Obstetrical and Pediatric Anesthesia

Peripartum cardiomyopathy and thromboembolism; anesthetic management and clinical course of an obese, diabetic patient

[Cardiopathie périnatale et thromboembolie ; prise en charge anesthésique adoptée et évolution clinique d'une patiente diabétique et obèse]

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Purpose: To describe the anesthetic management and clinical course of a patient with peripartum cardiomyopathy. We highlight the frequent occurrence of thromboembolic morbidity in this group of parturients, emphasizing the need for early consideration of prophylactic anticoagulation.

Clinical features: A 38-yr-old, diabetic, obese parturient was admitted with pulmonary edema and severe orthopnea at 31 weeks gestation. The respiratory rate was 44 breaths·min⁻¹, blood pressure 110/70 mmHg, pulse 120 beats·min⁻¹ and rales were heard in both lung fields. The diagnosis of peripartum cardiomyopathy was made based on sinus tachycardia with no evidence of ischemia on the electrocardiogram, and global left ventricular hypokinesis with an ejection fraction of 40–45% noted on transthoracic echocardiography. Cesarean delivery was planned to improve maternal respiratory status and hemodynamics. General anesthesia with invasive monitoring was planned, and surgery and anesthesia proceeded uneventfully. Less than 24 hr postoperatively, she sustained a thrombotic cerebral infarct leaving her hemiparetic and dysarthric. Subsequent investigations revealed a thrombophilic state due to elevated anticardiolipin antibody.

Conclusion: General anesthesia is an acceptable option in parturients with heart failure secondary to cardiomyopathy. Thromboembolic complications are common, and early consideration should be given to prophylactic anticoagulation.

Objectif : Décrire la prise en charge anesthésique et l'évolution clinique d'une patiente présentant une cardiomyopathie périnatale. Souligner l'occurrence fréquente de morbidité thromboembolique dans ce groupe de parturientes et insister sur la nécessité d'une prophylaxie avec des anticoagulants.

Éléments cliniques : Une parturiente de 38 ans, obèse et diabétique, a été admise souffrant d'œdème pulmonaire et d'orthopnée sévère à 31 sem de grossesse. La fréquence respiratoire était de 44 respirations·min⁻¹, la tension artérielle de 110/70 mmHg, le pouls de 120 battements·min⁻¹ et des râles étaient perçus dans les deux champs pulmonaires. Le diagnostic de cardiomyopathie périnatale a été établi, fondé sur la tachycardie sinusale, sans évidence d'ischémie à l'électrocardiogramme, et d'hypokinésie ventriculaire gauche comportant une fraction d'éjection de 40–45 % notée à l'échocardiographie transthoracique. Une césarienne a été décidée afin d'améliorer l'état respiratoire et l'hémodynamique de la mère. L'anesthésie générale accompagnée d'un monitoring effractif a été utilisée pendant l'opération qui s'est bien déroulée. Moins de 24 h après la césarienne, la mère a subi un infarctus cérébral thrombotique qui a entraîné hémiparésie et dysarthrie. Des examens ultérieurs ont révélé un état thrombophilique causé par des anticorps anticardiolipine élevés.

Conclusion : L'anesthésie générale constitue une option acceptable chez des parturientes présentant une insuffisance cardiaque secondaire à une cardiomyopathie. Des complications thromboemboliques sont fréquentes et il faudrait rapidement administrer des anticoagulants préventifs.

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Accepted for publication June 4, 2002.

Revision accepted October 17, 2002.

PERIPARTUM cardiomyopathy is a clinical entity that is associated with high maternal morbidity and mortality.¹ It is defined by the following four criterias: 1) development of cardiac failure in the last month of pregnancy or within five months of delivery; 2) absence of identifiable cause for the heart failure; 3) absence of recognizable heart disease prior to the last month of pregnancy; and 4) left ventricular systolic dysfunction demonstrated by echocardiographic criteria.¹ Little is known about the etiology of this disease, although myocarditis of an immune nature had been proposed in earlier literature and is cited in a recent review.² However, newer biopsy data from patients with peripartum cardiomyopathy have been less convincing. Rizeq *et al.* showed a low incidence of myocarditis in patients with peripartum cardiomyopathy, that was comparable to age- and sex-matched controls with idiopathic dilated cardiomyopathy.³ The incidence of peripartum cardiomyopathy is variable, ranging from 1:4,000 to 1:3,000 live births, with many authors believing that the previously reported lower incidences had been underestimated.¹ The reported mortality figures range from 25%–50%, and the usual causes of death are progressive heart failure, arrhythmias, or thromboembolism.² The anesthetic management for Cesarean delivery is challenging, since patients may be hemodynamically unstable.

