Use of remifentanil in a patient with peripartum cardiomyopathy requiring Caesarean section


Department of Clinical Anaesthesia, Royal Victoria Hospital, Grosvenor Road, Belfast BT12 6BA, UK
*Corresponding author

We describe a case of a 26 yr old primigravida at 39 weeks' gestation, with a diagnosis of peripartum cardiomyopathy, requiring urgent Caesarean section. The patient presented in severe heart failure and active labour. A general anaesthetic, using a target-controlled infusion of propofol and an intravenous infusion of remifentanil, was used to provide stable anaesthesia and analgesia for a successful delivery. The unusual diagnosis of peripartum cardiomyopathy and the potential benefits of the use of remifentanil in high-risk obstetric surgery are discussed.

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Peripartum cardiomyopathy is an uncommon cause of cardiac failure in obstetric patients. The management of such patients requires assessment of cardiac status and careful planning for the provision of safe analgesia and/or anaesthesia for delivery. Remifentanil is increasingly used as an anaesthetic agent providing cardiovascular stability in high-risk patients.1 We report the management of a patient presenting in active labour with congestive cardiac failure due to peripartum cardiomyopathy and describe the successful use of remifentanil and propofol in the anaesthetic technique for her subsequent Caesarean section.

Case report

A previously well 26 yr old, 70 kg primigravida presented in labour for urgent Caesarean section at 39+6 weeks' gestation, complaining of mild shortness of breath, nausea, vomiting and increasing tiredness over the previous 2 weeks. On examination, she was dyspnoeic, with a pulse rate of 130 beats min⁻¹ and arterial pressure of 90/60 mm Hg and had fine inspiratory crepitations in the mid-zones on chest auscultation. Initial management consisted of routine monitoring of maternal pulse, arterial pressure, oxygen saturation and temperature as well as fetal observations, with oxygen (35%) administered via a face mask. As symptoms were mild at this time, no further intervention other than close observation was undertaken and no firm diagnosis was made.

Over the next few hours, the patient's shortness of breath became worse and she developed a dry cough. An electrocardiogram (ECG) revealed left bundle branch block (LBBB) and a transthoracic echocardiogram demonstrated a markedly dilated left ventricle with an end-systolic diameter of 68 mm (normal=18-40 mm), global left ventricular dysfunction with an ejection fraction of 15%, mild functional mitral regurgitation and an enlarged left atrium. A chest x-ray showed cardiomegaly and signs of pulmonary oedema.

In view of the acute onset of symptoms over only a 2 week period with no previous history of heart disease and the echocardiographic findings of impaired left ventricular function without evidence of right ventricular strain, an initial diagnosis of peripartum cardiomyopathy with heart failure was made. It was felt unlikely that embolic disease or pre-existing heart disease was an aetiological factor in her deterioration. The patient was transferred to the regional cardiology unit (30 h after initial admission) for management of her congestive cardiac failure. Here, initial treatment consisted of oxygen, diuretics and digoxin as well as infusions of glyceryl trinitrate, dopamine and hydralazine. However, shortly after transfer, the patient began to experience contractions and vaginal examination showed that her cervix was 2 cm dilated. Early active labour was diagnosed and the patient was commenced on oral ranitidine 150 mg every 8 h.

Both the cardiac and obstetric anaesthetic on-call teams were contacted. A multidisciplinary decision was made that urgent Caesarean section under general anaesthetic would be the most appropriate method of delivery in view of the patient's haemodynamic status. Continuous fetal cardiotocograph monitoring demonstrated no fetal distress.

Preoperative monitoring was instituted, including invasive monitoring of arterial pressure via a radial arterial cannula, as well as measurement of pulmonary artery
pressures, continuous cardiac output, mixed venous saturation and central venous pressure (CVP) via a pulmonary artery flotation catheter (PAFC). During PAFC placement, the patient developed complete heart block with a ventricular rate of 70 beats min\(^{-1}\). This change in heart rhythm did not significantly alter the patient’s haemodynamic parameters or symptoms. The PAFC was removed and several attempts at transvenous pacing were made unsuccessfully. The patient spontaneously reverted to sinus rhythm with LBBD within 3–5 min. Before PAFC removal, right ventricular pressures of 50/16 mm Hg and pulmonary artery pressures of 48/24 mm Hg were recorded.

After oral administration of 30 ml of 0.3 M sodium citrate for antacid prophylaxis, preoxygenation and application of cricoid pressure, anaesthesia was induced in a semi-erect position using a target-controlled infusion (TCI) of propofol (target blood concentration 1.5 \(\mu\)g ml\(^{-1}\)), and an infusion of remifentanil at 2 \(\mu\)g kg\(^{-1}\) min\(^{-1}\). Rocuronium 70 mg was administered after loss of verbal response to achieve muscle relaxation. The patient was intubated 90 s later with a 7.5 mm internal diameter endotracheal tube following grade 1 view on laryngoscopy.

