Update in
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Case Report

Spinal anaesthesia for peripartum cardiomyopathy

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KEY WORDS

- peripartum cardiomyopathy,
- pregnancy,
- spinal anaesthesia,
- women

Summary

Peripartum cardiomyopathy is a rare and life-threatening cardiomyopathy of unknown aetiology that affects women in the last month of pregnancy or in the first five months postpartum. This is a case of a multiparous patient with congestive cardiac failure due to peripartum cardiomyopathy who had spinal anaesthesia for elective caesarean section.

INTRODUCTION

The cardiomyopathies are a group of diseases that affect the heart muscle and are not the result of hypertension or congenital or acquired valvular, coronary, or pericardial abnormalities.[1] Peripartum cardiomyopathy is a form of dilated cardiomyopathy that is defined as deterioration in cardiac function presenting typically between the last month of pregnancy and up to five months postpartum. It is rare but can be fatal.[1] As with other forms of dilated cardiomyopathy, peripartum cardiomyopathy involves systolic dysfunction of the heart, with decreased left ventricular ejection fraction and congestive cardiac failure. There is an increased risk of atrial and ventricular arrhymias, thromboembolism and even sudden death.

CASE REPORT

A 34-year-old booked gravida 3, para 2 Nigerian woman was admitted at a gestational age of 34 weeks with recurrent bilateral pedal oedema, facial puffiness, cough, chest pain, breathlessness, orthopnoea, weakness, fever and headache. Peripartum cardiomyopathy had been diagnosed four weeks previously and she had previously spent three weeks as an in-patient. She had had uncomplicated spinal anaesthesia one year ago for emergency caesarean section.

On examination she was 105kg and had bilateral pitting oedema up to the knee joints. Her pulse was 100 beats per minute, respiratory rate 24 per minute and her blood pressure 140/90mmHg. Auscultation of her chest revealed a third heart sound with gallop rhythm and examination of her abdomen showed non-tender hepatomegaly of 8cm.

Her laboratory results showed a packed cell volume of 37%. Urinalysis showed one plus of proteinuria. Results of serum electrolytes, creatinine and liver function tests were within normal values. An electrocardiogram showed atrial fibrillation with left axis deviation. Echocardiography showed normal cardiac chambers with posterior wall thickening in both ventricles. A diagnosis of congestive cardiac failure secondary to peripartum cardiomyopathy was made for which she was placed on atenolol and a low salt diet.

Elective caesarean section was planned at 36 weeks gestational age, after treatment of her heart failure had improved her breathlessness and orthopnoea. Spinal anaesthesia was explained to the patient and two units of blood were made available for the surgery.

In the operating room intravenous access was established. The patient was preloaded with 500ml 0.9% saline. Spinal anaesthesia was established using 1.5ml plain bupivacaine and 20mcg fentanyl. A wedge was placed under her left side. Intraoperative monitoring consisted of pulse oximetry, non-invasive blood pressure, electrocardiography, and measurement of blood loss and urine output. Intravenous ephedrine 6mg was administered at one and ten minutes after administration of spinal anaesthesia to correct hypotension.

A live female baby weighing 3.2kg, with Apgar scores of 6 at one minute and 8 at five minutes, was delivered five minutes after commencement of surgery. Immediately after delivery of the baby, oxytocin 10 IU was administered as a slow intravenous injection and 20 IU infused slowly in 500ml 0.9% saline. Estimated blood loss at the end of surgery was 500ml. She was discharged home on the ninth postoperative day to continue management by the cardiologist.

DISCUSSION

Normal pregnancy is characterised by an increase in cardiac output, a reduction in systemic vascular resistance and a modest decline in mean blood pressure. Identified risk factors for peripartum cardiomyopathy include advanced maternal age, multiparity, obesity, multiple gestation and black race.[2] It can occur without these risk factors and is recognised in women in their first pregnancy.[3]

The higher incidence of peripartum cardiomyopathy is also seen in developing countries and may be due to variations in local, cultural and puerperal practices, in

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addition to aetiological factors, environmental influence, diagnosis and the reporting pattern used.[3] The reported incidence of peripartum cardiomyopathy in non-African countries ranges from 1 in 3000 to 1 in 15000 live births. In South Africa, the reported incidence is higher (1 in 1000 live births). Higher incidences been reported from Haiti (1 in 300 live births have) and in Nigeria (1%), where it is particularly common among the Hausa and Fulani tribes.[4]

Patients with peripartum cardiomyopathy present with typical signs and symptoms of ventricular failure. Symptoms usually include one or more of the following: orthopnoea (breathlessness on lying flat), dyspnoea, swollen ankles, cough, palpitations and chest pain.[5] However, when the disease develops during the last month of pregnancy, the diagnosis of cardiac failure is difficult to make by signs and symptoms alone, since some of these symptoms such as fatigue and pedal oedema are common among normal parturients during late pregnancy. Further testing is required to establish the presence of cardiac failure. Peripartum cardiomyopathy is a disease of exclusion, wherein patients have no prior history of heart disease and there are no other known possible causes of heart failure. A high index of suspicion of peripartum cardiomyopathy is required for its diagnosis and the condition should be considered in any parturient with unexplained symptoms. Its spectrum may extend beyond the peripartum period.

