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Peripartum Cardiomyopathy Presenting at Cesarean Delivery

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Peripartum cardiomyopathy is defined as the onset of primary myocardial disease (without demonstrable cause) during the last month of pregnancy or within the first 5 months after delivery.[1] It occurs in less than one in 4,000 pregnancies and represents less than 1% of all cardiovascular disease that occurs during pregnancy.[2]

We describe two patients with peripartum cardiomyopathy whose initial sign of cardiac disease, acute left ventricular failure, occurred during anesthesia for cesarean delivery requiring immediate respiratory and cardiovascular support.

These cases are unique both for the timing of their myocardial decompensation as well as for their rapid recovery with aggressive cardiorespiratory therapy.

Report of Two Cases

Case 1. A 25-year-old gravida 3, para 1, abortus 1 presented in labor 7 h before her scheduled repeat cesarean delivery. Her primary cesarean delivery, necessitated by cephalopelvic disproportion, was performed after a long labor. Continuous lumbar epidural anesthesia was employed for labor as well as for cesarean delivery. Her operative and postpartum course was uneventful.

She had no personal or family history of cardiovascular disease, and her pregnancy had been unremarkable. She was given 1,300 ml lactated Ringer's solution iv immediately before induction of anesthesia. With left uterine displacement, her preanesthetic blood pressure was 120/70 mmHg, and her heart rate was 100 beats/min (sinus rhythm). Spinal anesthesia was induced with 65 mg 5% lidocaine in 7.5% dextrose solution. Supplemental oxygen was given via a face mask, and left uterine displacement was continued. Within 2 min, a T4 level of sensory anesthesia was detected by pinprick. While her abdomen was being prepared for surgery, she began to complain of chest discomfort and breathlessness. Her respiration appeared adequate and her level of anesthesia remained unchanged.

Approximately 8 min after induction, heart rate decreased precipitously to 35 beats/min (sinus bradycardia), and arterial blood pressure decreased to 80/40 mmHg. Her level of anesthesia had not changed. She responded to the second of two 0.4-mg doses of iv atropine after iv ephedrine 15 mg had not corrected her hypotension and bradycardia. Arterial blood pressure increased to 160/100 mmHg and heart rate increased to 160 beats/min (supraventricular tachycardia). One minute later, upon surgical incision, the obstetricians noted dark blood in the wound just as the patient, who was becoming increasingly agitated, coughed up a copious amount of frothy pink fluid. There was no change in vital signs. General anesthesia was induced immediately using 250 mg iv thiopental and 100 mg iv succinylcholine with cricoid pressure and tracheal intubation. Ventilation was controlled manually with an $F_{I_{O_2}}$ of 1.0 and peak inspiratory pressures of 70 cmH_2O. Arterial blood pressure was 160/100 mmHg, with a heart rate of 140 beats/min (sinus tachycardia). A full-term, living male infant was delivered 2 min later. Apgar scores at 1 and 5 min were 9 and 9, respectively. The *p*H of umbilical cord venous blood was 7.31. Five min postpartum, maternal pH_a was 7.25, Pa_{O_2} 42 mmHg, Pa_{CO_2} 42 mmHg, HCO_3^- 16 mEq/l, base deficit −9 mEq/l, and measured oxygen saturation was 70%. A positive end-expiratory pressure (PEEP) valve was added to the anesthesia breathing circuit. Maternal arterial blood pressure was 100/50 mmHg and heart rate was 120 beats/min (sinus tachycardia). Anesthesia was maintained with oxygen, iv increments of fentanyl (total dose 100 μg), diazepam (10 mg), and a 0.1% succinylcholine infusion. Twice during maintenance of anesthesia, the systolic arterial blood pressure decreased to 60 mmHg. Maternal heart rate remained 120 beats/min (sinus tachycardia). Arterial blood pressure was restored initially with iv calcium chloride (total dose 1 g) but ultimately required a dopamine infusion (5.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$). Ventilation with 100% oxygen and tidal volume of 800 ml with 7.5 cmH_2O PEEP improved Pa_{O_2} to 100 mmHg. During the operation she received 3 l of crystalloid iv, lost an estimated 600 ml of blood, and produced 200 ml of urine.

Upon admission to the recovery room, her heart rate was 100 beats/min (sinus rhythm). Arterial blood pressure was 105/50 mmHg. Immediate pulmonary artery catheterization via the right internal jugular vein showed pulmonary artery pressures of 45/25 mmHg and a pulmonary capillary wedge pressure of 28 mmHg. Cardiac output measured by thermodilution was 4.5 l/min. The calculated systemic vascular resistance was 1,800 $dynes \cdot s^{-1} \cdot cm^{-5}$. An immediate postoperative chest radiograph was remarkable for widespread bilateral interstitial infiltrates and a grossly enlarged cardiac silhouette. Intravenous furosemide (20 mg) improved urine output. An aspirate of pulmonary capillary blood showed no fetal cells (a sign of amniotic fluid embolism). Her coagulation profile was within normal limits.