Maintenance of anaesthesia was achieved by continuation of the TCI propofol (target plasma concentration range 1.0–2.5 \(\mu\)g ml\(^{-1}\)) and remifentanil infusions. After 5 min the remifentanil infusion rate was reduced to 1.2 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) for 28 min before being reduced again to 0.8 \(\mu\)g kg\(^{-1}\) min\(^{-1}\). The patient was ventilated with an air/oxygen mixture (\(F_{\text{O}_2}\) 0.5). Once the airway had been secured, a transoesophageal echocardiography (TOE) probe was passed in order to assess valvular and chamber function. There was no significant change from the preoperative transthoracic echocardiogram findings, revealing a dilated left atrium and ventricle in association with poor left ventricular systolic function and moderate mitral regurgitation.

During the perioperative period, the patient remained haemodynamically stable, her heart rate varying between 120 and 140 beats min\(^{-1}\) and her systolic and diastolic arterial pressures varying between 120 and 140 and between 57 and 80 mm Hg, respectively. Infusions of glyceryl trinitrate (0.7 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) and dopamine (10 \(\mu\)g kg\(^{-1}\) min\(^{-1}\)) were continued intraoperatively. A live female infant was delivered 8 min after induction of anaesthesia with Apgar scores of 6 and 8 at 1 and 5 min, respectively, and received intramuscular naloxone (20 \(\mu\)g), oxytocin was administered by slow intravenous infusion following manual massage of the uterus. The patient demonstrated remarkable haemodynamic stability during delivery and for the remainder of the case.

Postoperatively, the patient was transferred to the cardiac intensive care unit, still intubated, for continuous haemodynamic monitoring. Sedation was continued with an intravenous propofol infusion and a remifentanil infusion at 0.8 \(\mu\)g kg\(^{-1}\) min\(^{-1}\) to maintain a Ramsay sedation score of 3. A morphine infusion at 3 mg h\(^{-1}\) was also commenced on return to the intensive care unit. The remifentanil infusion was discontinued after 3 h and the patient was extubated uneventfully 2 h later when all sedation had been weaned. Due to the high risk of thromboembolism, prophylaxis with the low molecular weight heparin enoxaparin (40 mg subcutaneously every 24 h) was commenced. After a period of observation the patient was returned to the cardiology high dependency unit. Her subsequent post-operative course was uncomplicated.

Further investigations carried out before discharge 2 weeks later included a myocardial biopsy revealing a non-specific inflammatory picture and echocardiography which demonstrated no improvement in ventricular function. When reviewed at 6 months, the patient felt symptomatically much improved but continued to have a reduced exercise tolerance and on echocardiographic examination showed global impairment of left ventricular function associated with moderate mitral regurgitation and a calculated left ventricular ejection fraction of 16%. The female infant remained healthy.

Discussion

Peripartum cardiomyopathy is a rare congestive cardiomyopathy of unknown aetiology which is associated with extremely high mortality. It was first described as a separate entity in the 1930s, distinguishing it from other causes of heart failure associated with pregnancy due to ischaemic, valvular, metabolic or infective aetiologies. It has several established diagnostic criteria including: (i) development of cardiac failure in the last month of pregnancy or within 5 months after delivery; (ii) absence of a determinable cause for cardiac failure; (iii) absence of demonstrable heart disease before the last month of pregnancy; and (iv) echocardiographically demonstrable impairment of left ventricular function. Other clinical conditions, which may mimic heart failure, such as anoxic and pulmonary emboli, should be excluded when considering the diagnosis.

Our patient presented at 39+6 weeks’ gestation describing a 2 week history of increasing shortness of breath. Before this presentation she was in good health with no suggestion of previous cardiac disease. Cardiotocograph and fetal assessment were unremarkable and there was no suggestion of pre-eclampsia or other obstetric aetiology for her deteriorating condition.

The delay in our patient’s diagnosis after initial presentation may reflect the rarity of this condition. Incidence is reported to range from one in 1300 to one in 15 000 pregnancies. Peripartum cardiomyopathy is thought to be more prevalent in women over 30 years of age, multiparous women, those of African descent and in women receiving tocolytic therapy. Other possible risk factors including toxoaemia of pregnancy, cocaine abuse and nutritional deficiencies have also been reported. The aetiology of peripartum cardiomyopathy is not known, raising the question that it may not in fact be a separate entity.
Various theories include hormonal and immunological imbalance as well as myocarditis2 as possible aetiopathological factors, but to date no study has been conclusive in this regard.

Our patient presented with symptoms that made initial diagnosis of this condition difficult. The symptoms of fatigue, cough, dyspnoea and abdominal pain are characteristic of peripartum cardiomyopathy but also of many of the differential diagnoses. Our diagnosis was one of exclusion based on the absence of any previous history of cardiac disease in a young, normally healthy female presenting acutely in the last month of pregnancy in association with the absence of clinical or echocardiographic findings suggestive of embolic disease. As our patient was already in active labour and beginning to present acutely, we were faced with the dilemma of deciding which method of delivery and which forms of anaesthesia and analgesia were most appropriate. In view of the patient’s haemodynamic status and the possible effects of a vaginal delivery on her systemic vascular resistance (SVR) and cardiac workload, it was decided that a Caesarean section would be the safest method of delivery.

