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Case Report

Pulmonary Hypertension Crisis in the Immediate Postpartum Period

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Abstract

Pulmonary arterial hypertension (PAH) is one of the most complex, highest risk conditions in pregnancy [1]. The physiologic changes of pregnancy stress the cardiopulmonary system, especially in a patient with PAH, with significant risk of right heart failure and even death [2, 3]. Maternal mortality is historically cited greater than 30%, though more recent reviews have demonstrated lower maternal mortality rates [4, 5]. Emerging therapies and multidisciplinary care are likely contributors, though treatment remains challenging. We present a patient with PAH who presented at 27 weeks with decompensated heart failure. She received prostacyclin therapy in the ICU before delivery at 28 weeks for preeclampsia with severe features and worsening fetal status. The immediate postpartum course was complicated by a brief period of pulmonary artery pressures that superseded the systemic blood pressure requiring vasopressors, though no acute events occurred. The case demonstrates the physiologic change that occurs in the immediate postpartum period, necessitating expert, multidisciplinary care for safe management of PAH in pregnancy.

Keywords: Pulmonary arterial hypertension; Cardiac disease in pregnancy; Prostacyclin therapy

Introduction

Pulmonary arterial hypertension (PAH) is a complex, high-risk condition in pregnancy for which the updated 2025 modified WHO classification of maternal cardiovascular risk stratification (mWHO 2.0) suggests consideration of pregnancy termination [1]. The normal physiologic changes of pregnancy increase plasma volume and cardiac output but decreases systemic vascular resistance [6]. In PAH, the cardiovascular system is ill-equipped to adapt to these changes, and can lead to right heart failure, cardiogenic shock and risks maternal death [6,7]. Maternal mortality in cases of PAH is historically cited greater than 30%, though more recent reviews demonstrate lower maternal mortality rates [4,5]. Emerging therapies and multidisciplinary care are likely contributors, though management during pregnancy remains challenging.

Case Presentation

We describe a case of severe pulmonary arterial hypertension (PAH) in pregnancy. A 28-year-old G2P0010 at 27 week gestation was transferred from an outside facility to our labor and delivery for a higher level of care due to symptoms of volume overload Her medical history was significant for rheumatic heart disease, with subsequent development of severe mitral valve stenosis and PAH. Prior to pregnancy, she received care in the Philippines, where she lived, with angiotensin receptor blocker and diuretic (telmisartan/hydrochlorothiazide), which she self-discontinued upon learning she was pregnant. After immigrating to the United States, she established care with Cardiology and Maternal Fetal Medicine. She was hospitalized at 20 week gestation for symptoms of volume overload, at which time pregnancy termination was discussed but declined. She was started on furosemide and sent to our institution for specialized multidisciplinary management.

On presentation to our hospital, she reported worsening lower extremity edema, orthopnea, and dyspnea on exertion. Vital signs included a blood pressure 109/48 mmHg, heart rate of 95 beats per minute, and respiratory rate 17 breaths per minute. Physical examination was notable for 3+ pitting edema extending to the knees, and a 3/6 holosystolic murmur: lungs examination was clear to auscultation bilaterally. Transthoracic echocardiography demonstrated preserved left ventricular ejection fraction of 58% with severe mitral stenosis and regurgitation, moderate tricuspid regurgitation, and severely dilated right atrium. Obstetric ultrasound revealed severe fetal growth restriction (<1%ile) with intermittent absent end diastolic flow on umbilical artery dopplers. Cardiology and pulmonary hypertension teams were consulted. She underwent right heart catheterization which revealed pulmonary artery pressure of 79/43 mm Hg (mean 59). The patient was admitted to the intensive care unit for initiation of epoprostenol therapy and aggressive diuresis with a net negative fluid balance goal of 1-2 liters daily. A multidisciplinary meeting was convened, during which Pulmonology recommended delaying delivery as long as possible to optimize pulmonary hemodynamics. From an obstetric standpoint, progressive fetal compromise was identified as the primary limitation to prolonging pregnancy. With respect to the mode of delivery, cesarean section was determined to be the safest approach given high likelihood of fetal intolerance of labor, and the substantial maternal risk associated with urgent or emergent intrapartum delivery.

Her ICU course was complicated by hypoxia requiring supplemental oxygen, and preeclampsia with severe features due to elevated blood pressures and thrombocytopenia (83,000 plt/uL). Given the early gestational age and the potential side effect of thrombocytopenia related to epoprostenol, plan was to expectantly manage pregnancy. However, on day #3 of the epoprostenol drip, fetal heart tracing demonstrated multiple prolonged late decelerations. Repeat umbilical artery Dopplers showed persistent absent end diastolic flow. The decision was made to proceed with delivery to avoid an emergency. On the following day, at 28 weeks and five days, the patient was taken to the OR for cesarean delivery.

