

Severe Preeclampsia, Pulmonary Edema, and Peripartum Cardiomyopathy in a Primigravida Patient

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Peripartum cardiomyopathy (PPCM) is a rare form of heart failure of unknown etiology that is associated with late pregnancy and the early postpartum period. Although the complete pathogenesis of PPCM is not completely understood, the signs and symptoms are identical to those of left ventricular heart failure. The diagnosis of PPCM is made in a parturient only after other causes of heart failure are ruled out. Management of PPCM is similar to that of congestive heart failure with a few exceptions, such as avoiding the use of angiotensin-converting enzyme inhibitors dur-

ing pregnancy. This report describes the presentation and anesthetic management of a parturient who was admitted with a diagnosis of severe preeclampsia in whom pulmonary edema and heart failure developed, necessitating emergency cesarean delivery under general anesthesia. The patient was subsequently given a diagnosis of PPCM.

Keywords: Emergent cesarean delivery, peripartum cardiomyopathy, peripartum pulmonary edema, severe preeclampsia.

Peripartum cardiomyopathy (PPCM) is a rare life-threatening cardiomyopathy of unknown cause that occurs in the peripartum period in previously healthy women. Typically, it can occur in the last month of pregnancy or in the 5 months following delivery.¹⁻³ A recent, large study of 240,000 deliveries over a 10-year time span determined the overall incidence of PPCM to be 1 in 4,075,⁴ demonstrating that it is a rare occurrence. Multiple risk factors have been linked with the occurrence of PPCM. General risk factors for cardiovascular disease include hypertension, obesity, diabetes, smoking, preeclampsia, advanced maternal age, and malnutrition.¹

Symptoms of PPCM are identical to those of congestive heart failure and include fatigue, paroxysmal nocturnal dyspnea, pulmonary edema, pedal edema, and distended neck veins.¹ Symptoms such as fatigue, dyspnea, and edema are often present in late pregnancy, making it difficult to identify patients with PPCM.³ Additionally, preeclamptic parturients may manifest symptoms of respiratory distress due to capillary leak.⁵ We describe the presentation and anesthetic management of a parturient who was admitted with a diagnosis of severe preeclampsia, and whose health status quickly declined secondary to fulminant pulmonary edema. Subsequently PPCM was diagnosed.

Case Summary

A 26-year-old, 149.9-cm, 69 kg, gravida 1, para 0, white woman presented at 35 weeks' gestation to labor and

delivery for evaluation of worsening shortness of breath. At the time of admission, the patient described worsening shortness of breath over the last 3 days with an inability to lie flat as well as blurred vision and lower extremity edema. Her medical history was noncontributory, and she had an uncomplicated prenatal course, with the exception of excessive weight gain. Initial vital signs were blood pressure (BP) of 157/100 mm Hg, heart rate (HR) of 140/min, respiratory rate (RR) of 22/min, temperature of 36.6°C, and an oxygen saturation of 92% on room air. Results of the airway examination showed Mallampati class III but were otherwise normal. Fetal HR was in the 140/min range with good variability, and contractions were every 2 to 4 minutes. Physical findings included 2 to 3+ edema in the lower extremities, +4 deep tendon reflexes, and lung sounds revealed bilateral crackles at the bases. Dipstick urinalysis revealed 4+ protein. Based on these findings, the patient was admitted with a diagnosis of severe preeclampsia.

Preeclampsia toxicology laboratory tests (complete blood cell count, complete metabolic profile, liver function tests, and coagulation tests) were performed, and a stat chest x-ray film was ordered to rule out pulmonary edema. A magnesium bolus of 4 g, followed by 2 g/h intravenously (IV), was administered. Initial laboratory results were as follows: white blood cells, $16.1 \times 10^3/\mu\text{L}$; hemoglobin, 13.1 g/dL; hematocrit, 37.3%; platelets, $270 \times 10^3/\mu\text{L}$; international normalized ratio, 1.0; prothrombin time, 12.5 seconds; partial thromboplastin time, 26.4 seconds; fibrinogen, 596 g/dL; sodium, 136

mEq/L; potassium, 2.6 mEq/L; chloride, 101 mEq/L; carbon dioxide, 23 mEq/L; serum urea nitrogen, 7 mg/dL; creatinine, 0.7 mg/dL; glucose, 70 mg/dL; magnesium, 1.4 mg/dL; aspartate aminotransferase, 34 U/L; alanine aminotransferase, 20 U/L; and uric acid, 6.4 mg/dL. One hour and 40 minutes after admission the patient received labetalol, 10 mg IV, for treatment of severe hypertension (167/107 mm Hg). At this time furosemide, 10 mg IV, was administered based on chest x-ray results demonstrating pulmonary edema and evidence of worsening respiratory function.

