

Case Report: Anesthetic Management for A Pregnant Patient with Pulmonary Hypertension and Eisenmenger Syndrome

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ABSTRACT

Pregnancy in women with heart disease is associated with a higher risk of deterioration of the mother's cardiac status and adverse pregnancy outcomes, including complications such as arrhythmias, premature birth, fetal growth restriction, and a significant risk of maternal and fetal mortality. Few heart conditions pose a massive risk to the mother's life; these include Eisenmenger syndrome, pulmonary hypertension with right ventricular dysfunction, and Marfan syndrome. We present the case of a 34-year-old female patient with a history of congenital heart disease and Eisenmenger syndrome of 13 years' duration without medical treatment. She presented to the emergency department at 38 weeks' gestation with dyspnea on mild exertion, perioral cyanosis, arrhythmic heart sounds, a hyperdynamic precordium, and diastolic tricuspid and pulmonic murmurs. An emergency cesarean section was performed, resulting in termination of the pregnancy. with anesthetic management with fractional epidural block without success changing anesthetic technique to balanced general anesthesia, discharged patient to ICU orointubated, coupled to mechanical ventilator, without vasopressor support at the time, where she presents a torpid evolution requiring vasopressors without success, until death.

KEYWORDS: Pregnancy, Eisenmenger syndrome, maternal mortality, anesthesia management,

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INTRODUCCIÓN

Pulmonary hypertension is defined as a mean pulmonary artery pressure >20 mmHg at rest, measured by right heart catheterization.⁽¹⁾ It has a prevalence of 15–50/1,000,000 people and affects individuals of all ages and races, more frequently in women than in men. The causes are multifactorial; however, the most common is left heart disease (up to 78.3%), the most serious complication of which is Eisenmenger syndrome, which is a pulmonary-systemic shunt accompanied by cyanosis and increased pulmonary pressures secondary to pulmonary vascular pathology.⁽²⁾

Pulmonary hypertension can present in various clinical forms, classified as: pulmonary arterial hypertension, pulmonary arterial hypertension due to left heart disease, pulmonary hypertension due to pulmonary disease or hypoxemia, chronic thromboembolic pulmonary hypertension, and other obstructions of the pulmonary arteries, and of unknown or multifactorial mechanism. These processes generate

hypertrophy and hyperplasia of the vascular wall as well as vasoconstriction.⁽³⁾ In patients with a left-to-right shunt, there is a progressive increase in pulmonary vascular resistance and right ventricular pressure, ultimately resulting in reversed blood flow, leading to Eisenmenger syndrome as the most advanced outcome of pulmonary arterial hypertension.⁽⁴⁾

In pregnant patients, heart disease is associated with mortality rates of approximately 30% for patients with pulmonary hypertension, 36% in Eisenmenger syndrome, and 56% secondary to other conditions.⁽⁵⁾ The presence of Eisenmenger syndrome is also associated with a 40% miscarriage rate, premature birth in half of the cases, and maternal mortality in 50%, with the highest mortality occurring during the peripartum period, delivery, or the first week postpartum. Timely diagnosis and appropriate medical care focused on managing heart disease during pregnancy reduce potential maternal and fetal complications by establishing comprehensive and interdisciplinary treatment

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that supports the patient not only during pregnancy but also in the postpartum period. ⁽⁶⁾

CLINICAL CASE

We present the case of a 34-year-old female patient with a history of undiagnosed heart disease for the past 13 years, without medical treatment. Menarche at age 12, first sexual intercourse at age 16, regular 30x5 cycles, no menstrual history, G3, P0, C1, A1. Interpregnancy interval 13 years.

She attends the gynecology service for the first time on July 12, 2024, where she is diagnosed with a pregnancy of 28.1 weeks by ultrasound on June 24, 2024, with no labor and laboratory tests on June 25, 2024, which revealed a platelet count of 95,000 with a manual count of 102 platelets. A consultation with hematology was performed on August 29, 2024, which found no abnormal immature or neoplastic cells at any level, leading to a diagnosis of gestational thrombocytopenia and a recommendation to admit the patient for close monitoring of platelet count and relative rest. However, the patient decided not to be admitted of her own accord.