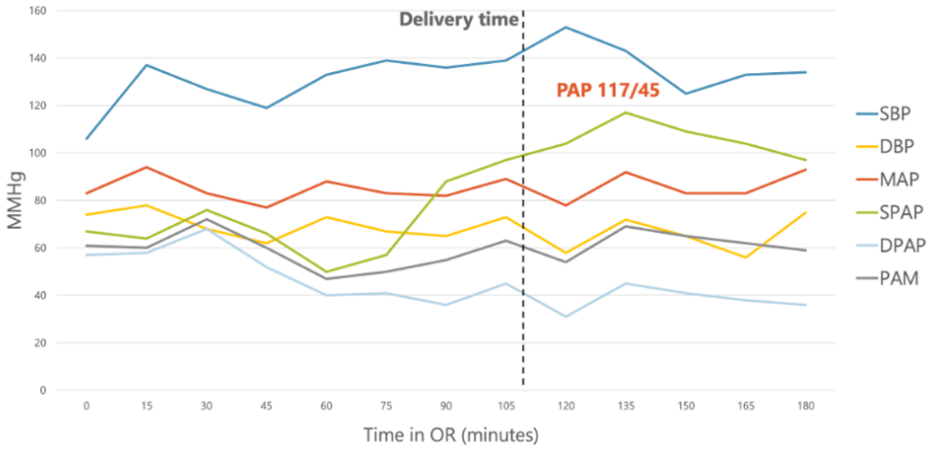

Figure 1: Intraoperative systemic and pulmonary artery blood pressures as recorded by arterial line and right heart catheter, respectively.

The patient was pre-cannulated with arterial femoral catheters prior to surgery anticipating the potential need for extracorporeal membrane oxygenation (ECMO). Both cardiac and obstetric anesthesia teams were co-managing intra-operatively. She underwent a classical primary cesarean section with an estimated blood loss of 400mL. Fetus weighed 778g and had Apgars 8 and 9 (1 and 5 minutes). Immediately after delivery, pulmonary artery pressures were noted to abruptly rise to 117/45 mmHg (mean 69 mmHg). Vasopressors were initiated to improve systemic pressures to the 140s-150s/60's-70s mmHg. She received intravenous diuretics with improvement in hemodynamic profile. There were no other acute events in the operating room. The patient was brought back to the medical ICU for postpartum care. She was continued on intravenous epoprostenol and boluses of diuretics. The femoral artery catheters were removed while in the ICU and right heart catheter was removed on postpartum day #4. Hemodynamic support with aggressive diuresis resulted in 30 pound weight loss since the initial admission at 27 weeks. She received a contraceptive implant prior to discharge. She remained inpatient on the medicine service for additional time to optimize outpatient pulmonary hypertension therapy.

Discussion

This case illustrates how normal physiologic changes in pregnancy - and particularly in the immediate postpartum period - can have profound consequences in patients with PAH. Following delivery, abrupt hemodynamic occur due to autotransfusion of uteroplacental blood and relief of caval compression, resulting in a marked increase in plasma volume and cardiac output [1]. In patients with PAH, these changes can lead to a rapid and dangerous rise in pulmonary artery pressures.

In this case, pulmonary artery pressures increased dramatically immediately after delivery, reaching levels comparable to systemic arterial pressures. Anticipation of this high-risk period allowed for extensive multidisciplinary preparation, including continuous pulmonary vasodilator therapy, aggressive diuresis, and use of systemic vasopressors to preserve adequate perfusion of both the pulmonary and systemic circulations. These interventions were critical.

Without them, the patient very likely would have suffered right heart failure with cardiogenic shock and ultimately death.

Conclusion

Pulmonary arterial hypertension carries significant risk for both maternal and fetal morbidity and mortality. Despite these risks, the number of women with PAH with viable pregnancies has increased in recent years, necessitating further research and guidelines for care. The postpartum period is a particularly delicate time. Our case demonstrates how physiologic changes of pregnancy can be detrimental to patients with PAH, underscoring the importance of discussing risks of pregnancy, recommending termination, and in cases of viable pregnancies, referring to expert care. Maternal fetal medicine, pulmonary hypertension specialists, cardiology, neonatology, and specialized anesthesia teams were all significant contributors to the positive maternal and fetal outcomes in this case. Multidisciplinary and highly specialized care are essential to the care of PAH in pregnancy.

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