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Pregnancy Complicated by an Unrepaired Ventricular Septal Defect with Eisenmenger Syndrome, Pre-eclampsia with Severe Features, and Oligohydramnios: A Case Report and Literature Review

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Abstract

Eisenmenger syndrome represents the most advanced form of pulmonary arterial hypertension associated with congenital heart disease, in which a chronic left-to-right shunt reverses to a bidirectional or right-to-left shunt. Pregnancy in this setting carries a maternal mortality rate of 30 to 50% and is classified as modified World Health Organisation (mWHO) class IV, making pregnancy an absolute contraindication. Concurrent pre-eclampsia with severe features further increases maternal and fetal risks. We report a 20-year-old primigravida at 30 weeks of gestation presenting with progressive dyspnea, hypertension, and central cyanosis. She had had an unrepaired congenital heart defect since infancy. On admission, blood pressure was 156/95 mmHg, respiratory rate 39 breaths/min, and peripheral oxygen saturation 85% despite oxygen therapy with a non-rebreather mask. Physical examination revealed a grade 4/6 systolic murmur with fixed splitting of the second heart sound, proteinuria, and digital cyanosis. Transthoracic echocardiography demonstrated a large ventricular septal defect with bidirectional shunting, confirming Eisenmenger syndrome. The diagnosis was unrepaired ventricular septal defect complicated by Eisenmenger syndrome, pre-eclampsia with severe features, and severe oligohydramnios. After multidisciplinary stabilization, an emergency cesarean section under general anesthesia delivered a live preterm female infant. A postplacental intrauterine device was inserted for contraception. The postpartum course was complicated by persistent hypoxemia requiring intensive cardiac care. This case highlights the importance of preconception counseling, early risk stratification, multidisciplinary management, timely delivery, meticulous peripartum care, and effective postpartum contraception in women with congenital heart disease.

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Introduction

Congenital heart disease (CHD) is one of the most common congenital abnormalities worldwide, with an estimated incidence of approximately 8 per 1,000 live births. Advances in pediatric cardiology and cardiac surgery have enabled more than 85% of affected children to survive into adulthood, resulting in a growing population of adults with congenital heart disease (ACHD) (Moodie, 2011; Mutluer & Çeliker, 2018). Despite improved survival, ACHD patients remain vulnerable to long-term complications including heart failure, arrhythmias, thromboembolic events, pulmonary hypertension, and sudden cardiac death (Niwa, 2018).

The increasing number of women with congenital heart disease reaching reproductive age has led to a rise in pregnancies complicated by cardiac disorders. Pregnancy induces substantial hemodynamic changes, including increased plasma volume, cardiac output, and heart rate, which may precipitate cardiovascular decompensation in women with underlying structural heart disease (Van Hagen & Roos-Hesselink, 2020). Cardiovascular disease accounts for approximately 4% of pregnancy-related complications and remains a significant contributor to maternal mortality worldwide (Farhan & Yaseen, 2019).

Ventricular septal defect (VSD) is the most common congenital cardiac lesion, representing approximately 40% of all congenital heart defects (Eckerström et al., 2023; Komatsu et al., 2023). Although most large VSDs are repaired during childhood, unrepaired defects may lead to chronic pulmonary overcirculation, pulmonary vascular remodeling, and ultimately pulmonary arterial hypertension (Harris, 2011).

Eisenmenger syndrome represents the most advanced stage of pulmonary arterial hypertension associated with congenital heart disease and is characterized by reversal of a longstanding left-to-right shunt into a bidirectional or right-to-left shunt (Campos & Benvenuti, 2017; Celermajer, 2017). The condition is classified as modified World Health Organization (mWHO) class IV and is considered an absolute contraindication to pregnancy because maternal mortality remains between 30% and 50% (Canobbio et al., 2017; Slaibi et al., 2021; Yuan, 2016).

Women with Eisenmenger syndrome face substantial maternal and fetal risks during pregnancy, including heart failure, worsening hypoxemia, fetal growth restriction, preterm delivery, and maternal death (Alsomali et al., 2022; Van Hagen & Roos-Hesselink, 2020). The coexistence of severe pre-eclampsia further increases the likelihood of adverse outcomes because pulmonary hypertension is independently associated with hypertensive disorders of pregnancy (Fox et al., 2019; Thomas et al., 2017).

This study aimed to describe the clinical characteristics, diagnostic findings, multidisciplinary management, and maternal-fetal outcomes of a pregnant woman with unrepaired VSD complicated by Eisenmenger syndrome, severe pre-eclampsia, and oligohydramnios.

Materials and Methods

Study Design

This study employed a descriptive observational design using a single-case approach.

