
Acute Pulmonary Edema in a Pregnant Patient with Undiagnosed Levo-Transposition of the Great Arteries

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Abstract

Challenging differential diagnosis

Levo-transposition of the great arteries (L-TGA) is a rare form of congenital heart disease that may go unrecognized until adulthood. Parturient women with L-TGA have a high likelihood of developing acute pulmonary edema and cardiac dysrhythmias during the peri-partum period. We present the case of a 32-year-old primigravida patient with previously unknown diagnosis of L-TGA, presenting with preeclampsia, whose peri-partum course was complicated by the development of acute pulmonary edema, complete heart block, and acute hypoxic respiratory failure. This case report highlights the predisposition of parturient women with undiagnosed congenital heart disease to develop acute hypoxic respiratory failure in the perioperative period and the importance of meticulous fluid management in such patients.

Keywords: Heart defects; Congenital; Heart failure; Pregnancy complications; Cardiovascular

Introduction

Pulmonary edema is a life-threatening event during pregnancy and continues to be associated with significant morbidity and mortality [1]. The physiological changes of pregnancy place a parturient woman at a higher risk of decompensation in the setting of acute pulmonary edema. The common causes of pulmonary edema during pregnancy include; administration of tocolytics, sepsis, pre-existing cardiac disease, fluid overload, preeclampsia and pregnancy-associated cardiac disease [2,3]. The presence of a combination of these factors possibly increases the likelihood of developing acute pulmonary edema and respiratory failure during the peri-partum period. In this report, we describe the clinical course of a primigravida patient who developed acute hypoxic respiratory failure during labor from a combination of fluid overload and preeclampsia in the setting of an undiagnosed pre-existing heart disease.

Case Description

A 32-year-old female, gravida 1, para 0, with no significant past medical history and normal physical examination except for class 1 obesity (BMI of 32.54kg/m²) presented for induction of labor for preeclampsia at 37 3/7 weeks of gestation. Preeclampsia was diagnosed on the basis of elevated blood pressure readings and a high urine protein creatinine ratio on routine antenatal visits.

Labor was induced using sublingual misoprostol 25 mcg and augmented with an oxytocin infusion started at 0.24 U/h and ranging from 0.04 U/h to 1.2 U/h. Her labor progressed to a cervical dilation of 3 cm, and she was noted to have increased blood pressure, with systolic blood pressure persistently higher than 160 mm Hg. A 6-gram intravenous (IV) bolus dose of magnesium sulfate was administered followed by an infusion ranging between 1.5 to 2.0 g/h. Her hypertension persisted, and a dose of IV labetalol 10 mg was administered. This resulted in a precipitous fall in her blood pressure, accompanied with intermittent fetal category 2 tracings with late decelerations. She received 3 fluid boluses (500 mL each) of lactated ringer's solution over the next 4 hours, a change in position to lateral decubitus, and supplemental oxygen leading to resolution of decelerations. She maintained good urine output (>0.5 mL/kg/h) throughout the duration of labor.

An epidural catheter was placed for labor analgesia at the L3–L4 interspace per patient's request 46 hours after induction of labor. She tolerated the procedure well and reported satisfactory analgesia from the epidural infusion consisting of 0.125% bupivacaine with fentanyl 2 mcg/mL administered at 6 mL/h. Seven hours after induction of labor analgesia, the patient developed dyspnea, hypoxemia, and had bilateral rales, on pulmonary examination. Supplemental oxygen was administered via a non-rebreather mask and her oxygen saturation stayed in the mid-90s. Acute pulmonary edema from fluid overload was suspected and a decision was made to proceed with urgent cesarean delivery. Furosemide 10 mg IV was administered, which was followed by brisk diuresis.

The epidural catheter was bolused incrementally with a solution comprising of lidocaine 2% with 1 mL of 8.4% sodium bicarbonate for a final volume of 20 mL. A sensory block level up to T4 dermatomal level was achieved bilaterally. After delivery of the fetus, uterine atony was noted which was treated with an infusion of IV oxytocin totaling 20 IU. The atony persisted leading to administration of intramuscular (IM) carboprost tromethamine 250mcg and methylergonovine 200 mcg IM, which eventually led to improved uterine tone. The estimated blood loss for the case was 800 mL and the urine output was 150mL. The patient received 2.2 L of crystalloids during the surgery. An additional 20 mg of IV furosemide was given at the end of the surgery. The baby had Apgar scores of 8 and 9 at 1 minute and 5 minutes respectively and was transferred to the newborn nursery in stable condition. Post-procedure, the patient became acutely hypoxemic with altered mental status. Her oxygen saturation decreased to 85% and rapid sequence intubation of the trachea was performed with propofol 150mg IV and succinylcholine 100 mg IV. The patient was then transferred to the surgical intensive care unit (SICU).

On admission to SICU, the patient's 12 lead electrocardiogram (ECG) revealed complete heart block, which was not noticed during the surgical procedure. She was hemodynamically stable and did not require pacing. Her chest x-ray showed features consistent with pulmonary edema (Figure 1).