Two hours after admission the patient appeared to be acutely decompensating, as evidenced by a labored RR of 36/min, oxygen saturation of 81% on 15 L of oxygen via a nonrebreather mask, rhonchi in all lung fields, BP of 157/112 mm Hg, and frequent late decelerations in fetal HR. An additional 10-mg dose of furosemide was administered IV, and the patient was transferred emergently on 15 L of oxygen to the operating room for immediate intubation and emergency cesarean delivery. Upon the patient's arrival in the operating room, her oxygen saturation was 68%, BP was 140/80 mm Hg, and sinus tachycardia was present with a HR of 125/min. Standard anesthesia monitors were applied, the patient was oxygenated with 100% oxygen, and a rapid sequence induction was performed using 100 mg of propofol, 100 mg of lidocaine, and 120 mg of succinylcholine IV. Direct laryngoscopy revealed frothy sputum bubbling from the glottic opening. The trachea was intubated with a 6.5-mm internal-diameter endotracheal tube.

Cesarean delivery commenced immediately (2 hours and 20 minutes after admission) following intubation and confirmation of the presence of end-tidal carbon dioxide (ETCO₂). Vital signs after induction were as follows: BP, 118/70 mm Hg; HR, 122/min; arterial oxygen saturation (SaO₂), 73%; and ETCO₂, 36 mm Hg. After intubation, oxygen saturations improved to only 70% to 80% despite ventilation with 100% oxygen, 400-mL tidal volume (TV), an RR of 20/min, 5 cm H₂O of positive end-expiratory pressure (PEEP), and intermittent suctioning of frothy secretions. Therefore, endotracheal tube placement was confirmed fiberoptically.

Anesthesia was maintained with 1.0% sevoflurane in 100% oxygen, and neuromuscular blockade was maintained with rocuronium. A right radial arterial line was placed as soon as possible. Throughout the case the patient was tachycardic in the range of 110/min, and blood pressure ranged from 120/70 mm Hg to 138/82 mm Hg, with oxygen saturations ranging from 68% to 83%. After delivery, 2 mg of midazolam was administered, and an infusion of 40 U of pitocin in 1 L of lactated Ringer's solution was started. Bleeding was minimal, with an estimated blood loss of 500 mL. Urine output was 600 mL, with a total of 1,600 mL of lactated Ringer's solution administered. Initial arterial blood gas analysis

performed 44 minutes after the start of the case revealed an uncompensated respiratory and metabolic acidosis with hypoxemia (pH, 7.19; PaO₂, 48 mm Hg; PaCO₂, 57.2 mm Hg; SaO₂, 78%; bicarbonate (HCO₃), 21.8 mEq/L; and base excess (BE), -6 mEq/L). By the end of the case, the acidosis was corrected, but hypoxemia continued to persist (pH, 7.37, PaO₂: 58 mm Hg, PaCO₂: 42 mm Hg, SaO₂, 83%, HCO₃ 24.4 mEq/L, BE -1 mEq/L) despite administration of fraction of inspired oxygen (FIO₂), 1.0.

Upon completion of the surgery (3 hours and 15 minutes after admission), the patient was transferred intubated to the intensive care unit (ICU) for definitive management of pulmonary edema and severe preeclampsia. Vital signs on admission to the ICU were as follows: BP, 125/83 mm Hg; HR, 97/min; oxygen saturation by pulse oximetry (SpO₂), 75% on synchronous intermittent mandatory ventilation (SIMV); RR, 18/min; TV, 450 mL; pressure support (PS), 10 mm Hg; and PEEP, 10 mm Hg. The infant was transferred to the neonatal ICU for close monitoring and was subsequently intubated 6 hours after delivery because of respiratory failure.