Study Subject

The subject was a 20-year-old primigravida at 30 weeks of gestation diagnosed with an unrepaired ventricular septal defect complicated by Eisenmenger syndrome, severe pre-eclampsia,

and oligohydramnios who received treatment at Dr. Mohammad Hoesin General Hospital, Palembang, Indonesia.

Data Collection

Clinical information was retrospectively obtained from medical records, including:

- Demographic characteristics
- Medical and obstetric history
- Physical examination findings
- Laboratory investigations
- Echocardiographic findings
- Obstetric ultrasonography results
- Therapeutic interventions
- Maternal and neonatal outcomes

Data Analysis

Data were analyzed descriptively. Clinical findings and management approaches were interpreted in accordance with current guidelines and the literature on congenital heart disease, pulmonary hypertension, and pregnancy.

Ethical Considerations

Written informed consent for publication was obtained from the patient. Institutional ethical approval was obtained in accordance with local regulations.

Results and Discussions

Case Presentation

History and presentation

A 20-year-old woman (gravida 1, para 0) at 30 weeks of gestation was referred by an obstetrician with a diagnosis of pre-eclampsia with severe features and cyanotic congenital heart disease in a live intrauterine pregnancy. She reported progressive dyspnea over the preceding two days, which worsened with advancing gestation. She denied nausea, vomiting, vaginal bleeding or fluid leakage, trauma, abdominal massage, recent coitus, or the use of herbal remedies. Bowel and bladder function were normal. There was no history of hypertension before pregnancy; elevated blood pressure was first recognized at 28 weeks of gestation. Her family reported that she had been diagnosed with a “hole in the heart” in infancy that had never been corrected and had remained asymptomatic. She denied easy fatigability or exertional syncope, and fetal movements were still perceived.

Her last menstrual period was 15 August 2023. She had been married for six months, and this was her first pregnancy. Her past medical history was notable for hypertension since 28 weeks of gestation and congenital heart disease diagnosed at two years of age, for which she had not received regular care. Diabetes mellitus, asthma, and allergy were denied, as was any relevant family history.

Physical examination

Upon admission, the patient was alert. Her blood pressure was 156/95 mmHg, heart rate 112 beats/min, respiratory rate 39 breaths/min, peripheral oxygen saturation 85% on a non-rebreather mask at 15 L/min, and temperature 36.6°C. Her body weight was 63 kg, and height was 162 cm (body mass index 24 kg/m^2). Examination of the neck revealed a jugular venous pressure of 5–2 cmH_2O, without lymphadenopathy. The lungs were clear, with no rales or wheezing sounds. Cardiac auscultation revealed a regular rhythm with a systolic murmur and a gallop; subsequent cardiology examination documented a grade 4/6 systolic murmur with maximal intensity at the fourth left

intercostal space along the parasternal line, non-radiating, with fixed splitting of the second heart sound. The extremities were warm with cyanosis of the fingertips and toes and no pretibial edema.

Obstetric examination revealed a fundal height of 23 cm (two finger-breadths above the umbilicus), a longitudinal lie with the fetal back on the right, no uterine contractions, and a fetal heart rate of 140 beats/min, with an estimated fetal weight of 1,500 g. Speculum examination showed a non-livid portio with a closed external os and no discharge, bleeding, or visible products. On vaginal examination, the cervix was soft and posterior, with no dilatation or effacement. The Gestosis index was 3 (edema 0; proteinuria ++ [1]; systolic 1; diastolic 1).

Investigations

Obstetric ultrasonography performed on admission demonstrated a single live fetus in cephalic presentation: biparietal diameter 8.13 cm, transcerebellar diameter 3.37 cm (~30 weeks 5 days), abdominal circumference 24.33 cm, head circumference 29.65 cm, femur length 5.85 cm, and estimated fetal weight 1,468 g. The amniotic fluid was reduced, with an amniotic fluid index of 3.83 cm, and the placenta was fundal-anterior. The findings were consistent with a 30-week single live cephalic fetus with oligohydramnios.

The key laboratory results are summarized in Table 1. Notable findings included a hemoglobin level of 15.8 g/dL and hematocrit of 45% without macrocytosis (mean corpuscular volume, 81.2 fL), leukocytosis ($13.67 \times 10^3/mm^3$), proteinuria (++), hematuria (+++), and elevated lactate dehydrogenase (353 U/L). Arterial blood gas analysis on 60% inspired oxygen showed a pH of 7.423, partial pressure of oxygen of 66.7 mmHg, and partial pressure of carbon dioxide of 23.8 mmHg, consistent with hypoxemia and compensatory hypocapnia. Chest radiography demonstrated cardiomegaly.

