POST-PARTUM CARDIOMYOPATHY AND STROKE. CASE REPORT

Malbora Xhelili[1], Ilirjana Zekja[2] , Eliverta Konda-Majko[3], Arben Rroji[4], Jera Kruja[1,2],
[1]UHC "Mother Teresa" , University of Medicine, Faculty of Medicine, Neurology Service, Tirana, Albania
[2]UHC "Mother Teresa", Neurology Service, Tirana, Albania
[3]UHC "Mother Teresa", Neurology Service, Cardiologist
[4]UHC "Mother Teresa", Radiology Service, Albania

Correspondent E-email: neuroalbania@live.com

Abstract

Background. Postpartum cardiomyopathy, also known as peripartum cardiomyopathy, is one of the rare causes of stroke in young females. Postpartum cardiomyopathy is defined as new- onset heart failure between the last month of pregnancy and 5 months post-delivery with no determinable cause. Risk factors include multiparity, advanced maternal age, multiple pregnancies, pre-eclampsia, chronic hypertension, smoking, alcoholism, malnutrition, and long-term tocolysis.
Methods: We describe the case of a 34-year-old patient who was admitted for shortness of breath, rapidly worsening into asthma cardiae three months after the delivery of her second baby. One week later, she presented in the neurology emergency room with an acute onset of left hemiparesis and difficulty speaking.
Results: The Magnetic Resonance Imaging showed a wedge-shaped hypodensity involving the gray and white matter of the right anterior temporal and parietal lobe, features suggestive of hemodynamic right-sided acute ischemic stroke.
Conclusion: This case highlights the importance of multidisciplinary team collaboration in the clinical approach for further evaluation and proper treatment of postpartum cardiomyopathy patients complicated by stroke.

Keywords: peripartum cardiomyopathy, female, stroke, interprofessional team members.

KARDIOMIOPATIA POST-PARTUM DHE INSULTI CEREBRAL

Abstrakt

Hyrje. Kardiomiopatia pas lindjes, e njohur gjithashtu si kardiomiopatia peripartum është një nga shkaqet e rralla të insultit tek femrat e reja. Kardiomiopatia pas lindjes përkufizohet si insuficiencë kardiake me prezantim për herë të parë midis muajit të fundit të shtatzënisë dhe 5 muaj pas lindjes, pa ndonjë shkak të përcaktuar. Faktorët e rrezikut përfshijnë multiparitetin, moshën e përparuar të nënës, shtatzënitë e shumëfishta, pre-eklampsinë, hipertensionin kronik, duhanin, alkoolizmin, kequshqyerjen dhe tokolizën afatgjatë.
Metodat: Përshkruajmë rastin e një pacienteje 34 vjeçare, e cila u shtrua për dispne që përkeqësohej me shpejtësi në astmë kardiake, tre muaj pas lindjes së foshnjës së dytë. Një javë

më vonë ajo u paraqit në Urgjencën e Neurologjisë me fillim akut të hemiparezës së majtë dhe vështirësi në të folur.
Rezultatet: Rezonanca magnetike tregoi një hipodensitet që përfshin lëndën gri dhe të bardhë të lobit anterior të djathtë dhe parietal, tipare që sugjerojnë për lezione akute ishemike hemodinamike në anën e djathtë.
Konkluzionet: Ky rast nxjerr në pah rëndësinë e bashkëpunimit në ekip multidisiplinar, në përqasjen klinike, për vlerësimin e mëtejshëm dhe trajtimin e duhur të pacientëve me kardiomiopati pas lindjes, të komplikuar me insult.

Fialë kyçe: kardiomiopati peripartum, femra, insulti, anëtarë të ekipit interprofesional