While the patient was in the ICU, the clinical picture and respiratory function initially improved, with oxygen saturations ranging from 93% to 98% with medical management. The management included seizure prophylaxis with magnesium infusion, heparin anticoagulation, fluid restriction, diuresis with furosemide, and afterload reduction with sodium nitroprusside (SNP) infusion. A pulmonary embolus was ruled out by spiral computed tomographic (CT) scan. However, on the second post-operative day the patient experienced another episode of fulminant pulmonary edema, necessitating increased ventilatory requirements. (The initial settings on SIMV were TV, 450 mL; RR, 10/min; FIO₂, 40%; PS, 10 mm Hg; and PEEP, 10 mm Hg.) The new settings using assist/control ventilation were as follows: TV, 450 mL; RR, 10/min, FIO₂, 80%; PS, 10 mm Hg; and PEEP, 12 mm Hg. Brain natriuretic peptide results at the time of acute decompensation were more than 1,600 pg/mL. The Cardiology Service was consulted, and a bedside echocardiogram was performed, which revealed an ejection fraction of approximately 30%, with severe left ventricular global hypokinesis and left atrial dilation. The patient was diagnosed with PPCM of pregnancy based on the echocardiogram and on clinical symptoms of congestive heart failure and pulmonary edema developing at the time of delivery.

During a 4-day stay in the ICU, medical management continued and included furosemide diuresis, sodium and fluid restriction, blood pressure control with SNP and captopril, and slow weaning from assist/control ventilation. A formal transthoracic echocardiogram revealed severely reduced left ventricular systolic function, moderate to severe global hypokinesis of the left ventricle, and an ejection fraction of 15% to 25%. However, the

patient's symptoms continued to improve with the described medical management. The patient was extubated on postoperative day 4 and subsequently discharged to home 9 days after delivery on a regimen of lisinopril and carvedilol.

At discharge, she was counseled on the risks associated with subsequent pregnancies and was started on a regimen of oral contraceptives. Subsequently she had an intrauterine device placed. Nine months after discharge the patient's ejection fraction had improved to 40% to 50%. However, she still required continuous oxygen therapy for exercise-induced hypoxemia.

Discussion

• *Preeclampsia vs Peripartum Cardiomyopathy.* Preeclampsia is a multiorgan disease, characterized by hypertension and proteinuria, in the parturient after 20 weeks' gestation. Although edema is no longer required for diagnosis, it is frequently present. Preeclampsia is considered to be severe when BP is greater than 160 mm Hg systolic and 110 mm Hg diastolic, proteinuria is greater than 5 g, or signs and symptoms are consistent with end organ damage.⁶ Signs and symptoms of end organ damage may include neurologic symptoms, such as headache or visual disturbances, impaired coagulation, impaired hepatic function or cyanosis, or pulmonary edema.⁶ Pulmonary edema, ascites, and gestational proteinuria have been attributed to capillary leak syndrome.⁵ Generalized edema is also frequently present in the preeclamptic parturient. Moreover, parturients with severe preeclampsia may exhibit increased symptoms because of a capillary leak. As can be seen in this case, symptoms such as these can make diagnosis of PPCM difficult because some parturients with preeclampsia may experience dyspnea, fatigue, and pedal edema. Furthermore a history of preeclampsia is frequently reported in cases of PPCM.¹ Additionally, oxidative stress, which is thought to contribute to or stimulate the pathophysiology associated with preeclampsia, is thought to be a key component in the development of PPCM.¹

• *Risk Factors for Peripartum Cardiomyopathy.* Common reported risk factors for PPCM are advanced maternal age, multiparity, multiple gestations, African American race, obesity, malnutrition, gestational hypertension, preeclampsia, diabetes, poor prenatal care, breast-feeding, cesarean delivery, substance and tobacco abuse, prolonged tocolysis, and family history.¹⁻³ Preeclampsia alone rarely leads to heart failure in healthy women,¹ but as demonstrated in this case, in which the patient's only risk factor was preeclampsia, PPCM can occur. The patient did have excessive weight gain during pregnancy, which possibly could have been an early sign of fluid retention secondary to PPCM; however, this is not an uncommon finding in parturients. It is therefore important to maintain a high level of suspicion to di-

agnose the rare cases of PPCM, as there are no specific criteria for differentiating subtle symptoms of normal late pregnancy from PPCM.^{1,3,7} A comparison of possible signs, symptoms, and diagnostic criteria between normal pregnancy, severe preeclampsia, and PPCM are presented in the Table.

