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Case Note

Peripartum cardiomyopathy undergoing caesarean section under epidural anaesthesia

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Abstract
Purpose: To report a case of peripartum dilated cardiomyopathy presenting for emergency caesarean section, this was successfully managed with Epidural Anaesthesia.
Clinical features: A parturient suffering from idiopathic peripartum cardiomyopathy (E.F. 18%) was brought for an emergency caesarean section. Epidural anaesthesia was performed and 2% Lignocaine with adrenaline total 13ml was injected into the epidural space. The patient's haemodynamic status was monitored with NIBP, ECG, pulse oximetry. Patient's perioperative course was uneventful.
Conclusion: In patients suffering from peripartum cardiomyopathy, undergoing caesarean section epidural anaesthesia is an acceptable anaesthetic alternative.

Key words: Cardiomyopathy, epidural, caesarean section, anaesthesia

Peripartum cardiomyopathy occurs in approximately 1/10,000 deliveries[1] and can result in severe ventricular dysfunction during late pregnancy or early puerperium[2]. We present a patient with peripartum cardiomyopathy requiring caesarean section that was managed with epidural anaesthesia.

Case report

A 26-yr-old primigravida at 33 wks gestation, with a Mallampatti class II airway was on regular antenatal check up. This time presented with history of shortness of breath and pedal oedema of 10 days. On examination she was dyspnoeic on lying down, her respiratory rate was 52/min, pulse rate 92/min, blood pressure 100/70 mmHg and JVP was raised.

Her investigations showed TC 12,100, polymorphs 80%, lymphocytes 18%, Haemoglobin 14.2%, RBS 104mg/dl, sodium 141meq/l, potassium 4meq/l, urea 40mg/dl, creatinine1mgldl and platelets 160000 with INR 1.

Echocardiography revealed global hypokinesia, with EF 18%, moderate MR, mild TR. Patient was thus diagnosed as a case of peripartum cardiomyopathy. She was treated with tab. Lanoxin 0.25mg BD, inj. Lasix 20mg IV BD and was planned for emergency LSCS.

On arrival in the operating room, her BP was 140/100 mm Hg. Pulse 103/min, SpO2 98%. On auscultation, chest was B/L clear. There was systolic murmur heard over mitral area. Peripheral venous cannulation was done with 18G IV cannula and inj. Ringers lactate infusion was started. ECG, pulse oximetry, NIBP were attached for continuous monitoring. Central venous cannulation was done into right internal jugular vein. Epidural catheter was inserted at L3-L4 interspaces in sitting position. Catheter was fixed at 10cm. Correct placement was confirmed by injecting 2% lignocaine with adrenaline 3ml as a test dose. Later, 2% lignocaine with adrenaline 5ml was injected epidurally. After 5 min, another 5ml of the drug was administered epidurally. Sensory level up to T7 was achieved. Following 10 min of epidural analgesia blood pressure dropped down to 80/60 mmHg and Inj Dobutamin started at the rate of 7µg/kg/min. Her blood pressure then remained maintained at 100-130mmHg systolic and 60-90 mmHg diastolic through out the surgery. Male baby 1.6 kg was delivered with Apgar score of 10/10. A total of 1000ml of Ringer's lactate and 500ml of normal saline was administered intraoperatively.

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Patient was then transferred to the ICU. ECHO was performed a week after delivery which revealed an Ejection Fraction of 35%. She was discharged after 10 days and at the time of discharge she was haemodynamically stable.

Discussion

Peripartum cardiomyopathy is a relatively rare form of acute heart failure associated with pregnancy[3]. It was recognized first in the 19th century by Ritchie[4] and is defined as the onset of acute heart failure in the last trimester or early postpartum period in the absence of infectious, metabolic, toxic, ischemic or valvular causes of myocardial dysfunction[5]. The diagnosis presents a challenge as many parturient in the final month of pregnancy experience dyspnoea, fatigue and peripheral oedema.

Demakis in 1971 established the criteria of peripartum cardiomyopathy[6] which includes

1. Development of heart failure in the last month of pregnancy or within the first five postpartum months.
2. Absence of a determinable aetiology and
3. Absence of demonstrable heart disease before the last month of pregnancy.

More recently it has been suggested that echocardiographically demonstrable impairment in LV function should be added as a fourth criteria[7].

Sixty percent of cases usually present with peripartum cardiomyopathy within the first 2 months postpartum but up to 7% may present in the last trimester of pregnancy[2,6]. Geographical variations exist with a higher incidence reported in areas of Africa because of malnutrition and local customs in the puerperium[8,9]. Predisposing factors include maternal age greater than 30yr, multiparous or eclamptic patients, twinning, racial origin (black), hypertension and nutritional deficiencies[6]. In the majority of cases there is no family history. The mortality rate of peripartum cardiomyopathy is 30-60% and may be caused by severe pulmonary congestion, and or thromboembolic events[2, 10].

Survivors have a 50-80% risk of developing cardiac failure during future pregnancies with an associated mortality rate of 60%[11]. Peripartum cardiomyopathy usually presents with symptoms of worsening cardiac failure. These include dyspnoea on exertion, fatigue, ankle oedema, embolic phenomena, atypical chest pain and haemoptysis. Examination may reveal evidence of a raised CVP, tachycardia, cardiomegaly with a gallop rhythm (S3), mitral regurgitation, pulmonary crackles and peripheral oedema. Chest radiographs may show cardiomegaly with pulmonary oedema and pulmonary venous congestion. The electrocardiogram may show non specific ST and T wave changes, atrial or ventricular arrhythmias and conduction defects. Echocardiographic changes include dilatation of the left atrium and ventricle with global hypokinesia[2,3,12].

