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CASE REPORTS

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Peripartum cardiomyopathy: postpartum decompensation and use of non-invasive cardiac output monitoring

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ABSTRACT
The utility of a non-invasive cardiac output monitor (NICOM™) in guiding the peripartum management and identification of postpartum complications in a patient with severe peripartum cardiomyopathy is reported. A 31-year-old nulliparous woman at 35 weeks of gestation presented with a three-week history of worsening dyspnea and progressive functional deterioration. A transthoracic echocardiogram showed severe left ventricular systolic dysfunction with an ejection fraction <20%. Cardiac status was monitored using NICOM™ during labor and delivery. The baseline values were: cardiac output 5.3 L/min, total peripheral resistance 1549 dynes.sec/cm^5, stroke volume 42.1 mL and stroke volume variation 18%. She received early epidural analgesia during labor, titrated slowly with a loading dose of 0.0625% bupivacaine 10 mL and fentanyl 25 μg, followed by patient-controlled epidural analgesia (0.0625% bupivacaine with fentanyl 2 μg/mL, infusion at 10 mL/h, bolus dose 5 mL and lockout interval 10 min). After epidural drug administration, total peripheral resistance decreased, cardiac output increased, and satisfactory analgesia was obtained. She had an uneventful vaginal delivery with a forceps-assisted second stage after prophylactic administration of furosemide 20 mg. NICOM™ was discontinued after delivery. Fifteen hours post-delivery, the patient developed cardiogenic shock, which resolved after aggressive therapy with inotropes and furosemide. NICOM™ can be used to guide treatment during labor and delivery in patients with critical peripartum cardiomyopathy. We suggest that use of NICOM™ be extended into the postpartum period to detect signs of cardiac decompensation in such patients.

Keywords: Peripartum cardiomyopathy; Cardiogenic shock; NICOM™

Introduction

Peripartum cardiomyopathy (PPCM) is characterized by left ventricular systolic dysfunction (left ventricular ejection fraction (LVEF) <45%) typically presenting within the last month of pregnancy and up to five months postpartum, in the absence of previously known heart disease.[1] The incidence of PPCM in the USA is 1 in 2289–4000 live births.[2] Its etiology is unknown, although genetic and environmental factors may play a role.[3] The pathophysiology includes deteriorating systolic function with a reduced LVEF and an increased risk of congestive heart failure, thromboembolism, arrhythmias, and sudden cardiac death.[4] While nearly 50% of patients recover cardiac function within 3–6 months, PPCM may recur in subsequent pregnancies. Overall mortality exceeds 10%.[3]

The purpose of this case report is to demonstrate the use of a non-invasive cardiac output monitor (NICOM™, Cheetah Medical Inc, Portland, OR, USA) as a supplement to clinical examination, and to discuss the management and postpartum complications encountered in a case of PPCM.

Case report

A 31-year-old nulliparous woman at 35 weeks of gestation (92 kg, 157 cm, body mass index 37.4 kg/m^2) presented with a three-week history of worsening dyspnea, orthopnea, paroxysmal nocturnal dyspnea and progressive functional deterioration consistent with New York Heart Association (NYHA) class IV symptoms. She had no significant past medical history. Pregnancy had been complicated in the third trimester by

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well-controlled insulin-dependent gestational diabetes mellitus.

Evaluation of dyspnea included a ventilation/perfusion scan, reported as low risk for pulmonary embolism, and duplex compression ultrasonography of the legs which revealed no evidence of deep vein thrombosis. Subsequently, transthoracic echocardiography (TTE) showed severe global left ventricular systolic dysfunction, with a LVEF of <20%, a left ventricular end diastolic diameter of 6.4 cm (normal <5.7 cm) and a dilated left atrium. Liver enzymes were mildly elevated, and an abdominal ultrasound revealed a diffuse fatty liver with no morphological stigmata of chronic liver disease. She was transferred to the coronary care unit (CCU) where she was normotensive but in frank pulmonary edema. Intravenous furosemide and digoxin and a nitroglycerin patch were given with good initial improvement and hemodynamic stability. The next morning she went into spontaneous labor and was transferred to the delivery unit.

Clinically, she was not in respiratory distress. Examination revealed a blood pressure (BP) of 133/88 mmHg, heart rate (HR) of 126 beats/min, respiratory rate (RR) of 18 breaths/min, and oxygen saturation of 95% on supplemental oxygen 15 L/min via a face mask. Arterial blood gases were unremarkable: pH 7.41, $PaCO_2$ 32 mmHg, PaO_2 111 mmHg, HCO_3 20 mEq/L, base excess −4.6 mmol/L, SaO_2 98%. Airway examination revealed very edematous soft tissues, unrestricted atlanto-axial extension in a short neck, thyromental distance of two finger breadths, and poor mandibular subluxation; her Mallampati score was IV. She was given another intravenous bolus of furosemide 40 mg.