The patient gradually recovered over the next 48 h and was weaned from ventilatory and inotropic support. A ventilation–perfusion lung scan was interpreted as being incompatible with the diagnosis of pulmonary embolus. Serial electrocardiograms (ECG) and cardiac isoenzymes showed no evidence of myocardial infarction. A right bundle branch block was seen in some ECG tracings. An echocardiogram performed three days postpartum demonstrated severe biventricular hypokinesis, ventricular dilatation, and normal valvular motion. Ejection fraction was estimated at 30%. Signs of cardiac failure disappeared over the next few days, and the patient was transferred to the postpartum floor. Six days postpartum, radionuclide ventriculogram confirmed normal ejection fraction (70%).

The patient was discharged 1 week after delivery, taking no medi-

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cations. She was able to care for her two children in a normal fashion. Follow-up care was provided by her obstetrician and cardiologist. Subsequent clinical and diagnostic examinations revealed that her heart size and left ventricular function have remained normal.

Case 2. A 17-year-old primagravida who had received no antenatal care was admitted in premature labor at 30 weeks estimated gestational age. There was no personal or family history of cardiovascular disease. Initial laboratory findings supported a tentative diagnosis of hydrops fetalis.

Tocolysis was attempted unsuccessfully with subcutaneous terbutaline. The patient then labored until fetal distress developed. She was taken to the operating room for emergency cesarean delivery. Three liters lactated Ringer's solution had been infused iv during the 10 h since her admission.

Preanesthetic arterial blood pressure was 140/80 mmHg. Rapid-sequence induction of general anesthesia with 300 mg iv thiopental and 100 mg iv succinylcholine proceeded uneventfully. After endotracheal intubation, auscultation of both lungs revealed right upper lobe rhonchi, which persisted throughout the operation. Anesthesia was maintained with 50% oxygen and 50% nitrous oxide and 1% enflurane. A premature, living male child with Apgar scores at 1 and 5 min of 2 and 4, respectively, was delivered and taken to the neonatal intensive care unit. Enflurane was discontinued, gas flows were changed to 40% oxygen and 60% nitrous oxide, and 5 mg iv morphine sulfate and 4 mg iv diazepam were given. An iv infusion of 0.1% succinylcholine was used to maintain muscle relaxation. The mother's anesthetic and surgical course was uneventful. The trachea was extubated when she was responsive, breathing spontaneously with an arterial blood pressure of 130/80 mmHg and a heart rate of 100 beats/min (sinus rhythm). An additional liter of lactated Ringer's solution was infused iv during surgery.

Immediately after her arrival in the recovery room, the patient became acutely dyspneic. Coarse rales were heard in both lungs. Analysis of arterial blood revealed pH_a 7.38, Pa_{O_2} 33 mmHg, and Pa_{CO_2} 30 mmHg. Her trachea was reintubated and ventilation controlled with an FI_{O_2} of 1.0 and PEEP of 10 cmH_2O. Frothy, pink fluid filled the endotracheal tube. Arterial blood pressure rose to 180/120 mmHg. Furosemide 20 mg and sodium nitroprusside ($4\ \mu g \cdot kg^{-1} \cdot min^{-1}$) were infused iv. A chest radiograph showed an enlarged cardiac silhouette and bilateral interstitial infiltrates. Pulmonary artery pressures of 63/33 mmHg, pulmonary capillary wedge pressure of 30 mmHg, and central venous pressure of 18 mmHg were recorded. Cardiac output measured by thermodilution was $5.1\ l \cdot min^{-1}$.

The infant died at 4 h of age as a consequence of α-thalassemia. The mother gradually improved over the next 72 h. Serial ECGs and cardiac isoenzymes showed no evidence of myocardial infarction. Her coagulation profile was within normal limits. An echocardiogram 2 days postpartum demonstrated ventricular wall hypokinesis and dilatation with a calculated ejection fraction of 30%. The patient's cardiorespiratory status continued to improve over the next week. A radionuclide ventriculogram 8 days after delivery confirmed that contractile function and ejection fraction had returned to normal (70%). She was discharged 3 weeks later, being treated with 0.25 mg digoxin po daily. Follow-up care was provided by her obstetrician and cardiologist. Subsequent clinical and diagnostic examinations confirmed tht her heart size and left ventricular function remained normal.