Treatment of this condition is as for other forms of congestive cardiac failure[3]. This includes bed rest, diuretics, vasodilators, ACE inhibitors and anticoagulation to counter the risk of endocardial clot formation. Digoxin is often added to the above regimen.

The clinical course and outcome of this disease appears to be variable. Prognosis depends on the degree of cardiomegaly at presentation. There appears to be an initial high risk period with a mortality rate of 25-50% in the first 3 months post partum.

In this case termination of pregnancy was planned through caesarean section as the mother was having progressive dyspnoea. With good care both the neonate and the parturient survived the operation without any morbidity.

The optimum anaesthetic technique for patients undergoing caesarean section with peripartum or other forms of congestive cardiomyopathy is controversial and both general anesthesia[13,14] and regional anesthesia[15,16] have been described.

Brown et al described the use of general anaesthesia because they feared catastrophic effects of reduction in systemic vascular resistance caused by epidural blockade[13]. Where as Mellor and Bo Denham considered that both the methods of general anaesthesia and epidural anaesthesia were dangerous and described the use of infiltration anaesthesia supplemented with bilateral ilioinguinal nerve block[17.]

The goals during the management of anaesthesia in patients with cardiomyopathy include

- Avoidance of drug induced myocardial depression.
- Maintenance of normovolaemia and
- Prevention of increased ventricular afterload.

Epidural anaesthesia produces changes in preload and afterload that mimic pharmacologic goals in the treatment of cardiomyopathy. Caution is indicated to

avoid sudden onset of blockade of sympathetic nervous system.

General anaesthesia may be needed for urgent caesarean section[10]. Performing a rapid sequence induction during general anaesthesia on a patient with compromised cardiac function can be very challenging.

Carefully administered regional anaesthesia avoids stress of general anaesthesia. The vasodilatation caused by regional anaesthesia is beneficial in left ventricular dysfunction[18]

In this case we preferred epidural anaesthesia as slowly titrated epidural anaesthesia avoids the use of cardiodepresant drugs and improves myocardial performance by reducing left ventricular afterload. The advantage of epidural anaesthesia over spinal anaesthesia is that it prevents sudden and rapid reductions in systemic vascular resistance and thereby preload, which might be disastrous in low cardiac output condition.

Conclusion

In patients suffering from peripartum cardiomyopathy, undergoing caesarean section, epidural anaesthesia is an acceptable anaesthetic alternative.

References

1. Thornhill ML, Camann WR. Cardiovascular disease. In: Chestnut DH (Ed.). Obstetric Anaesthesia. St. Louis: Mosby, 1994:765.
2. Homans DC. Current concepts. Peripartum cardiomyopathy. N Engl J Med 1985; 312: 1432- 7[Medline]
3. Morrison WL, Petch MC. Peripartum Cardiomyopathy. Hospital update 1991;17 : 693- 698
4. Ritchie C. Clinical contributions to the diagnosis and treatment of certain chronic diseases of the heart. Medical Surgery Journal 1849; 12:333
5. Report of the WHO/ISFC task force on the definition & classification of cardiomyopathies. British Heart Journal 1980; 44:672- 673
6. Demakis JG, Rahimtoola SH. Peripartum Cardiomyopathy. Circulation 1971; 44 964-968
7. Lampert MB, Lang RM. Peripartum Cardiomyopathy AM Heart J 1995; 130: 860-70
8. Davidson NM, Passy EHO Peripartum cardiac failure. Quarternary Journal of Medicine 1978; 47:431- 461
9. Davidson NM, Passy EHO. The Etiology of Peripartum Cardiac failure. American Heart Journal 1979; 97: 535 – 536
10. Chan F, Kee WDN. Idiopathic dilated cardiomyopathy presenting in pregnancy. Can J Anesth 1999; 46: 1146 – 9
11. Veille J-C. Peripartum cardiomyopathies: a review. Am J Obstet Gynecol 1984; 148: 805 – 18
12. Malinow AM, Butterworth JF, Johnson MD, Safon L, Rein M, Hartwell B, Datta S, Lind L, Ostheimer GW. Postpartum cardiomyopathy at caesarean delivery anesthesiology 1985; 63: 545- 547
13. Brown G, O' Leary M, Douglas L, Herkes R. Perioperative management of a severe peripartum cardiomyopathy. Anaesthesia Intensive care 1992; 20: 80-3
14. Lavies NG, Turner DAB. Peripartum Cardiomyopathy. A rare cause of pulmonary oedema in late pregnancy. Anaesthesia 1989; 44: 770-2
15. George LM, Gatt SP, Lowe S. Peripartum Cardiomyopathy: four case histories and a commentary on anaesthetic management. Anaesthetic Intensive Care 1997; 25: 292-6
16. Gambling DR, Flanagan ML, Huckell VF, Lucas SB, Kim J HK. Anaesthetic management and non invasive monitoring for caesarean section in a patient with cardiomyopathy. Can J Anaesthesia 1987; 34:505-8
17. Mellor DJ, Bo Denham A. Infiltration anaesthesia in the management of caesarean section in a patient with peripartum cardiomyopathy Anaesthesia 1996; 51: 409.
18. Sharrock NE, Bading B, Mineo R, Blumenfeld JD. Deliberate hypotensive epidural anaesthesia for patients with normal and low cardiac output. Anesth Analg 1994; 79:899-904.