A multidisciplinary plan for early titrated epidural analgesia with an assisted second stage vaginal delivery was made. A repeat TTE was essentially unchanged and ruled out an intra-cardiac thrombus (Appendix A).

An 18-gauge intravenous cannula and a 20-gauge intra-arterial cannula were inserted. A peripherally-inserted central cannula was placed under ultrasound guidance since conventional cannulation was difficult due to edematous tissues. In addition to standard monitoring, NICOM™ was used. Baseline values were: BP 133/88 mmHg (mean arterial pressure 103 mmHg), HR 126 beats/min in sinus rhythm, cardiac output (CO) 5.3 L/min, total peripheral resistance (TPR) 1549 dynes.sec/cm^5, stroke volume (SV) 42.1 mL and stroke volume variation (SVV) 18% (SVV <10% is most likely not responsive to fluids; >12% is most likely responsive to fluids). Although she became increasingly short of breath, her hemodynamic status was unchanged from baseline. A lumbar wedge was used to promote left uterine displacement and limit aortocaval compression. NICOM™ data were recorded at 1-min intervals until the end of her stay on the delivery unit (Figs. 1 and 2).

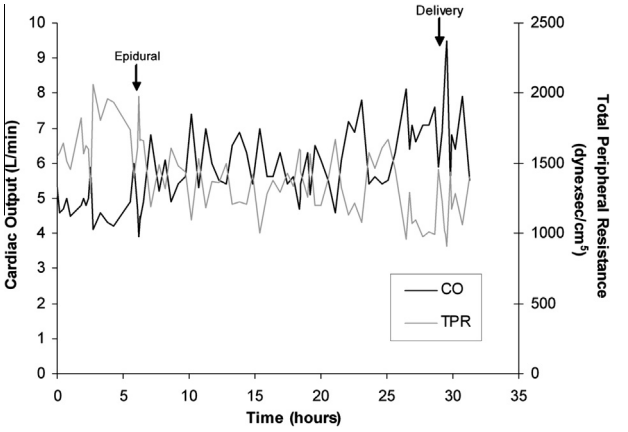

Fig. 1 Cardiac output (CO) and total peripheral resistance (TPR) according to NICOM™ measurements as a function of time during labor. Epidural insertion and delivery are demonstrated by the arrows.

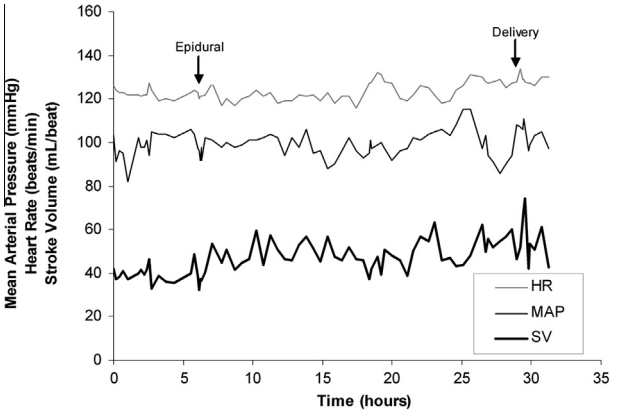

Fig. 2 Mean arterial pressure (MAP), heart rate (HR), and stroke volume (SV) according to NICOM™ measurements as a function of time during labor. Epidural insertion and delivery are demonstrated by the arrows.

The fetus was monitored with continuous cardiotocometry.

An epidural catheter was placed at the L3-4 interspace using ultrasound guidance. Correct placement was tested using the epidural electrical stimulation test, which consists of stimulating the epidural catheter using an electric current at 1–10 mA to elicit a motor response.[5] To limit changes in TPR, a 10 mL loading dose of 0.0625% bupivacaine with fentanyl 25 μg was titrated over 15 min, followed by patient-controlled epidural analgesia using 0.0625% bupivacaine and fentanyl 2 μg/mL, with an infusion rate of 10 mL/h, bolus dose of 5 mL, a lockout interval of 10 min and a 4-hourly limit of 80 mL.

After epidural drug administration, TPR decreased (13.9%), CO increased (13.8%), SV increased (18%),