She returned to the emergency department on 09/02/2024 with dyspnea on exertion and with SaO₂ at 90% in ambient air. Paraclinical tests were performed, highlighting a decrease in platelet count to 36, so a consultation with hematology was requested, who suggested admitting the patient for daily blood counts for close monitoring.

The patient remained hospitalized for 5 days under the care of the gynecology department; however, she progresses slowly, presenting with oxygen desaturation 80% in ambient air and mild exertional dyspnea. Therefore, an internal medicine consultation was requested on September 2, 2024, who reported finding the patient with acropachy, clubbed fingers (drumstick fingers), and a grade III systolic murmur at the tricuspid focus, suggesting her admission to the Intensive Care Unit (ICU)

The ICU was consulted and ordered an electrocardiogram, which revealed right heart chamber enlargement. An echocardiogram and cardiology evaluation were also requested. The patient was admitted under their care with a diagnosis of 35.4 weeks gestation (based on a second-trimester ultrasound), gestational thrombocytopenia, and probable cyanotic heart disease (to be determined).

A transthoracic echocardiogram was performed on 03.09.2024, which reported dilation of the right chambers, preserved left ventricular systolic function with an LVEF of 54%, global right ventricular systolic dysfunction (RVFD 34%), non-assessable left ventricular diastolic function, no significant valvular disease, pulmonary artery systolic pressure of 121 mmHG, and persistence of a hypertensive ductus arteriosus with a right-to-left shunt and Eisenmenger syndrome.

A patent ductus arteriosus (PDA) was observed with a 14 mm pulmonary orifice and a 5 mm aortic orifice, exhibiting a right-to-left shunt, Eisenmenger syndrome, ambient oxygen saturation of 78%, and a hypertensive ductus arteriosus with no possibility of closure.

After one day in the ICU, a complete diagnosis was made: congenital heart disease of the patent ductus arteriosus type, hypertension with a right-to-left shunt, Eisenmenger syndrome, right heart chamber dilation, and a 35.5-week pregnancy with normal progression. It was decided to discharge the patient from the ICU service and admit her to the gynecology service, which stated that she should remain under close surveillance with continuous monitoring due to the high risk of hemodynamic decompensation, pending acceptance to a tertiary care unit. However, the patient decided to voluntarily leave the service despite medical recommendations and extensive information regarding possible consequences and complications

The patient attended to her third consultation at the emergency department on September 25, 2024, presenting with regular abdominal pain associated with uterine contractions, with ENA 8/10 and reporting severe dyspnea. Upon arrival at the emergency department, her vital signs were BP 102/87 mmHg, HR 105 bpm, RR 22 bpm, SaO₂ 82%, temperature 36.5°C, she was neurologically intact, Physical examination revealed pallor of the skin, slight perioral cyanosis, arrhythmic heart sounds, a hyperdynamic precordium in S3 or S4, with diastolic tricuspid and pulmonic murmurs, capillary refill time of 3 seconds, edema of the extremities and clubbing of the fingers. Therefore, an admission diagnosis was made of pregnancy of 38 weeks gestation by Capurro + Satisfied Parity + Eisenmenger phenomenon + thrombocytopenia. Severe pulmonary hypertension. Evaluated by the gynecology and internal medicine services, who recommend urgent termination of pregnancy due to exacerbation of heart disease

The patient was admitted to the operating room, type 1 monitoring was performed, with the following vital signs on admission: BP 180/97mmHg, HR 71 bpm, SatO₂ 80%, generalized pallor of the integuments, peripheral cyanosis, hypoventilated lung fields, lower limb edema ++. Supplemental oxygen was administered with a reservoir mask at FiO₂ 60%, and an epidural block was performed by positioning the patient in the left lateral decubitus position. It was administered

