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CASE REPORT
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Conservative Management of Placental Abruption

Muhammad Adhitya Wicaksono[1], Robyanoor Ahyadi[2], Bambang Abimanyu[2], Ruth Widhiati[2], Hermin Sabaruddin[2]

[1]Obstetrics and Gynecology Specialist Education Program, Faculty of Medicine, Lambung Mangkurat University/ Ulin Regional Hospital, Banjarmasin, Indonesia
[2]Fetomaternal Division, Department of Obstetrics and Gynecology, Faculty of Medicine, Lambung Mangkurat University/ Ulin Regional General Hospital, Banjarmasin, Indonesia
Coresponding author: adit.wicakson22@gmail.com

Abstract:

Placental abruption is a severe obstetric complication. This case involves a 43-year-old woman with chronic hypertension, severe preeclampsia, and IUGR at 26-27 weeks gestation. Underweight (BMI 18.1), she presented with suspected placental abruption, oligohydramnios, and fetal growth restriction. This study highlights the importance of early detection and a multidisciplinary approach in improving outcomes. The patient received conservative treatment, including magnesium sulfate, corticosteroids, antihypertensives, and nutritional support. Despite maternal stability, fetal IUGR, and hypoxia led to an emergency cesarean at 33-34 weeks. A 1055g baby girl was delivered with Apgar scores of 6, 7, and 8. Placental examination revealed retroplacental hemorrhage and calcification. This case emphasizes the importance of early diagnosis, intensive monitoring, and timely intervention in high-risk pregnancies with placental abruption and severe preeclampsia. A multidisciplinary approach is essential to improve maternal and neonatal prognosis.

Keywords: cesarean section; high-risk pregnancy; IUGR; oligohydramnios; placental abruption; severe preeclampsia

Introduction

Intrauterine hematoma is a common pregnancy complication, which can occur at any stage of pregnancy, with obstetric hemorrhage observed in 5–25% of first-trimester cases, which poses a risk to both the mother and the fetus.[1] The incidence in the first trimester ranges from 4–22%, with small hematomas being more common during this period, while larger masses tend to appear in the second trimester.[2] Studies indicate that hematomas, regardless of onset time, are linked to higher risks of adverse pregnancy outcomes such as vaginal bleeding, miscarriage, preterm birth, pregnancy-induced hypertension, preeclampsia, gestational diabetes, intrauterine growth restriction, and fetal death.[3,4]

Intrauterine hematomas include retroplacental, subchorionic, subamnionic, and intraplacental types. Retroplacental and marginal hematomas are well-studied, whereas intraplacental hematomas are rare and poorly researched, with limited literature discussing their distinct clinical significance and outcomes.[1,2]

Recent histopathological findings highlight the unique morphological characteristics of intraplacental hematoma, which differs from parabasally located intervillous thrombohematoma.[5] Intraplacental hematomas occur in the intervillous space, while retroplacental hematomas form between the basal plate and myometrium, lifting placental tissue. Intraplacental hematomas carry greater maternal–fetal risks, yet research often combines all intrauterine hematomas, with no specific studies focusing solely on intraplacental hematoma outcomes.[4,6]

Recent advances in placental pathology have provided greater insight into the relationship between chronic hypertension and placental abnormalities. Rounded intraplacental hematomas (RIHs), first described by Fitzgerald et al. in 2011, are a special form of placental thrombohematoma associated with significant placental compression and infarction.[7] These lesions are predominantly parabasally located, have a rounded morphology, and represent a unique marker of placental vascular compromise. The identification of RIHs highlights the important role of chronic hypertension in disrupting placental structure and function, further emphasizing its contribution to poor pregnancy outcomes.[5]

This case report discusses the clinical complexities, diagnostic challenges, and therapeutic approaches in the management of a high-risk pregnancy complicated by chronic hypertension, severe preeclampsia, and retroplacental hemorrhage. By describing this case, this report aims to improve the understanding of intrauterine hematoma, particularly retroplacental hemorrhage, and its impact on maternal and fetal outcomes, while emphasizing the importance of timely intervention and multidisciplinary management.