Table 1. Selected laboratory findings on admission

Parameter	Result	Reference range
Hemoglobin	15.8 g/dL	11.40–15.00 g/dL
Hematocrit	45%	35–45%
Erythrocytes	$5.54 \times 10^6/mm^3$	$4.00–5.70 \times 10^6/mm^3$
Leukocytes	$13.67 \times 10^3/mm^3$	$4.73–10.89 \times 10^3/mm^3$
Platelets	$247 \times 10^3/\mu L$	$189–436 \times 10^3/\mu L$
Mean corpuscular volume	81.2 fL	85–95 fL
Lactate dehydrogenase	353 U/L	135–214 U/L
Albumin	3.5 g/dL	3.5–5.0 g/dL
Creatinine	0.54 mg/dL	0.50–0.90 mg/dL
Uric acid	5.3 mg/dL	—
Urine protein (qualitative)	++	Negative
Arterial pH (FiO_2 60%)	7.423	7.35–7.45
Arterial pO_2	66.7 mmHg	83–108 mmHg
Arterial pCO_2	23.8 mmHg	35–45 mmHg

FiO2, fraction of inspired oxygen; pCO2, partial pressure of carbon dioxide; pO2, partial pressure of oxygen.

Transthoracic echocardiography performed on the day of admission showed extreme right axis deviation, right ventricular hypertrophy, right and left atrial enlargement, and bifascicular block (right bundle branch block with left posterior fascicular block). A subsequent echocardiographic study reported a left ventricular ejection fraction of 50% with left ventricular hypertrophy and a large VSD with bidirectional shunting, confirming the diagnosis of Eisenmenger syndrome.

Diagnosis

Based on the clinical, laboratory, and echocardiographic findings, the patient was diagnosed with a 30-week primigravid pregnancy complicated by pre-eclampsia with severe features, a suspected cyanotic congenital heart defect (unrepaired VSD) with Eisenmenger syndrome, a single live cephalic fetus, and severe oligohydramnios. Functional status corresponded to New York Heart Association (NYHA) class IV.

Management and outcome

Initial management included monitoring of vital signs, general condition, and fetal heart rate; a magnesium sulfate protocol (4 g slow intravenous bolus of 40% solution followed by a maintenance infusion of 6 g in 500 mL Ringer's lactate); oral nifedipine 10 mg every 8 hours; and planned antenatal corticosteroids for fetal lung maturation. The patient was co-managed by the cardiology and intensive care teams. Given progressive respiratory failure and hypoxemia, she was intubated and placed on mechanical ventilation. An emergency abdominal delivery was planned with informed consent, which included the possibility of death on the operating table.

A lower-segment cesarean section was performed under general anesthesia using a Pfannenstiel incision. A live female neonate was delivered, weighing 1,730 g (length 41 cm, head circumference 31 cm), with Apgar scores of 5 and 7 at 1 and 5 minutes, respectively. The placenta was delivered intact. The estimated blood loss was 300 mL. A post-placental intrauterine device was inserted for contraception before closure. Postoperative orders included oxytocin infusion, tranexamic acid, analgesia, and antibiotic prophylaxis with continued hemodynamic monitoring.

During the postpartum period, the patient remained dyspneic with persistent hypoxemia (oxygen saturation 60–75% on supplemental oxygen) and labile hypertension. Methyldopa was added for blood pressure control, and furosemide, N-acetylcysteine, and antibiotics were administered for volume management and a suspected respiratory infection. Repeat echocardiography confirmed a large VSD with a left ventricular ejection fraction of 49%. The patient was transferred to the cardiac intensive care unit for ongoing management, including consideration of a pulmonary vasodilator. Definitive surgical correction of the defect was deferred because the precise anatomy of the lesion had not been fully delineated at the time of delivery.

Discussion

Establishing the diagnosis

The cornerstone of evaluating congenital heart disease in pregnancy is careful risk stratification. Pre-pregnancy assessment ideally includes a detailed history, physical examination, electrocardiography, echocardiography, and exercise testing; reduced exercise tolerance, symptoms of heart failure, or an irregular rhythm increases the risk of pregnancy (Mutluer & Çeliker, 2018; Van Hagen & Roos-Hesselink, 2020). The modified World Health Organization (mWHO) classification provides an essential first step in recognizing risk and determining which patients should be promptly referred to tertiary or quaternary centers (Table 2). Class I comprises mild lesions not associated with meaningful excess morbidity or mortality, whereas class IV denotes conditions, such as pulmonary arterial hypertension of any cause, in which pregnancy is contraindicated (Canobbio et al., 2017; Halpern et al., 2023).