112 mg (15 ml) of 7.5% ropivacaine in a divided manner through an epidural catheter. A latency of 15 minutes was allowed; however, adequate analgesia and anesthesia were not achieved, so it was decided to change the anesthetic technique to balanced general anesthesia. A live, male infant was delivered, with a single nuchal cord, 38 weeks gestation by Capurro scale, vigorous tone, weighing 2900 grams, measuring 50 cm in length, Apgar score 8/9, Silverman score 0/0. It was used as a uterotonic Oxytocin 5 IU IV, achieving

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adequate uterine tone. The surgical-anesthetic procedure concluded with an estimated blood loss of 500 ml and urine output of 300 ml. As the patient did not meet extubation criteria, an evaluation was requested by the intensive care unit. The patient was admitted to their ward and placed on mechanical ventilation. An arterial blood gas analysis was performed, yielding the following results: pH 7.0, PCO₂ 28.3, PO₂ 38.5, Na 127, K 4.71, HCO₃ 19, lactate 1.7, PO₂/FiO₂ 96.3, Hb 13.9.

After 3 hours, mechanical ventilation was discontinued, and the patient was maintained on oxygen therapy via a non-rebreather mask with an FiO₂ of 70%. The following vital signs were reported: BP 133/80 mmHg, HR 90 bpm, RR 20 rpm, SaO₂ 80%, MAP 70.

After 12 hours, the patient's condition deteriorated, with Sao₂ below 70% and increased ventilatory effort, so it was decided to re-intervene on the airway by performing orotracheal intubation under direct laryngoscopy coupled with volume-controlled mechanical ventilation with FiO₂ at 100%, PEEP 9, RR 20, VT 300, achieving a maximum saturation of 60%. Vital signs were BP 90/40 mmHg with MAP 50 mmHg, so it was decided to use a vasopressor such as norepinephrine at 0.25 mcg/kg/min and dobutamine at 3 mcg/kg/min. However, the patient's clinical condition deteriorated with severe symptomatic bradycardia. A code blue was activated, and 1 mg of atropine was administered without improvement in heart rate. Two additional doses were repeated with severe refractory bradycardia, and the patient subsequently went into sudden cardiopulmonary arrest. Advanced cardiopulmonary resuscitation was initiated according to updated ACLS guidelines. Seven cycles of resuscitation were performed with three doses of epinephrine without return of spontaneous circulation. The cause of death was declared to be refractory cardiogenic shock and another congenital heart malformation.

DISCUSSION

Pulmonary hypertension is a clinical disorder defined by elevated pulmonary arterial pressure that affects about 1% of the adult population ⁽⁷⁾ and although its biopathology is complex and remains under study, it has been established that it can be idiopathic; associated with chronic diseases such as left heart failure, lung parenchymal diseases and/or thromboembolic diseases; hereditary due to genetic defects and alterations of protein receptors such as the bone morphogenetic protein receptor type 2, due to a defect in a member of the transforming growth factor β (TGF- β) signaling family, due to mutations in the kinase, in the activin receptor type 1, or endoglin; and it can be caused by drugs or toxins (anorexigenics fenfluramine and dexfenfluramine, methamphetamines which represent up to 18% of PAH). ⁽⁸⁾ There are currently six types of classification for pulmonary hypertension: passive, hyperkinetic, obstructive, obliterative, vasoconstrictor and polygenic, the current classification

overlaps with the scheme of the British cardiologist Dr. Paul Wood who gathered the first data in his cohort research at National Heart and Brompton hospitals, seminal descriptive publications on the "primary pulmonary hypertension" by Castleman and Bland (1946) ⁽⁹⁾

The initial evaluation of patients with suspected pulmonary hypertension includes an electrocardiogram (ECG), in which the pattern may indicate dilation right atrial enlargement or right ventricular hypertrophy (R:S elevation greater than 1 in lead V1 without other causes, or if the R wave amplitude in lead V1 is greater than 7 mm), deformation of the RV (ST segment depression in V1 to V3); ⁽¹⁰⁾ a chest x-ray which is abnormal in 90% of patients presenting dilation of the central pulmonary artery, peripheral devascularization and dilation of the right atrium and RV; and a transthoracic echocardiogram, which can provide quantitative data such as systolic displacement of the tricuspid annular plane, right atrial area index, myocardial performance index (TEI), change in fractional area of the right ventricle, cavity dilation, and right ventricular hypertrophy ⁽¹¹⁾.