Case

A 44-year-old woman, gravida six para four (G6P4A1), came to Ulin Hospital in November 2024 at 33 weeks 2 days of gestation with complaints of "tight abdomen," oligohydramnios, and significant maternal complications. Her medical history included chronic hypertension diagnosed five years earlier, treated with Amlodipine 5 mg once daily. Her obstetric history showed several high-risk factors, including previous placental abruption, recurrent pregnancy loss, and a current pregnancy complicated by severe preeclampsia (PEB), oligohydramnios, and intrauterine growth restriction (IUGR). The fetal weight was estimated to be only 1200 g, indicating a significant risk for poor neonatal outcomes.

In mid-October 2024, at 26–27 weeks' gestation, Mrs. Ari was admitted to Idaman Hospital with four hours of vaginal bleeding. She was diagnosed with grade II placental abruption, severe preeclampsia, and premature rupture of membranes (PROM),

with blood pressure ≥160/110 mmHg. Immediate stabilization included Magnesium Sulfate (4 g IV bolus, then 1 g/hour infusion) for eclampsia prevention. Dexamethasone 6 mg IM every 12 hours for two doses was administered to promote fetal lung maturity. Hypertension was managed with Methyldopa 500 mg three times daily and Nifedipine 10 mg PRN, while Tranexamic Acid 1000 mg IV addressed bleeding. Biophysical profile evaluations showed good fetal status, allowing conservative management to prolong pregnancy, enhance neonatal survival chances, and ensure close maternal–fetal monitoring despite significant risks.

In late November 2024, the patient was re-admitted to Ulin Hospital with severe oligohydramnios (AFI 2.3 cm), ascites, and hypoalbuminemia (2.3 g/dL), placing her at high risk for placental dysfunction and preterm delivery. Management included several days of 20% IV albumin to correct hypoalbuminemia, fluid restriction to 1000 mL/day to prevent worsening ascites, and nutritional supplementation with VIP Albumin capsules, iron, and calcium for maternal and fetal health. Antihypertensive therapy with Methyldopa 500 mg three times daily and Nifedipine 10 mg was continued to maintain blood pressure control. These measures aimed to stabilize maternal condition and optimize outcomes before potential preterm delivery.

On December 2, 2024, an emergency cesarean section was performed at 33 weeks and 4 days of gestation due to deteriorating maternal and fetal conditions, including non-reassuring fetal heart rate patterns and worsening maternal preeclampsia. A 1055 g female infant was delivered with Apgar scores of 6, 7, and 8 at 1, 5, and 10 minutes, respectively. The placenta showed significant retroplacental hemorrhage and calcification, consistent with a history of previous placental abruption. During the procedure, bilateral fimbriectomy was performed for sterilization.

Postoperatively, the patient was managed with oxytocin infusion (20 IU in Ringer Lactate) at a rate of 20 drops per minute for 24 hours to minimize uterine atony and promote hemostasis. Seizure prophylaxis was continued with maintenance infusion of MgSO4 (1 g/hour) for 24 hours after delivery. Pain management included Ketorolac 30 mg IV three times daily, and broad-spectrum antibiotics were given with Ceftriaxone 1 g IV twice daily to prevent postpartum infection. To reduce the risk of further bleeding, Tranexamic Acid 500 mg IV three times daily was given for 48 hours.

Blood pressure management remains a priority with Methyldopa and Nifedipine continued as needed. Postoperative monitoring includes strict fluid balance, wound care, and daily hemoglobin level evaluation to monitor for postpartum anemia.

Both mother and baby improved. The newborn received NICU care for prematurity, with respiratory and nutritional support. Mrs. Ari was discharged on day five in stable condition, with controlled blood pressure, improved albumin, and follow-up advised for postpartum blood pressure and nutrition monitoring.