Discussion

Idiopathic myocardial failure first was associated with the puerperium in the nineteenth century; however, it was not until the middle of this century that a separate disease entity was first described.[2] Predisposing factors for this disease include maternal age greater than 30 years, race (more black patients than white), multiparity, multiple gestation, toxemia, poor nutritional status, geography (a higher incidence in tropical and subtropical climates), and breast feeding of infants.[2]

Peripartum cardiomyopathy occurs most commonly in the second postpartum month.[2] Patients often present with left ventricular failure, dyspnea, edema, and gallop rhythms.[3] Emboli to both cerebral and systemic circulations may occur.[4,5] Laboratory investigation of these patients usually yields nondiagnostic findings. Electrocardiography may show varied abnormalities such as left ventricular hypertrophy, low voltage changes with T-wave inversion, or interventricular conduction defects.[2,3] Echocardiography has shown dilated, hypokinetic ventricles in most cases.[2,6,7]

A limited number of patients have undergone endomyocardial biopsies. Degenerative changes, mural thrombi, and intracellular and extracellular deposition of fibrous material have been seen by light microscopy.[2,8] The latter may account for the impaired myocardial contractility characteristic of peripartum cardiomyopathy.[8]

The course of the disease relates to the duration and severity of cardiomegaly.[1,3] Recommended treatment includes the usual therapies for cardiac failure, including bed rest, low sodium diet, digoxin, and diuretics. Anticoagulation, suppression of lactation, and steroid immunosuppression also have been advocated.[2]

The overall mortality rate from peripartum cardiomyopathy is between 30% and 60%. Ten to 20 per cent of patients die during their initial hospitalization.[2] Survivors have a 50–80% chance of developing cardiac failure during future pregnancies, with an associated mortality rate of 60%.[2] One study, limited to women whose cardiomegaly resolved within 6 months and who later became pregnant found the incidence of recurrence of cardiac decompensation to be 25%. There was no cardiac-related maternal mortality in that study.[3]

Common necropsy findings include a grossly dilated heart with biventricular hypertrophy, pale myocardium with endocardial thickening, and mural thrombi. Heart valves and coronary arteries are typically normal.[2]

Our patients first showed signs of cardiac failure at the time of otherwise uneventful anesthetics. This most likely related to an inability to meet the increased cardiac demand that normally accompanies the intrapartum and postpartum periods.

The differential diagnoses of intraoperative pulmonary edema in a parturient includes many different causes. Noncardiac causes of pulmonary edema include amniotic fluid or pulmonary emoblism, beta-mimetic tocolytic therapy, and toxemia. Cardiac causes of pulmonary edema include decompensated valvular and subvalvular cardiac

disease, myocardial ischemia and infarction, fluid overload, and cardiomyopathy.

The differential diagnosis that was faced in the operating room and intensive care unit was restricted by our patients' histories and clinical presentations.

Beta-mimetic tocolytic therapy has been associated with low wedge pressure pulmonary edema. Hemodynamic measurements in patients receiving terbutaline therapy typically include normal wedge pressures and high cardiac outputs.[9] The first patient did not receive tocolytic therapy. Our second patient, who received terbutaline, had cardiac outputs that were lower than would be expected immediately postpartum, and her pulmonary capillary wedge pressures were elevated. Neither of our patients showed any signs of toxemia during their pregnancy.

Only embolic events or cardiac disease seemed likely diagnoses. The results of initial diagnostic tests were incompatable with either pulmonary embolus or amniotic fluid embolus, as was the rapid resolution of the patients' conditions. Cardiac causes of the acute cardiac failure then were investigated.

Neither of our patients had any antenatal history of progressive dyspnea, fatigue, syncope, or chest pain. Clinical examination of these patients before cesarean delivery failed to reveal any signs of heart disease. Postpartum electrocardiograms and serial cardiac return isoenzymes were nondiagnostic of myocardial infarction.

A cardiomyopathy can be classified as to the differences in its presentation: congestive, restrictive, or hypertrophic. Congestive cardiomyopathies include the following: familial cardiomyopathy, alcoholic cardiomyopathy, cardiomyopathy secondary to neuromuscular diseases, and peripartum cardiomyopathy.[10] Evidence for the diagnosis of congestive cardiomyopathy is gathered from the physical examination and presentation of biventricular failure. Cardiac arrhythmias often are seen. Echocardiography is employed to exclude pericardial effusion and to evaluate left ventricular contractility.[10] Cardiac output is typically reduced and will not increase with exertion or stress (such as in the puerperium).

In both patients, postpartal chest radiography demonstrated cardiomegaly. Pulmonary arterial catheterization showed increased pulmonary capillary wedge pressures and decreased cardiac output. Two-dimensional echocardiography revealed normal valve motion but dilated ventricles with poor wall motion and very low ejection fractions, confirming our belief that iatrogenic fluid overload was not the cause of the congestive heart failure.

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