In our case, it was not possible to determine the origin of the pulmonary hypertension, as this would have required multiple tests and laboratory examinations that were not performed. However, our patient's electrocardiogram showed right ventricular hypertrophy and ST segment depression in V1, and the chest X-ray revealed a dilated pulmonary artery. and the transthoracic echocardiogram confirmed dilation and hypertrophy of the right chambers, global systolic dysfunction of the right ventricle with FACVD 34%, persistence of the hypertensive ductus arteriosus with right-to-left shunt with Eisenmenger syndrome, pulmonary artery systolic pressure 121mmHG

Clinically, the presenting symptoms of pulmonary arterial hypertension are usually dyspnea, fatigue, abdominal distension, lower extremity edema, weakness, exercise limitation, or bendopnea. Angina pectoris (caused by right Ventricle ischemia or compression of the left main coronary artery) and syncope (caused by a severe decrease in cardiac output), those are high-risk presenting symptoms that constitute a pulmonary arterial hypertension emergency. Although there is no classic physical examination for pulmonary hypertension, those that indicate the presence or consequences of right-sided pressure and volume overload should be monitored. In severe cases, a right-sided S3 heart sound, right ventricular elevation, increased jugular venous pressure, and pulsatile liver may be observed ⁽¹²⁾. In the case presented, the patient reported dyspnea, fatigue, and edema of the extremities, which may be inherent to her pregnant condition. Similarly, physical examination revealed S3 and grade I jugular venous distension and clubbing of the fingers, a sign present in some heart diseases.

Eisenmenger syndrome is a systemic pulmonary shunt accompanied by cyanosis and increased pulmonary pressures secondary to pulmonary vascular disease that can develop

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from any intracardiac communication where pressures are high. It has a survival rate of 80% at 10 years after diagnosis and 42% at 25 years. This diagnosis should be considered in any cyanotic patient and should be confirmed by ultrasound, which will show increased pulmonary pressure and intracardiac shunting. Treatment is nonspecific and consists of supportive care and avoiding situations that could decompensate the patient, such as surgery or pregnancy ⁽¹³⁾ In 1979, Gleicher et al. analyzed published cases of Eisenmenger syndrome during pregnancy, evaluating 70 pregnancies in 44 women; 52% of them died during pregnancy; 30% of pregnancies ended in the death of the mother. The risks of death associated with the first, second, and third pregnancies were 36%, 27%, and 33%, respectively. A successful first pregnancy did not guarantee safety in subsequent pregnancies. ⁽¹⁴⁾ Seventy percent of deaths occurred during delivery or during the week following delivery, with the causes being excessive bleeding in 35% of cases and thromboembolism in 44% of cases.

Maternal mortality associated with cesarean delivery was 80%, while for vaginal delivery it was 34%. Ninety-six percent of deaths occurred within the first 15 days after delivery. No maternal deaths were recorded when pregnancy was terminated during the first trimester. Only 26% of pregnancies reached full term, 55% were preterm, and 32% were small for gestational age. Fetal complications are high and include premature birth in 85-88% of cases, small for gestational age in 83%, fetal mortality in 8-27%, and neonatal mortality in 25%. ⁽¹⁵⁾