With a recorded ejection fraction of 15% and the possible effects of haemodynamic compromise associated with sympathetic blockade, a general anaesthesia technique was felt to be the best method of providing stable analgesia and anaesthesia for delivery. Regional anaesthesia may have provided an advantageous reduction in afterload but its effects on haemodynamic status were felt to be less predictable than that of a general anaesthetic using suitable agents.

Remifentanil, a synthetic opioid, has several distinctive pharmacokinetic properties, which include a unique metabolism by plasma and tissue esterases and a context-sensitive half-life of 3–4 min, independent of the duration of infusion.8 Remifentanil’s opioid properties allow control of the intraoperative stress response and allow a more rapid recovery than other commonly used opioids. As our patient had significant haemodynamic instability, we felt that remifentanil could provide us with an agent which could significantly reduce the stress response and subsequent possible detrimental effect on SVR. The dose regimen chosen for the propofol and remifentanil infusions were based on the authors’ experience and practice in patients undergoing cardiac surgery.

The transfer of conventional opioids across the placenta may result in neonatal depression. The use of remifentanil would, in theory, through its metabolism and short duration of action, avoid this problem. Data on the pharmacokinetics of remifentanil in the neonate suggest a similar pattern of metabolism to older children and adults.9 10

In this case the infant was born with Apgar scores of 6 and 8 at 1 and 5 min, respectively. The decision to administer naloxone was taken by the paediatrician in attendance, who before the start of the procedure had been informed of the nature of the anaesthetic agents to be given. It is our opinion that the use of remifentanil minimized CNS and respiratory depression in the infant, a feeling supported by the experience of others.11

The decision to insert a PAFC in the presence of LBBB may be controversial in view of the risk of inducing complete heart block, but was taken as it was felt that the information obtained, such as pulmonary capillary wedge pressure and cardiac output, with the ability to derive parameters such as stroke volume and SVR, would be very helpful in the perioperative management of the patient. Various arrhythmias have been described during cardiac catheterization, but as the right bundle branch lies below the tricuspid valve, a pulmonary artery catheter may interfere with the normal conduction and result, as in our case, in complete heart block in those patients with pre-existing LBBB.12 13

Treatment of peripartum cardiomyopathy follows the same principles as that for other causes of cardiac failure, including diuretics, inotropic support, vasodilators and, more controversially, angiotensin-converting-enzyme inhibitors.2 Thromboembolic complications are not uncommon and the use of anticoagulants is often recommended.16

The use of immunosuppressives is controversial in that the vast majority of patients, like our patient, show only non-specific inflammatory changes on myocardial biopsy. However, cardiac transplantation has been successful in some patients.2 The mortality rate in the USA has been reported to range from 25% to 50%, the majority of deaths occurring within 3 months after delivery as a result of either progression of cardiac failure or sudden death associated with arrhythmias or thromboembolic phenomena.2 15 Data on recovery of myocardial function, assessed by both symptoms and echocardiography, suggest that some patients who survive will continue to have impaired cardiac function. The recurrence of peripartum cardiomyopathy in a further pregnancy in a patient previously diagnosed with the condition, whether or not cardiac function has recovered, is widely reported and most experts would discourage future conception in view of the extremely high risk of increased morbidity and mortality associated with both gestation and delivery.16 17

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Reduction of vasopressor requirement by hydrocortisone administration in a patient with cerebral vasospasm

J. A. Alhashemi

Division of Critical Care Medicine, London Health Sciences Centre – University Campus, University of Western Ontario, London, Ontario, Canada N6A 5A5. Present Address: Department of Anesthesia & Critical Care, King Abdulaziz University Hospital, King Abdulaziz University, PO Box 31648, Jeddah 21418, Saudi Arabia

A 67-yr-old female received hypertensive, hypervolaemic treatment for cerebral vasospasm after severe subarachnoid haemorrhage. After 3 days of continuous vasopressor infusion and despite adequate hydration and normal cardiac function, the phenylephrine dose had to be increased to obtain the same systolic blood pressure. This tachyphylaxis to phenylephrine infusion was probably caused by down-regulation of α adrenoceptors, and was reversed by giving i.v. hydrocortisone.

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Vasoactive drugs are used to induce hypertension in patients with subarachnoid haemorrhage (SAH) who develop cerebral vasospasm and are being treated with triple-H therapy (hypertension, hypervolaemia and haemodilution). This treatment is often needed for up to 2 weeks, beyond which time clinical vasospasm is rarely observed. Prolonged administration of adrenoceptor agonists has been associated with decreased adrenoceptor density, which may increase the need for these drugs to achieve the same induced hypertension. In this report, hydrocortisone reversed the increased dose requirement for phenylephrine in a patient with cerebral vasospasm. This is the first report of the use of hydrocortisone for tachyphylaxis.

Case history

A 67-yr-old otherwise healthy woman presented with Hunt and Hess' grade 4 SAH (World Federation of Neurosurgical Surgeons grade 5 haemorrhage) and was immediately admitted to the intensive care unit (ICU) for...