Table 2. Maternal cardiovascular risk according to the modified WHO classification

mWHO class	Risk description	Representative lesions
I	No detectable increase in maternal mortality and no/mild increase in morbidity	Small/mild uncomplicated pulmonary stenosis, patent ductus arteriosus, mitral valve prolapse; successfully repaired simple lesions

II	Small increase in maternal mortality and moderate increase in morbidity	Unoperated atrial or ventricular septal defect; repaired tetralogy of Fallot; most arrhythmias
II–III	Moderate increase in mortality and morbidity	Mild left ventricular impairment; hypertrophic cardiomyopathy; native or tissue valve disease; repaired coarctation
III	Significant increase in maternal mortality or severe morbidity; expert counseling and intensive monitoring required	Mechanical valve; systemic right ventricle; Fontan circulation; unrepaired cyanotic heart disease; other complex CHD
IV	Extremely high maternal mortality risk; pregnancy is contraindicated, and termination should be discussed	Pulmonary arterial hypertension of any cause; severe systemic ventricular dysfunction; severe mitral or symptomatic aortic stenosis

CHD, congenital heart disease; mWHO, modified World Health Organization classification. Adapted from Canobbio et al.

VSD is the most common congenital cardiac lesion, accounting for approximately 40% of cases, and is generally regarded as a simple lesion when appropriately treated in childhood (Eckerström et al., 2023; Komatsu et al., 2023). Large defects typically impose a substantial left ventricular volume load and cause heart failure early in life, so most are corrected in childhood; consequently, the majority of uncorrected defects encountered in adults are small (Harris, 2011). However, an uncorrected VSD with a persistent significant shunt may progress to pulmonary hypertension and has been associated with an increased risk of pre-eclampsia (Halpern et al., 2023; Harris, 2011). In a large multicenter cohort, pre-eclampsia occurred 4.59 times more frequently in women with uncorrected VSD than in controls (Harris, 2011).

In the present case, the diagnosis was an unrepaired cyanotic VSD with Eisenmenger syndrome. The chronicity of the disease was suggested by an elevated normal hematocrit (45%) without an accompanying rise in mean corpuscular volume (81.2 fL), in keeping with secondary erythrocytosis, together with a documented diagnosis of a cardiac defect in infancy. Auscultation revealed a grade 4/6 systolic murmur that was maximal at the fourth left intercostal space with fixed splitting of the second heart sound—the latter typically reflecting an atrial septal defect or pulmonary arterial hypertension with early pulmonary valve closure. The classic harsh VSD murmur may become softer or disappear once Eisenmenger physiology supervenes, as the shunt gradient falls (Ayad & Almamoury, 2023).

Impact of the disease on pregnancy

Although VSD is an acyanotic defect, cyanosis may emerge in adulthood when a persistent or uncorrected shunt leads to pulmonary vascular disease. Up to one-third of patients with cyanotic heart disease experience cardiovascular complications during pregnancy, predominantly heart failure and worsening hypoxemia, requiring hospitalization. The probability of live birth decreases steeply with the severity of cyanosis, reaching only approximately 12% in women with oxygen saturation below 85%; such women are generally advised against pregnancy (Van Hagen & Roos-Hesselink, 2020).

Pulmonary hypertension, defined as a mean pulmonary arterial pressure of ≥ 25 mmHg at rest, is among the least tolerated conditions during pregnancy. It most often arises from a longstanding left-to-right shunt that increases pulmonary flow and, ultimately, pulmonary vascular resistance, producing shunt reversal and bidirectional flow, which defines Eisenmenger syndrome (Van Hagen & Roos-Hesselink, 2020). The physiological burden of pregnancy aggravates this process: pulmonary vascular disease prevents the expected gestational fall in pulmonary vascular resistance, the

necessary rise in cardiac output cannot be achieved, plasma volume increases by up to 50% in the third trimester, and the fall in systemic vascular resistance augments right-to-left shunting and worsens hypoxemia and cyanosis (Canobbio et al., 2017). Labor and the puerperium are the most hazardous periods, with hemodynamic shifts from blood loss, uterine contraction, and withdrawal of sympathetic tone, and the prothrombotic state of pregnancy may contribute to in situ pulmonary thrombosis (Canobbio et al., 2017; Yuan, 2016).