We present a case of cardiomyopathy occurring early in the third trimester, the anesthetic management, and the patient's clinical course.

Case report

A 38-yr-old G7P1A5 obese woman was referred to our hospital, when a pregnancy was discovered at 25 weeks gestation. Past medical history included fibromyalgia, hypercholesterolemia, and poorly controlled diabetes mellitus with painful polyneuropathy of upper and lower extremities. She was admitted for glycemic control. Laboratory testing for thrombophilia revealed negative ANA and RF factor, and anticardiolipin 14 MPL (IgM phospholipid; normal < 10 MPL, borderline 10–14 MPL, positive > 15 MPL). The first pregnancy, 20 years previously, was complicated by gestational diabetes requiring insulin, and Cesarean delivery. The baby weighed 5 kg. She subsequently had five first trimester miscarriages that were not investigated. Family history was notable for diabetes and aorto-coronary bypass at age 59, in her mother.

Five weeks after discharge from hospital, she was readmitted with acute dyspnea. Respiratory rate was 44 breath·min⁻¹, oxygen saturation was 88% on room air, blood pressure (BP) 110/70 mmHg and pulse 120

beats·min⁻¹. Auscultation of the lungs revealed rales bilaterally. Initial investigations showed hemoglobin 101 g·L⁻¹, troponin I 6.55 ng·mL⁻¹ (normal < 0.15 ng·mL⁻¹), pulmonary edema on *x-ray*, and sinus tachycardia on the electrocardiogram (ECG) with no evidence of ischemia. Blood gases showed pH 7.46, pCO₂ 31.6 mmHg, HCO₃ 22.2 mmol·L⁻¹ and pO₂ 67.8 mmHg with FiO₂ 0.21. Bedside transthoracic echocardiography showed an ejection fraction (EF) of 45%, diffuse segmental wall motion abnormalities, diastolic dysfunction and pulmonary arterial pressure of at least 50 mmHg. The left and right ventricular sizes were normal. There was trace aortic insufficiency, and mild to moderate mitral regurgitation. The differential diagnosis was ischemic cardiomyopathy with congestive heart failure, acute pulmonary embolism, or cardiomyopathy from some other cause (including peripartum, viral, or idiopathic.) She was transferred to the care of the cardiologists in the coronary intensive care unit where she was anticoagulated with continuous heparin infusion, and given furosemide intravenously.

The next day her respiratory rate was 18–24 breath·min⁻¹ and oxygen saturation 97% with FiO₂ 0.21. Doppler studies of the legs revealed no thromboses, and pulmonary ventilation perfusion scan showed a low probability of embolus. She had proteinuria (800 mg·24 hr⁻¹), but as she was normotensive this was attributed to diabetic nephropathy. Oral metoprolol was started at 25 mg twice daily and she continued to require furosemide. She did not receive inotropic medication or afterload reducing agents.

At this time, a multidisciplinary meeting was held with the cardiologist, the treating obstetrician, and the consulting cardiac anesthesiologist. The cardiologist favoured the diagnosis of peripartum cardiomyopathy. He felt that ischemic cardiomyopathy was unlikely in view of two main factors. First, the hypokinesis seen on the echocardiogram was diffuse and not limited to a specific territory. Second, the continued lack of ECG abnormality suggested another pathology. The cardiologist's recommendation was to discontinue anticoagulation, and to perform early delivery by Cesarean section to improve maternal respiratory and hemodynamic status.

Preoperative evaluation revealed an anxious patient, with orthopnea. Her weight was 115.8 kg, BP 120/85 mmHg, heart rate 105 beats·min⁻¹ and respiratory rate 18 breath·min⁻¹. She had a Mallampati 3 airway, with normal mandibular length. Coagulation was normal, and hemoglobin was 85 g·L⁻¹. The fall in hemoglobin level since admission was not explained. General anesthesia was planned in order to avoid the potential difficulties of a high thoracic block in this dyspneic patient.