The World Health Organization uses four categories determined by diagnosis: class I, mild, uncomplicated pulmonary stenosis; class II, unoperated atrial septal defects or ventricular septal defects and reconstructed tetralogy of Fallot; class III, mechanical valve prostheses, systemic right ventricle, Fontan circulation, unrepaired cyanotic heart disease, other complex congenital heart disease, Marfan syndrome with an aorta of 40-45 mm; and class IV, pulmonary hypertension/Eisenmenger syndrome, systemic ejection fraction of less than 30%, systemic dysfunction in NYHA classes III-IV, severe mitral stenosis, and severe symptomatic aortic stenosis. ⁽¹⁶⁾

In this case, neuraxial blockade was chosen as the first anesthetic technique since the main objective of anesthetic management in a pregnant patient with heart disease is to maintain systemic vascular resistance as stable as possible, avoid aortocaval compression, hypoxemia, hypercavitary stress, and acidosis, which are factors that can increase pulmonary pressure ⁽¹⁷⁾. It was administered 15 ml of 7.5% ropivacaine at the L2-L3 level. After latency, a deficit in motor and sensory block was observed, so it was decided to change the anesthetic technique to balanced general anesthesia.

In patients with Eisenmenger syndrome, the goal of anesthesia is to maintain cardiac output without worsening

the intracardiac shunt. Regional anesthesia can reduce systemic vascular resistance proportionally to the extent of sympathetic blockade. As a result, the right-to-left shunt increases. However, general anesthesia also offers advantages such as the ease with which nitric oxide can be administered and the ability to regulate CO₂ and oxygen levels, thus minimizing pulmonary vasoconstriction. Nevertheless, this technique can increase pulmonary vascular resistance due to the release of catecholamines during laryngoscopy. A review covering the years 1966 to 2002 affirms the safety of regional anesthesia in Eisenmenger syndrome; however, epidural anesthesia can produce incomplete sensory block and may require rapid conversion to general anesthesia ^(18, 19), as was the case in our patient.

General anesthesia is a technique used in emergency cesarean sections. However, among the ventilatory parameters, it is suggested to avoid positive pressure ventilation and high PEEP levels, as this can lead to decreased venous return, worsening of right ventricular function, and a decrease in systemic blood pressure, which would increase the right-to-left shunt. ⁽²⁰⁾ Although general anesthesia is not recommended due to secondary hemodynamic effects, cases have been reported in which it has been considered the first choice. We report the case of a 19-year-old patient at 33 weeks of gestation diagnosed with Eisenmenger syndrome. Relevant physical examination revealed cyanosis and clubbing of the fingers. Her vital signs on admission were reported as follows: heart rate 84 bpm, respiratory rate 24 bpm, blood pressure 140/95 mmHg, and SpO₂ 74% assisted by a face mask at 6 liters per minute. Auscultation revealed a loud, grade 4/6 systolic murmur of P2 in the pulmonic area. Jugular venous distension and mild edema in the lower extremities were observed. In this case, general anesthesia was chosen due to thrombocytopenia. A slow sequence induction was performed with 10 mg of etomidate (due to its stability. Hemodynamically), atracurium 30 mg IV (to avoid further exacerbation of liver function). Anesthesia was maintained with sevoflurane (end-tidal concentration of 1-2%) in oxygen and remifentanyl infusion at a rate of 0.08–0.10 µg•kg–1 min–1. During the intraoperative period, the patient was hemodynamically stable. The only noteworthy finding was the SpO₂ reading, which remained between 67% and 76% throughout the procedure without incident. Blood loss of 100 ml and urine output of 200 ml were reported. The patient was not extubated in the operating room as she did not meet the criteria for extubation and was admitted to the ICU. After 3 hours, she was extubated and reported to be hemodynamically stable; however, after 12 hours, she was reintubated due to oxygen saturation below 70% and increased work of breathing, and subsequently died.