• *Peripartum Cardiomyopathy vs Idiopathic Dilated Cardiomyopathy.* Clinical manifestations and physiologic changes of PPCM are similar to that of idiopathic dilated cardiomyopathy (IDC).² Injury to the myocardium is the initiating component that precipitates cell death. Initially, compensatory mechanisms, including sympathetic stimulation, the renin-angiotensin-aldosterone system, antidiuretic hormone production, release of atrial natriuretic peptide, tumor necrosis factor (TNF- α), and mechanical factors (ie, increased end-diastolic stretch on the ventricle), aid in maintaining cardiac output. If persistent and considerable damage occurs, the myocardium fails to generate sufficient contractile force to produce an adequate cardiac output. Congestive heart failure is, therefore, the most common manifestation. What distinguishes the 2 pathologies is the onset at which they present and their prognosis. Symptoms of late pregnancy, including increased blood volume, resting cardiac output, stroke volume, and HR, may all be a factor in peripartum heart failure in patients with IDC, particularly during the late second trimester when the greatest cardiovascular load of pregnancy exists.^{2,8} However, onset of PPCM symptoms predominantly appears later in pregnancy or postpartum. Prognostically, IDC takes on a long, protracted clinical course in contrast to PPCM, which results in immediate deterioration, as observed in this case.² Ultimately, PPCM remains a diagnosis of exclusion.⁹ Other causes of cardiomyopathy must be excluded prior to accepting a diagnosis of PPCM.⁹ Echocardiogram, electrocardiogram, and chest radiograph are required for the diagnosis of PPCM and should be considered in any parturient exhibiting signs and symptoms that may raise the suspicion of heart failure, such as paroxysmal nocturnal dyspnea, chest pain, cough, neck vein distention, new murmur, and pulmonary crackles.^{1,3}

• *Etiology of Peripartum Cardiomyopathy.* Although multiple hypotheses exist regarding the etiology of PPCM, such as abnormal response to the hemodynamic changes of pregnancy, viral infections, myocarditis, inflammatory cytokines, and nutritional deficiencies, the actual etiology remains unknown.¹⁻³ A recent review of the literature regarding PPCM revealed that myocarditis, cytokines, viral infections leading to myocarditis, and autoimmune factors are the most likely mechanisms leading to PPCM.⁹

• *Diagnostic Criteria for Peripartum Cardiomyopathy.* Current diagnosis of PPCM is based on the presence of 4 clinical criteria. They are: (1) the development of cardiac failure in the last month of pregnancy or within

Normal pregnancy	Severe preeclampsia	PPCM
Fatigue	Fatigue	Fatigue
Dyspnea	Respiratory distress due to capillary leak (crackles on auscultation)	Paroxysmal nocturnal dyspnea
Weight gain	Weight gain	Weight gain
Peripheral edema	Peripheral edema ^a Hypertension (> 160/110 mm Hg) Proteinuria (> 5 g/dL) Headache Blurred vision Seizures Hepatic tenderness 2nd subscapular hematoma HELLP syndrome Impaired coagulation Decreased creatinine clearance Increased uric acid Low to normal cardiac filling pressures High SVR Low, normal, or high CO LVH and dysfunction	Peripheral edema Cough Distended neck veins Chest pain Pulmonary edema Abdominal tenderness 2nd venous engorgement Thromboembolism New murmur Palpitations EF < 45% End-diastolic dimension > 2.7 cm/m ² BSA Fractional shortening < 30%

Table. Comparison of Normal Pregnancy, Severe Preeclampsia, and PPCM

Not all signs, symptoms, and diagnostic features may be present. Severe preeclampsia can develop after 20 weeks' gestation. PPCM can develop in the last month of pregnancy or within 5 months after delivery and lacks an identifiable cause of heart failure or recognized heart disease before the last month of pregnancy.

^a No longer part of diagnostic criteria, although often present.