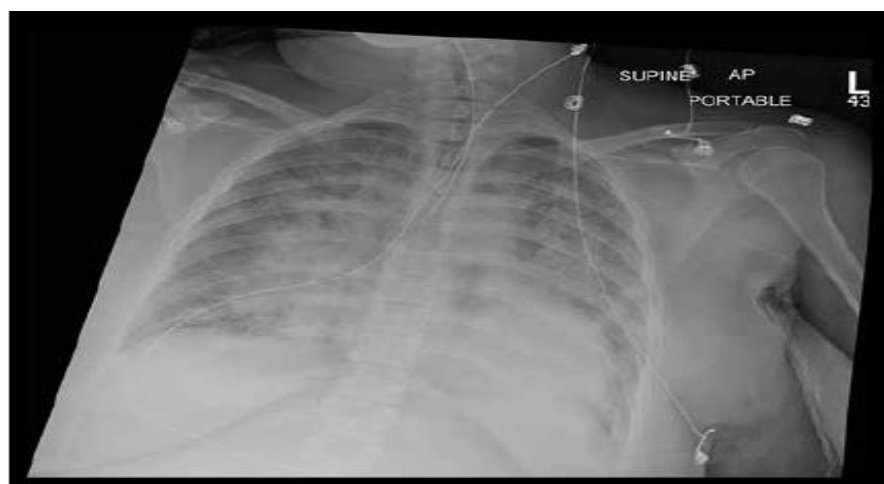


Figure 1: Chest x-ray of the patient showing bilateral fluffy infiltrates indicating a diagnosis of acute pulmonary edema.

She received an additional 20 mg of IV furosemide along with positive pressure ventilation with a positive end-expiratory pressure of 8 cm H₂O. A transthoracic echocardiogram showed L-TGA with a systemic right ventricle and ejection fraction approximately 45% to 50% with right ventricular hypertrophy (Figure 2). Her blood pressure remained stable and her trachea was extubated the next day, after meeting necessary criteria. A repeat 12 lead ECG revealed junctional rhythm. The patient was discharged from the SICU on post-operative day 2 and was discharged from the hospital on post-operative day 4.



Figure 2: Echocardiogram of the patient showing L-transposition of the great arteries with swap of the left and right ventricles. The left atrium (LA) drains into the right ventricle (RV) and the RV ejects into the systemic circulation via the aorta, while the right atrium (RA) drains into the left ventricle (LV) ejecting blood into the pulmonary circulation via the pulmonary artery.

Discussion

This report illustrates the development of acute pulmonary edema and hypoxic respiratory failure in a parturient patient with undiagnosed congenital heart disease when exposed to the hemodynamic stress of labor and delivery. The presence of preeclampsia and fluid overload likely added to the hemodynamic insult on an at-risk heart. Our patient was otherwise healthy with a normal functional capacity at baseline and hence a diagnosis of a pre-existing cardiac condition was never suspected.

She was adequately being treated with magnesium sulfate and anti-hypertensives for preeclampsia. Incremental fluid boluses were administered with close observation of hemodynamics to treat fetal decelerations. The recommended interventions for treatment of fetal insufficiency is fluid loading along with position change and delivery of oxygen [4]. We feel that it was the combination of preeclampsia, fluid overloading, and failing of the systemic right ventricle (RV) from L-TGA which led to the development of acute pulmonary edema. The administration of methylergonovine potentially exacerbated her condition, contributing to the development of acute respiratory failure [5]. Oxytocin has also been shown to cause acute pulmonary edema in parturient women from water intoxication [6,7] and could have had an additive effect. However, in the setting of acute blood loss from uterine atony, use of uterotonics such as oxytocin and ergot alkaloids are recommended [8]. Caution should be exercised during their administration and the patient closely monitored.

L-TGA (also known as congenitally corrected transposition of the great arteries) is a rare form of congenital heart disease (CHD) accounting for 0.02 to 0.07 per 1000 live births [9,10]. Characteristic features of L-TGA include a left and right ventricles swap with the RV ejecting blood into the systemic circulation via the aorta, and the left ventricle (LV) ejecting blood into the pulmonary circulation [11]. Most of these patients remain asymptomatic as the RV hypertrophies and provides adequate cardiac output. However, in conditions of increased afterload or preload, as in our patient's case, the hypertrophied RV can fail to maintain adequate cardiac output, leading to the development of acute pulmonary edema.

It is possible that in our patient's case, the development of a new complete heart block contributed towards the development of acute pulmonary edema. The anatomy of the conduction system is abnormal in patients with L-TGA, with the right ventricle (AV) node not being able to connect normally to a penetrating AV conduction bundle [11]. This leads to the presence of a second AV node positioned anteriorly, which gives rise to an elongated AV bundle [12]. This change in anatomy predisposes these patients to developing heart block and reentry tachycardias [11]. We are unsure if our patient had a complete heart block throughout labor or whether it coincided with the development of acute pulmonary edema. Since the patient had no significant medical history, an ECG was not done in the preoperative period. During surgery, the ECG tracing on the monitor did not show a complete heart block, but the accuracy of detection of a new arrhythmia with telemetry is limited. Our patient did not require pacing because her junctional rhythm was able to maintain adequate perfusion.

The management of hypotension and fetal insufficiency in parturient women with preeclampsia can be challenging. Judicious administration of IV fluids is advised but it is debatable if absolute fluid restriction should be applied due to the fear of an underlying heart disease. Further studies are required to assess if the utilization of pressors instead of fluids should be the first-line treatment of patients with hypotension and signs of fetal insufficiency.

Conclusion

We present this case of a parturient woman who developed hypoxic acute respiratory failure in the peripartum period triggered by an episode of acute pulmonary edema. We believe that it was the combination of acute systolic heart failure (in a congenitally malformed heart) and fluid overload in the setting of preeclampsia which led to such a presentation. Such presentations are rare and can be associated with significant morbidity and mortality. This case report exemplifies the need for judicious administration of IV fluids in all parturient women, especially with preeclampsia, and warns the physicians of possible underlying heart disease that might manifest for the first-time during labor.

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