Among congenital lesions, VSD is the most frequent cause of Eisenmenger physiology, followed by atrial septal defects and patent ductus arteriosus, and Eisenmenger syndrome secondary to VSD carries a higher mortality (approximately 44%) than that secondary to atrial septal defects (Alsomali et al., 2022). Women with pulmonary hypertension also develop pre-eclampsia, including pre-eclampsia with severe features, more frequently than women without heart disease (Thomas et al., 2017). Impaired uteroplacental flow in mothers with congenital heart disease is associated with adverse fetal outcomes, including fetal growth restriction, fetal death (1%), preterm birth (14%), and low Apgar scores (6%) (Van Hagen & Roos-Hesselink, 2020). In this patient, the cardiac disease aggravated the pregnancy through pre-eclampsia with severe features and the need for early delivery; severe pre-eclampsia occurs more often in NYHA class III–IV heart failure, which is also associated with longer intensive-care stays and higher maternal mortality than class I–II disease (Anwar et al., 2020).

Management considerations

Cardiovascular evaluation should ideally be performed before conception. For women with unplanned pregnancies, the diagnostic work-up must be individualized, weighing the potential risks to the mother and fetus. For complex lesions in which continuation of pregnancy poses a significant risk of maternal morbidity or mortality, an open discussion of therapeutic termination is essential. However, the therapeutic benefit of termination after 20 weeks is debated because many cardiopulmonary adaptations are already established (Canobbio et al., 2017). Eisenmenger syndrome is classified as mWHO class IV, and women typically undergo elective abdominal delivery around the 32nd week because approximately half of the increase in circulating blood volume occurs within the first 30 weeks (Alsomali et al., 2022).

The optimal mode of delivery in Eisenmenger syndrome with severe pulmonary hypertension remains undefined. Cesarean sections avoid the repetitive increases in cardiac output that accompany uterine contractions and shorten the duration of hemodynamic disturbance; it is generally indicated in high-risk situations, such as advanced pulmonary hypertension, and vaginal delivery is associated with less blood loss and fewer abrupt hemodynamic changes, but carries the risks of prolonged labor, acidosis, hypercapnia, and hypoxia that can raise pulmonary arterial pressure (Canobbio et al., 2017; Alsomali et al., 2022; Olsson & Channick, 2016). Neuraxial anesthesia is preferred whenever feasible, including in mWHO class III–IV lesions, because epidural anesthesia minimizes sudden changes in systemic blood pressure; general anesthesia is reserved for cardiopulmonary decompensation, anticoagulation, severe thrombocytopenia, or patient refusal of neuraxial techniques (Canobbio et al., 2017; Yuan, 2016; Meng & Arendt, 2021). Large fluid boluses and excessive blood loss should be avoided; volume overload should be managed with diuretics, and inhaled nitric oxide with supplemental oxygen should be considered for cardiac decompensation, while oxytocin should be used with caution (Canobbio et al., 2017).

In this case, abdominal delivery was performed at 30 weeks because pulmonary hypertension complicating a longstanding VSD contraindicated continuation of pregnancy, induction risked further cardiac deterioration, and pre-eclampsia with severe features independently warranted timely delivery. General anesthesia was selected in the context of impending respiratory failure and hypoxemia, allowing controlled ventilation and postoperative hemodynamic support; surgical correction of the defect was deferred pending complete anatomical delineation. Given the very high

mortality rate of the postpartum period, the patient was managed in an intensive care setting with close monitoring, in line with recommendations to delay discharge for 7–10 days to detect right ventricular failure and to titrate pulmonary vasodilators (Canobbio et al., 2017).

Because Eisenmenger syndrome is an absolute contraindication to pregnancy, effective contraception is essential, particularly in young, sexually active patients. A levonorgestrel-releasing intrauterine device offers an effective and safe option, whereas combined hormonal contraceptives, especially those containing ethinylestradiol, carry an increased thromboembolic risk (Van Hagen & Roos-Hesselink, 2020). Therefore, a postplacental intrauterine device was inserted. Irreversible options, such as tubal ligation, were not undertaken, reflecting the cardio-obstetric team's intention to pursue future correction of the lesion and preserve the possibility of a subsequent, better-tolerated pregnancy.

Conclusion

This case illustrates the convergence of three high-risk conditions—an unrepaired ventricular septal defect with Eisenmenger physiology, pre-eclampsia with severe features, and severe oligohydramnios—in a young primigravida. Eisenmenger syndrome remains an absolute contraindication to pregnancy, with a maternal mortality rate of 30–50%, and the physiological changes of pregnancy can precipitate rapid cardiac decompensation. Optimal outcomes depend on early multidisciplinary planning, accurate mWHO risk stratification, timely abdominal delivery after fetal viability, meticulous peripartum hemodynamic management, intensive postpartum monitoring, and effective contraceptive counseling. This case reinforces the central role of preconception counseling and structured risk assessment in women with congenital heart disease.

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