Abbreviations: PPCM, peripartum cardiomyopathy; HELLP, hemolysis, elevated liver enzymes, and low platelet count; EF, ejection fraction; BSA, body surface area; SVR, systemic vascular resistance; CO, cardiac output; LVH, left ventricular hypertrophy. (Adapted from Selle et al,¹ Sibai and Stella,⁵ and Polley.⁶)

5 months of delivery, (2) the absence of another cause to which the cardiac failure can be attributed, (3) no signs or symptoms of heart disease prior to the last month of pregnancy, and (4) a left ventricle ejection fraction of less than 45%.^{3,9}

• *Treatment of Peripartum Cardiomyopathy.* Current treatment of parturients presenting with PPCM is similar to that of other patients with heart failure, with the notable exception that the fetus must also be protected from possible negative consequences of treatment. Treatment should be goal-directed and focused on optimizing hemodynamics, relief of symptoms, and treatment of precipitating factors, and, if possible, the implementation of long-term treatment plans.^{1,9} Generally, these treatment objectives can be met through the judicious use of diuretics to relieve pulmonary edema, afterload reduction to decrease the work of the heart, and control of hypertension that may be present.^{1,2,9} It is also important to differentiate between compensated and uncompensated heart failure. β -blockers, such as carvedilol, labetalol, atenolol, and metoprolol, have been safely used in pregnancy to control hypertension.¹ However, in the setting of acute decompensated heart

failure, they are contraindicated because of their negative inotropic and chronotropic effects and may actually worsen the heart failure.¹ Labetalol, which was administered to this patient shortly after admission, is commonly used to treat hypertension associated with preeclampsia; however, it may worsen cardiac function and contribute to pulmonary edema when associated with acute PPCM. Alternatively, hydralazine could have been used because of its afterload-reducing ability.

Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers are commonly used in the management of heart failure. However, they are contraindicated in pregnancy because of possible fetal death; nonetheless, they are considered to be safe during the postpartum period, even in mothers who are breast-feeding, and should strongly be considered to manage PPCM after delivery.¹ Hydralazine is the drug of choice for afterload reduction during the antepartum period and has a long and safe history of use to control hypertension in parturients.^{2,6} When combined with isosorbide dinitrate, hydralazine has been demonstrated to decrease mortality in patients with systolic dysfunction.¹⁰ Other potent vasodilators, such as nitroglycerin and nitroprusside, have

been used safely to reduce afterload during pregnancy; however, these medications are reserved for the most serious of cases when other methods of afterload reduction have failed and the mother or fetus is in jeopardy.^{2,10} Afterload reduction should be undertaken with great care, given the risk of deterioration of the fetal HR and a rapid and profound drop in maternal blood pressure. Continuous fetal monitoring is recommended if viability has been achieved.¹⁰ Anticoagulation with heparin can be used safely during pregnancy and postpartum to help reduce the incidence of thrombus formation. Warfarin is contraindicated during pregnancy and should be avoided until the postpartum period. Parturients with New York Heart Association Class III or greater symptoms of heart failure should be admitted to the hospital, and if diagnosis of PPCM is made during the antepartum period, urgent delivery may be indicated.¹⁰

• *Prognosis of Peripartum Cardiomyopathy.* Effective treatment helps women recover cardiac function and reduces morbidity and mortality, although there are limited prospective studies on the long-term prognosis of PPCM. In one series of 92 patients with PPCM, ejection fraction improved approximately 15% to 20% at 1 year; however, 72% of patients did not fully recover cardiac function at 5 years.¹¹ Subsequent pregnancies increase the risk of worsening cardiac function and possible death.⁹ Patients who have a history of PPCM and subsequently become pregnant require appropriate medical management and a multidisciplinary approach throughout pregnancy and delivery to improve the outcome for the mother and infant.¹²

• *Anesthetic Management of Peripartum Cardiomyopathy.* Anesthetic management for a parturient with PPCM depends on the severity of the cardiomyopathy and the patient's cardiopulmonary status. If the patient is unstable or is demonstrating signs of worsening pulmonary edema and impending respiratory failure, intubation may be required. Anesthesia providers should consider the urgency of the situation, maternal and fetal status, available equipment (ie, difficult airway equipment, hemodynamic monitoring), medication, and support personnel when deciding to intubate a parturient in distress. In this case, the patient rapidly decompensated soon after admission necessitating urgent intubation and emergency cesarean delivery. The decision was made to quickly transport the patient to the operating room because this provided a more controlled environment for intubation and preparation for an emergency cesarean delivery.