Introduction

Postpartum cardiomyopathy, also known as peripartum cardiomyopathy (PPCM), is defined as new-onset heart failure between the last month of pregnancy and 5 months post-delivery with no determinable cause (1). Postpartum cardiomyopathy is a rare cause of heart failure and stroke in young females. Heart failure in the peripartum period was first described in 1849. The overall incidence of PPCM ranges from 1 in 1300 to 1 in 15,000 pregnancies. However, the incidence fluctuates globally and is higher in developing countries. The 2010 European Society of Cardiology (ESC) (2) Working Group defined PPCM as an idiopathic cardiomyopathy with the following characteristics:
1. The development of heart failure (HF) towards the end of pregnancy or within five months following delivery.
2. The absence of an identifiable cause of HF.
3. Left ventricular (LV) systolic dysfunction with an LV ejection fraction (LVEF) of less than 45 percent. The LV may or may not be dilated.
The exact mechanism of disease is unknown; however, different hypotheses have been described regarding its etiology, comprising: viral myocarditis, nutritional deficiencies, autoimmunity, hemodynamic stresses, vascular dysfunction, hormonal insults, and underlying genetics. Altered prolactin processing, and elevated soluble Fms-like tyrosine kinase 1 (Flt 1) (3) have also been associated with the pathogenesis of PPCM. Prolactin is a hormone released from the pituitary gland late in pregnancy and after delivery that stimulates breast milk production. But prolactin may have adverse effects on the heart muscle by limiting its blood supply and causing cell death. During pregnancy, increased oxidative stress leads to cleavage of prolactin by cathepsin D into an abnormal 16-kDa protein. This protein damages the heart and blood vessels. Soluble Flt 1 is secreted by the placenta and inhibits vascular endothelial growth factor signaling, which leads to angiogenic imbalance and endothelial dysfunction (4). Relaxin-2, a hormone produced by the ovaries, breast, and placenta, has a potential beneficial effect in PPCM. It increases cardiac output and decreases vascular resistance. However, postpartum cardiomyopathy is a diagnosis of exclusion, despite many attempts to establish the exact etiology and pathophysiology (5).
Risk factors are increased parity, increased maternal age, smoking, preclampsia, eclampsia, chronic hypertension, alcoholism, use of tocolytics, and malnutrition (6).
Patients usually present with shortness of breath, orthopnea, cough, hemoptysis, paroxysmal nocturnal dyspnea, and ankle edema. Tachycardia, elevated jugular venous pressure, the third heart sound (S3) (7), and a displaced apex beat are common. About 6 % of patients (8) of PPCM

present with thromboembolic complications such as deep vein thrombosis (9), pulmonary thromboembolism, stroke (10), acute limb ischemia (11), etc.
Here, we report a rare case of a young female with peripartum cardiomyopathy complicated by stroke.

Case report

A 34- year-old female ex-smoker patient, three months after giving birth to her second child, presented in the Emergency Room after developing a 40 minute episode with slurring of speech, difficulty moving her left half of the body, and left hemihypoesthesia. She was hospitalized a week prior in the Pulmonary Department with severe fatigue, palpitations, a non-productive cough, low blood pressure (90/50mmHg), and progressive dyspnea with an 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 her grandmother died around the age of 50 from cardiac disease.
On examination, the patient had a temperature of 36.6°C, heart rate of 123 beats per minute, blood pressure was 90/70 mmHg, respiratory rate of 22 breaths per minute and oxygen saturation of 93%. There was no peripheral edema. The chest examination and other systems were unremarkable.
On investigation, laboratoric tests were all normal,except of a high NTproBNP (4973.30). (Details are shown in Table 1.)

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

On neurological examination, the patient had left-sided upper motor neuron type facial nerve palsy, muscle strengths in the left upper and lower limbs were 4/5 and 5/5, respectively, on the Medical Research Council (MRC) scale, and there was an ipsilateral Babinski sign.
A computed tomography scan of the head and a computed tomography angiography (CTA) of the supraaortic arteries were performed immediately and were both normal. The MRI showed a wedge- shaped hypodensity involving the gray and white matter of the right anterior temporal and parietal lobe, features suggestive of hemodynamic right sided acute ischemic stroke. [Figure 1]. An electrocardiogram showed sinus tachycardia.
Transthoracic echocardiography (TTE) showed global hypokinesia of the left ventricular wall with an LVEF of 25%, moderate mitral regurgitation, and left ventricular dilatation.
A diagnosis of right sided ischemic stroke (hemodynamic) with peripartum cardiomyopathy was formulated. The patient was treated with Aspirin 100 mg daily, Furosemide 20 mg twice a day, Spironolactone 25 mg daily, Metoprolol 12,5 mg daily, and prophylactic low molecular weight heparin (UFH) 4000 units subcutaneously once a day. She was initiated Entresto (Sacubitil/Valsartan) 50 mg daily,under rigorous surveillance of the hemodynamic parameters. At the time of discharge, her speaking improved significally and there were no motor deficits. The patient was counseled about avoiding subsequent pregnancies. Anticoagulation was started two weeks following the ischemic stroke to avoid the risk of bleeding, as the infarct involved more than one-third of the right middle cerebral artery region. Anticoagulation in PPMC is administered when LVEF is < 30%.