Where available, electrocardiography (ECG) and echocardiography should be obtained in any woman suspected of having peripartum cardiomyopathy. However, the ECG may be normal. Echocardiography is used to both diagnose and monitor the effectiveness of treatment of peripartum cardiomyopathy.[6] Echocardiography is essential in identifying chamber enlargement and in quantifying left ventricular and valvular function. Peripartum cardiomyopathy is diagnosed by clinical examination in settings where echocardiography is not available. It should be suspected in patients who develop symptoms and signs of heart failure in the last month of pregnancy or within five months of delivery.[7]

Treatment of peripartum cardiomyopathy is similar to that for other types of congestive heart failure. Careful attention must be paid to foetal safety and to excretion of drug or drug metabolites during breastfeeding after delivery. A non-medication regimen, including restriction of daily salt and water intake, is very important particularly in women with symptoms and signs of heart failure. In general, the goal is to reduce the amount of volume returning to the heart (preload reduction), decrease the resistance against which the heart must pump (afterload reduction) and increase the contraction force of the heart (inotrophy).[2]

Appropriate drug treatments for heart failure include diuretics to control volume overload (preload). Consider addition of a beta-blocker after signs and symptoms of heart failure have begun to improve, as they improve symptoms, ejection fraction, and survival. Other treatment options during pregnancy include hydrallazine and nitrates.[8] Pregnant women should not receive angiotensin converting enzyme inhibitors, angiotensin receptor blockers or warfarin because of potential teratogenic effects. The teratogenic effects occur particularly in the second and third trimester with foetopathy characterized by foetal hypotension, oligohydramnios, anuria, and renal tubular dysplasia. After delivery, the treatment is identical to that for non-pregnant women with dilated cardiomyopathy. Angiotensin converting enzyme (ACE-) inhibitors are the mainstay of treatment.

Complications of peripartum cardiomyopathy include thromboembolism, arrhythmias and organ failure. Obstetric and perinatal complications include miscarriage, premature delivery, small for dates baby, intrauterine growth retardation, foetal death and congenital malformations.[9] Congestive cardiac failure is associated with higher infant mortality.[9]

Its prognosis is related to the recovery of ventricular function. Failure of the heart size to return to normal after delivery is associated with excess morbidity and mortality. The risk of developing peripartum cardiomyopathy in subsequent pregnancies remains high, especially if left ventricular dysfunction persists. Multiparity increases the risk of irreversible cardiac damage in subsequent pregnancies. Recurrence of heart failure ranges between 21 and 80% in subsequent pregnancies.[9]

Management of labour and delivery

The cardiovascular stress of labour and delivery may lead to cardiac decompensation. A parturient with peripartum cardiomyopathy requires special anaesthetic care during labour and delivery. The anaesthesiologist plays a vital role in managing these patients in a high-dependancy area of the hospital, providing labour analgesia, optimising the medical condition of these mothers for caesarean section and administering anaesthetia for urgent or elective caesarean section.[9] Anaesthetic management for caesarean section in peripartum cardiomyopathy patients can be a challenge. Both general and regional anaesthesia have been used. Anaesthetic drugs with myocardial depressant effects should be used with caution. Where vasopressor and beta-adrenergic agonists are not available, ketamine offers better cardiac stability. However the sympathomimetic effects of ketamine cause tachycardia and increased afterload that may cause haemodynamic deterioration in patients with severe cardiomyopathy.

Where available, all peripartum cardiomyopathy patients should be managed in an intensive care unit or high care area as they are prone to develop left ventricular failure and pulmonary oedema in this period, requiring strict fluid management.[9]

Regional analgesia reduces the cardiac stress of labour pain. Peripartum cardiomyopathy during the antepartum period demands intensive foetal and maternal monitoring. A multidisciplinary approach involving an obstetrician, cardiologist, anaesthesiologist and perinatolgist may be required to provide optimal care to such patients.

Once peripartum cardiomyopathy is identified, the goal of therapy is to alleviate symptoms of congestive heart failure. As mentioned, diuretics and sodium restriction can be used for preload reduction. The patient's haemodynamic status is carefully followed and fluid management is guided by data from invasive monitors. Regional techniques are safer for labour analgesia as well as anaesthesia. Invasive monitoring is recommended in severe cases.

Patients with peripartum cardiomyopathy require counselling concerning the risks of subsequent pregnancies. Collaboration among the obstetrician, cardiologist, and anaesthesiologist is essential to optimise care.

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