Anesthetic management should be based on the same principles used to manage any patient with severe cardiomyopathy; that is, the goals should be to reduce preload and afterload and improve contractility.¹³ The anesthetic plan should include the avoidance of myocardial depressants, careful fluid management to avoid worsening pulmonary edema, judicious use of diuretics and vasodi-

lators, maintenance of normal sinus rhythm with normal to low HR, and avoidance of wide swings in blood pressure.^{14,15} Nitroglycerin or nitroprusside may be needed to help accommodate for the autotransfusion of blood at the time of delivery.¹⁶ Arterial line placement is recommended to help meet these goals.¹³ Consideration should be given to placement of a pulmonary artery catheter and the availability of providers with experience in placement and management of transesophageal echocardiography if general anesthesia is required.¹⁶ A less invasive alternative to a pulmonary artery catheter is the FloTrac sensor (Edwards Lifesciences, Irvine, California), which bases its calculations on arterial waveform characteristics and stroke volume variation. When the FloTrac is combined with an arterial line and central line in patients undergoing mechanical ventilation, continuous cardiac output, stroke volume, stroke volume variation, and systemic vascular resistance can be determined.^{17,18}

For patients who are hemodynamically stable with no obstetrical indications for cesarean delivery, vaginal delivery may be attempted. In such cases, continuous epidural anesthesia or combined-spinal epidural anesthesia may be considered if the patient is not currently anticoagulated.^{14,15} Vaginal delivery minimizes blood loss, confers greater hemodynamic stability, avoids surgical stress, and reduces the risk of postoperative pulmonary complications and infection.¹⁵ If cesarean delivery is required, general anesthesia,^{12,19} epidural anesthesia,²⁰ combined spinal-epidural anesthesia,^{16,21} or continuous spinal anesthesia may be administered.¹⁶

Neuraxial anesthetic techniques have the advantage of reducing preload and afterload, and help minimize cardiac output fluctuations associated with vaginal and cesarean delivery. However, rapid onset of a sympathetic block with subsequent hypotension should be avoided. Several case reports have described hemodynamic stability and reliable anesthesia with the use of low-dose combined spinal-epidural anesthesia^{13,16,21} or continuous spinal anesthesia¹⁶ for cesarean delivery in patients with PPCM. With low-dose combined spinal-epidural techniques, 40% to 50% of the typical spinal dose of bupivacaine is administered followed by slow titration of epidural anesthesia with bupivacaine to achieve a T4 sensory level.^{13,16} Velickovic and Leicht¹⁶ reported hemodynamic stability with slow incremental administration of bupivacaine through a continuous spinal anesthesia catheter in a parturient with severe recurrent peripartum PPCM requiring cesarean delivery for twins. In contrast, significant hypotension has been reported with the administration of 5 mg of 0.5% hyperbaric bupivacaine with 0.3 mg diamorphine with a low-dose combined spinal-epidural technique in a parturient with severe PPCM.²² In this case the hypotension was easily treated with ephedrine, and an adequate surgical block was obtained with incremental administration of 10 mL of 0.5%

bupivacaine. These case reports indicate that low-dose combined spinal epidural and continuous spinal anesthesia can be safely administered with appropriate invasive monitors and early treatment of hypotension for elective cesarean delivery in parturients with PPCM.

If general anesthesia is required for a patient with known or suspected PPCM, the principles should be similar to those used for any patient with heart failure. Several case reports have described the use of modified rapid sequence induction using thiopental, sufentanil, and succinylcholine¹⁹ or using propofol and succinylcholine,¹² followed by maintenance with volatile anesthetics, or use of total intravenous anesthesia with a target controlled infusion of propofol and remifentanil.²³ The short half-life of remifentanil has the advantage of being easily titrated and has minimal impact on the neonate. If longer acting narcotics are required prior to delivery, the neonatologist should be informed and be prepared for neonatal resuscitation and the possible administration of naloxone.¹⁹

Conclusion

Although PPCM is a relatively rare disease process, this case demonstrates the need for vigilance and a high index of suspicion for PPCM in parturients with subtle symptoms of heart failure, such as shortness of breath and edema. A thorough history and physical examination are essential in differentiating preeclampsia from PPCM. Early diagnosis, intervention, and treatment may prevent or, at least, lessen symptoms of PPCM and improve maternal and fetal outcomes. When faced with an acutely decompensating parturient requiring immediate intubation, the anesthesia provider should call for help early, and consider transport to an operating room, which provides a more controlled environment for airway management and preparation for an emergency cesarean delivery. Anesthetic management principles are similar to those used to manage patients with cardiac failure and should be based on the severity of the cardiomyopathy. If PPCM is suspected, a multidisciplinary team that includes anesthesia providers, obstetricians, cardiologists, and critical care specialists will be needed to help aid in the diagnosis and management.

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