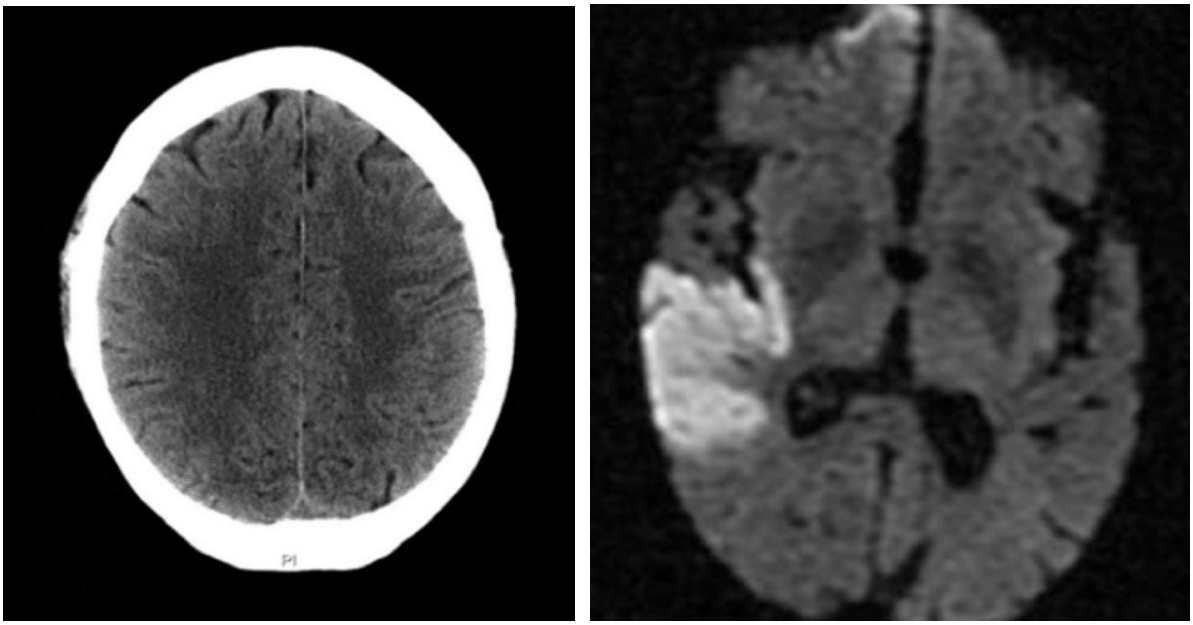

Figure 1. Normal CT scan and Magnetic Resonance (MRI) showing right sided acute ischemic stroke.

Discussion and Recommendations

Peripartum cardiomyopathy is a rare disease of unknown cause that strikes women and is associated with a high mortality rate. Strokes in young adults are uncommon, and the diagnosis is challenging and requires vigilance.
In our patient, the etiology was secondary to hypokinesia of the left ventricle (EF=25%) due to peripartum cardiomyopathy. Maternal age > 30 years,smoking, and family history are the risk factors for PPCM in this case. Other conventional risk factors for PPCM, such as multiple pregnancies, the use of tocolytics, and preeclampsia, or eclampsia were not present in our patient. The management of PPCM complicated by stroke requires a multidisciplinary approach that involves a cardiologist, neurologist, obstetrician, psychologist and physiotherapist.
Ongoing studies are needed to help researchers better understand the cause of PPCM and develop new treatments. Health care professionals have tried treatments that alter the immune system, such as intravenous γ-globulin, but they're not proven. Researchers have also focused on the role of prolactin in PPCM, as it may have adverse effects on the heart muscle by limiting its blood supply and causing cell death. Bromocryptine is a medication that inhibits the pituitary secretion of prolactin. Early studies suggest it helps treat PPCM, but more research is needed.

Conclusion

PPCM is a rare cause of stroke in postpartum patients, and an interprofessional approach is essential in the diagnosis and management. One should consider PPCM as a differential diagnosis in any patient presenting with shortness of breath and cough during puerperium. Early diagnosis and therapy prevents further complications.

Conflict of interest. None declared.

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