In the operating room, invasive arterial BP was recorded in addition to standard monitoring. Pre-induction blood gas values showed a pO_2 76 mmHg with oxygen 3 L·min⁻¹ via nasal prongs, pH 7.48, pCO_2 37 mmHg, and HCO_3^- 27.5 mmol·L⁻¹. Premedication consisted of 30 mL sodium citrate and midazolam 2 mg *iv*. A modified rapid sequence induction with opioid was used. Induction drugs included sufentanil 50 µg, thiopental 100 mg, lidocaine 100 mg. Succinylcholine 200 mg facilitated an easy intubation. Anesthesia was maintained with isoflurane 0.4% in 100% oxygen. Muscle relaxation was achieved with rocuronium. At the time of intubation oxygen saturation dropped to 88%, rose to 92% with positive pressure ventilation, and improved to 98% with 10 cm H₂O positive end-expiratory pressure. There was no hypotension on induction, nor did the BP rise above 120 mmHg systolic. Heart rate remained stable at 100–110 beats·min⁻¹. A healthy male infant was delivered, with Apgar scores of 9 and 9 at one and five minutes, requiring no naloxone. The birth weight was 1770 g. The umbilical venous pH was 7.31, and the baby was transferred to the neonatal intensive care unit. There was short-lived uterine atony, requiring 100 U·L⁻¹ oxytocin *iv* infusion, and rectal administration of misoprostol 800 µg. Morphine 10 mg and sufentanil 200 µg were given after delivery. After skin closure, a pulmonary artery catheter was inserted via the right internal jugular vein. The pulmonary artery pressure was 55/31 mmHg, and central venous pressure was 19 mmHg. Cardiac output and wedge pressure were not recorded in the chart. Nitroglycerin *iv* was started to reduce preload which was judged to be high based on the pulmonary diastolic pressure. At this time, arterial blood gas analysis revealed a pH 7.37, pO_2 146 mmHg, pCO_2 51 mmHg and HCO_3^- 29 mmol·L⁻¹ on an inspired fraction of oxygen of 1.0. Hematocrit was 29%. No vasopressors were required. Estimated blood loss was 500 mL, and the patient received 1500 mL of crystalloid. Duration of surgery was 96 min. The patient was transferred to the coronary intensive care unit intubated, ventilated and sedated. Initial vital signs were BP 120/70 mmHg and pulse 100 beats·min⁻¹. Hemodynamic variables were pulmonary artery pressure 36/20 mmHg, wedge pressure 22 mmHg, and central venous pressure 14 mmHg. Cardiac output was not measured. Vasoactive medication included only *iv* nitroglycerin infusion. Within two hours, she was weaned to an FiO_2 of 0.5 and pressure support ventilation. Initial neurological assessment revealed a patient responding appropriately to verbal stimuli and moving all four limbs.

Postoperative pain was managed with parenteral morphine.

Seventeen hours postoperatively, she was noted to be confused, and not obeying commands. Examination revealed a dense right sided hemiparesis. Urgent head computed tomography scan showed a focal hypodense area in the left temporal lobe. Anticoagulation was deferred because of the risk of cerebral hemorrhage.

At this time the pulmonary edema had improved and ramipril was added to her therapy.

The following day, a transesophageal echocardiogram ruled out an embolic source for the cerebrovascular insult, with negative bubble contrast studies. The echocardiogram showed diffuse left ventricular hypokinesis with an EF of 40%.

Her respiratory status continued to improve and she was extubated on the third postoperative day. After noting that she was aphasic, she was transferred to the neurological intensive care unit.

Repeat testing for thrombophilia was performed. The IgM anticardiolipin value was 19 U (normal < 15 U) with normal ANA and RF values. The neurologic insult was ultimately attributed to thrombosis of the left middle cerebral artery precipitated by a thrombophilic state. Anticoagulation was started with clopidogrel, low dose aspirin, and *sc* heparin. Lifelong warfarin therapy was planned.

Her condition improved over the next four weeks, with return of speech, and limited movement in her right leg. She was then transferred to a rehabilitation centre.

Diuretic requirement persisted in the months following discharge, and she was eventually evaluated by the cardiac transplantation service. She is presently listed for a cardiac transplantation.

Discussion

Since the etiology of heart failure was unclear in this case, clinical decision-making was challenging. Despite seven days of diuresis and beta blockade, the patient was still orthopneic upon arrival in the operating room. This influenced the choice of anesthetic technique as mentioned above. Perhaps better medical optimization of her heart failure would have made management easier. However, the cardiologist maintained that more invasive diagnostic procedures, such as coronary angiography, were not indicated and that also there would be no advantage to adding preoperative inotropes or vasodilators.