The same article presents the case of a 30-year-old pregnant woman, with a previous pregnancy (with a history of a cesarean section 6 years prior without complications), diagnosed with a 33-week pregnancy and a 10-year history of

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untreated congenital heart disease. She presented to the emergency department with vital signs of HR 84 beats/min, RR 20 breaths/min, BP 125/80 mmHg, and SpO₂ 58% on room air and 75%–88% on oxygen at 6 L/min by face mask. She had marked cyanosis and clubbing of her fingers. Auscultation revealed a loud P2 and a grade 5/6 systolic murmur in the pulmonary area. There was moderate lower extremity edema. After 3 days of observation, a cesarean section was performed under epidural anesthesia. An epidural catheter was inserted at the L1-L2 intervertebral space in the left lateral decubitus position. Incremental doses of 3–5 mL of 2% lidocaine without epinephrine were administered every 5 minutes, and a sensory block at the level of T6 was achieved with 21 mL of lidocaine over a 30-minute period. The sensory blockade level was intentionally kept no higher than T6 to avoid significant hemodynamic changes. The patient was hemodynamically stable throughout the surgery, with a blood loss of 200 mL and a urine output of 200 mL. After surgery, the patient was monitored in the ICU for 2 days before being transferred to the obstetric ward. Her condition was improving, and her SpO₂ was 93% on oxygen at 6 L/min. On the fifth postoperative day, she experienced a syncopal episode after walking to the bathroom. No other significant postoperative complications occurred. Both mother and baby were well 6 months later⁽²¹⁾. However, a meta-analysis shows no significant difference in perioperative mortality between general and regional anesthesia, and both approaches have significant morbidity and mortality [8]. The choice between general and epidural-spinal anesthesia should be made after considering the patient's specific physiology⁽²²⁾. There is generally little data to justify choosing one anesthetic technique over another⁽²³⁾.

In our case, when deciding to change the anesthetic technique, pre-oxygenation was initiated with a face mask at 5 L/min. Induction was performed with fentanyl 200 mcg IV, propofol 100 mg IV, and cisatracurium 8 mg IV. Direct laryngoscopy was performed with a #3 curved blade and a #7.5 endotracheal tube. On the first attempt, proper placement was verified clinically and with positive capnography. The endotracheal tube was secured at 21 cm and connected to an anesthesia machine under volume control, with the following parameters: VT 275, respiratory rate 12, I:E (1:2), PEEP 4, Pmax: 4.5, FiO₂: 66%. Gas mixing was performed, and the average oxygen saturation was 80%. Anesthesia was maintained with sevoflurane (end-tidal concentration of 1–2%). During the anesthetic phase, the patient remained hemodynamically stable without the use of vasopressors. The uterotonic used in this clinical case was a synthetic analog of oxytocin; as it is considered the first-line drug⁽²⁴⁾, however, in these patients, administration via an infusion pump is recommended since rapid administration of oxytocin has been reported to cause decreased systemic vascular resistance (SVR), tachycardia, and maternal death⁽²⁵⁾.

The most dangerous period for exacerbation of pulmonary hypertension is reported to be the puerperium, with a mortality rate of up to 70% between days 2 and 30 postpartum. The highest incidence of maternal mortality during this period is caused by refractory right heart failure, pulmonary hypertensive crisis, thromboembolism, volume depletion, and preeclampsia⁽²⁶⁾.

CONCLUSION.

Currently, an increasing number of young women with congenital heart disease are presenting to gynecologists and anesthesiologists expressing their desire to have children. Based on current evidence, heart disease is no longer an absolute contraindication for pregnancy; however, it carries a high mortality rate, and therefore, these women should be counseled and advised, knowing that pulmonary hypertension and Eisenmenger syndrome are a deadly combination not only for the mother but also for the baby. The decision to become pregnant or to continue a pregnancy in the context of maternal heart disease must consider the objective medical risk and the emotional value the pregnancy holds for the woman and her partner. The physician's goal is to educate the patient and her partner. Patient management should be multidisciplinary and comprehensive; the role of the anesthesiologist should be focused on maintaining hemodynamic equilibrium, avoiding increases and decreases in vascular resistance, ensuring adequate blood volume and venous return not only during pre-anesthetic and intra-anesthetic assessments but also during post-anesthetic and post-operative care.

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