK(IU/L)	78	30-200
CK-MB Imuno (ng/ml)	2,8	< 5,2
Troponine I (ng/ml)	0,001	< 0,034
NTproBNP	**4973.30**	**< 125**

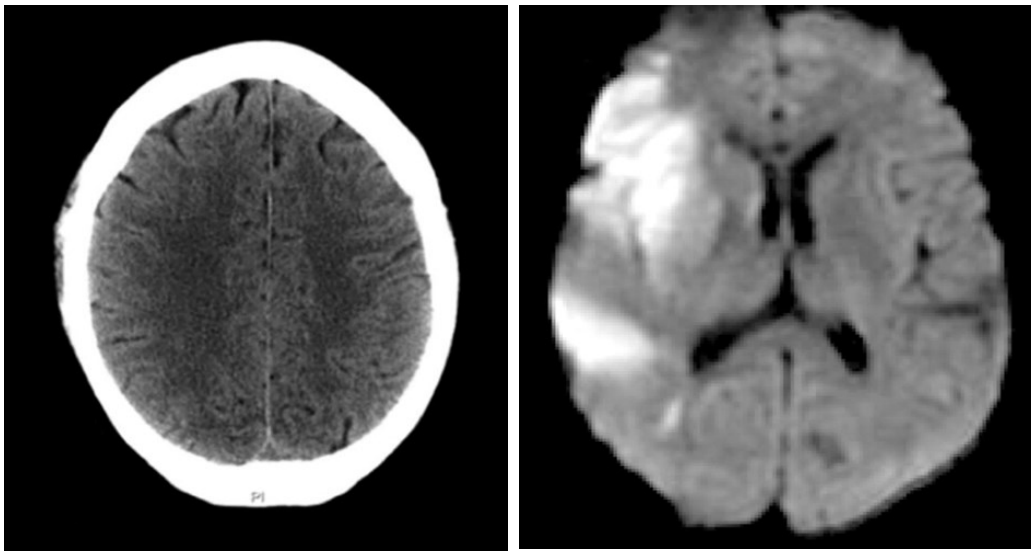

Figure 1. *Normal CT scan and Magnetic Resonance (MRI) showing right side acute ischemic stroke.*

Discussion and Recommendations

Perinatal cardiomyopathy is a rare disease of unknown etiology that affects women and is associated with a high mortality rate. Stroke is rare in young people and is difficult to diagnose and requires attention. In our patient, the etiology was secondary to left ventricular hypokinesia (EF=25%) due to peripartum cardiomyopathy. Maternal age >30 years, smoking, and family history are risk factors for PPCM in this setting. Other generally accepted risk factors for PPCM, such as multiple gestation, use of tocolytics, preeclampsia, or eclampsia, were absent in our patient. Treatment of PPCM due to stroke requires an interdisciplinary approach involving cardiologists, neurologists, gynecologists, psychologists, and physiotherapists.

Ongoing research is needed so that researchers can better understand the causes of PPCM and develop new treatments. Medical professionals have tried treatments that alter the immune system, such as intravenous gamma globulin, but these have not been proven. Researchers have also focused on the role of prolactin in PPCM, as prolactin can adversely affect the heart muscle by restricting blood supply and causing cell death. Bromocriptine is a drug that suppresses the secretion of prolactin from the pituitary gland. Early studies suggest that it may help treat PPCM, but more research is needed.

Conclusion

PPCM is a rare cause of stroke in postpartum patients, so an interprofessional approach to diagnosis and treatment is important. PPCM should be considered in the differential diagnosis of all patients presenting with dyspnea and cough postpartum. Early diagnosis and treatment can prevent further complications.

Conflict of Interest

None declared.

Ethical statement

Authors state that the research was conducted according to ethical standards.

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