The choice of parenteral opioids rather than a neuraxial technique for postoperative analgesia was based on the patient's strong aversion to spinal injections and the pre-existing painful diabetic neuropathy.

The anesthetic considerations for a patient with heart failure presenting for Cesarean section are simi-

lar regardless of etiology. Hemodynamic goals include maintenance of normal to low heart rate to decrease oxygen demand, and prevention of large swings in BP. These goals may be achieved with either general or regional techniques.⁴ We used an opioid based induction to avoid the myocardial depression that is associated with high dose thiopental or propofol. We felt that pulmonary artery catheterization could be delayed until after delivery due to the relative hemodynamic stability preoperatively. Postoperative care would be facilitated by the presence of a pulmonary artery catheter.

A recent case report of the management of Cesarean section in a parturient with peripartum cardiomyopathy and complete left bundle branch block, describes the use of remifentanyl and propofol for induction and maintenance of anesthesia.⁵ Insertion of a PA catheter was complicated by complete heart block, but resolved with removal of the catheter. This case report is interesting because it addresses the concern for neonatal well-being when using opioid-based regimens for maternal reasons. In our case, we informed the neonatologist of our plans, and there was adequate preparation for neonatal resuscitation. Interestingly, with the use of remifentanyl in McCarroll *et al.*'s case, naloxone was administered to the infant, while in our case none was needed.

Shnaider *et al.* present a case of peripartum cardiomyopathy in a morbidly obese primigravida at 36 weeks gestation.⁶ The patient had an EF of 20%, and was treated medically for ten days. She was eupneic on arrival to the operating room. They used a combined spinal-epidural technique, and maintained stable hemodynamics with a sensory level at T5. The patient we describe was more dyspneic and would probably not have tolerated a T5 block. Pirlet *et al.* described another patient managed with regional anesthesia.⁷ This patient had an EF of 13%, which improved to 20% with medical management, and was brought for semi-urgent delivery by Cesarean section. She was monitored with pulmonary artery catheterization pre-induction, and was then given 5 mg intrathecal hyperbaric bupivacaine. Significant hypotension rapidly resulted (BP 65/35 mmHg) which was treated with ephedrine infusion. Subsequent epidural boluses of 10 mL bupivacaine 0.5% were needed to obtain a sensory level to T3. This case report suggests that even low intrathecal doses of local anesthetic may cause major hemodynamic effects.

Chan and Ngan Kee⁸ present another case of cardiomyopathy that presented at 18 weeks gestation. The patient was managed medically, with systemic anticoagulation, until 31 weeks, when fetal distress and

maternal liver dysfunction forced an emergency Cesarean section. There was severe cardiac dysfunction based on pre-induction hemodynamic measurements, and a general anesthetic technique was used. Inotropic support was needed for low cardiac index. The patient continued to be treated for heart failure with moderate improvement. Two weeks after discharge she suffered a severe embolic stroke despite monitored warfarin therapy. She died five months later.

The case report by Chan and Ngan Kee, as well as the present case both emphasize the importance of thromboembolic complications associated with peripartum cardiomyopathy. The recent review of peripartum cardiomyopathy by the National Heart, Lung, and Blood Institute suggests that "patients with significantly depressed left ventricular function (EF < 35%) may benefit from anticoagulation therapy".¹

Further, even in the absence of cardiomyopathy, thromboembolism is a common cause of maternal death. In the United Kingdom it is the most common cause of maternal mortality, based on the Report on Confidential Enquiries into Maternal Deaths.⁹ This report recommends that high risk patients receive heparin prophylaxis. Aside from the later discovered thrombophilic state, our patient had several pre-existing risk factors for thromboembolism including obesity, prolonged bedrest, advanced maternal age, hypokinetic left ventricle and the procoagulant effect of pregnancy. One wonders whether her outcome would have been different had she been receiving perioperative anticoagulation. In our institution, it is the surgical service that ultimately decides if perioperative anticoagulation prophylaxis is required. We learn from this case that the anesthesiologist should take a more active interest in this aspect of patient care.

In summary, peripartum cardiomyopathy is a severe form of heart failure that causes significant mortality, and whose incidence may have been under-reported in earlier literature.¹ Ruling out other causes of cardiomyopathy is essential for diagnosis, and has important implications for treatment. Medical management must address the classical goals of heart failure therapy, and should include consideration of thromboembolic prophylaxis. Various options are appropriate for anesthetic management.

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