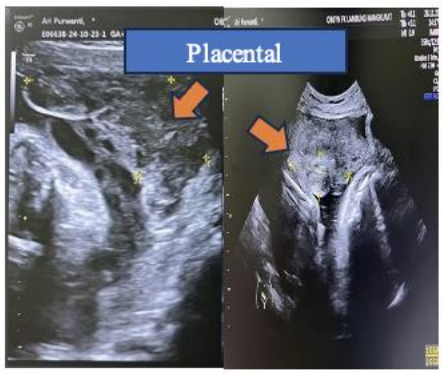

Figure 1. USG 23-10-2024

Table 1. Laboratory examination

Date/Time	Test	Parameter	Result	Reference Value
29-11-2024 09:51 WITA	Complete Blood Count	Hemoglobin (Hb)	14.5	12-16 g/dL (female), 13-17 g/dL (male)
		Leukocytes (Leu)	7,700	4,000-10,000 /µL
		Platelets (PLT)	226,000	150,000-450,000 /µL
		Hematocrit (Hct)	40.2	36-46% (female), 40-50% (male)
	HBs-Ag	NR	Non-reactive	
	Anti-HIV	NR	Non-reactive	
	PT/APTT	11.3/31.5	10-13 sec / 25-35 sec	
29-11-2024 09:51 WITA	Blood Chemistry	Random Blood Glucose (RBG)	74	<140 mg/dL (fasting)
		SGOT/SGPT	47/36	10-40 U/L
		Albumin (Alb)	2.0	3.5-5.0 g/dL
		Urea/Creatinine	46/1.23	10-50 mg/dL / 0.6-1.2 mg/dL
		Uric Acid	7.2	3.4-7.0 mg/dL
		LDH	410	140-280 U/L
		Na/K/Cl	138/4.1/108	135-145 mmol/L / 3.5-5.0 mmol/L / 95-105 mmol/L
29-11-2024 09:51 WITA	Urinalysis	Color	Yellow	Clear yellow
		Protein	Negative	Negative
		Ketones	Negative	Negative
		Blood	Negative	Negative
		Leukocytes	Negative	Negative
Urinalysis (Sediment)	Leukocytes	1-2	<5 HPF	
		Erythrocytes	2-3	<3 HPF
		Bacteria	Positive	Negative
30-11-2024 07:41 WITA	Lipid Profile	Total Cholesterol	282	<200 mg/dL
		HDL	49	>40 mg/dL
		LDL	216	<130 mg/dL
		Triglycerides	323	<150 mg/dL
02-12-2024 18:01 WITA	Complete Blood Count	Hemoglobin (Hb)	16.2	13-17 g/dL
		Leukocytes (Leu)	20,400	4,000-10,000 /µL
		Platelets (PLT)	167,000	150,000-450,000 /µL
		Hematocrit (Hct)	45.7	36-46% (female), 40-50% (male)
		Albumin (Alb)	2.3	3.5-5.0 g/dL
		FT3	1.88	2.3-4.2 pg/mL
		FT4	10.38	12-22 pmol/L
		TSHs	1.0859	0.4-4.0 µIU/mL
03-12-2024 06:56 WITA	Complete Blood Count	Hemoglobin (Hb)	14.7	13-17 g/dL
		Leukocytes (Leu)	23,900	4,000-10,000 /µL
		Platelets (PLT)	195,000	150,000-450,000 /µL
		Hematocrit (Hct)	43.3	36-46% (female), 40-50% (male)

Figure 2. Retroplacental bleeding during surgery.

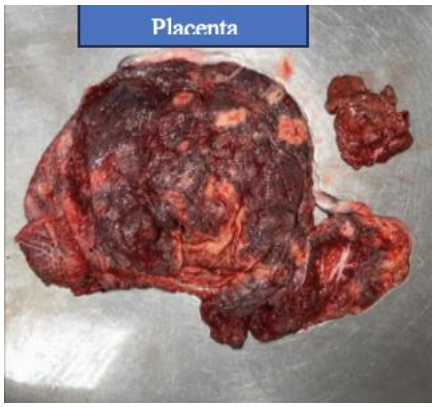

Figure 3. Postoperative retroplacental hemorrhage (size: 3 cm x 4 cm)

Discussion

A 44-year-old multiparous woman with chronic hypertension and prior abruption developed placental abruption complicated by severe preeclampsia, oligohydramnios, and IUGR. Long-term uteroplacental insufficiency and preeclampsia-induced endothelial damage caused retroplacental hemorrhage and calcification observed during cesarean delivery.[3]

At 26–27 weeks, vaginal bleeding and hypertension indicated maternal–placental compromise. Severe oligohydramnios and IUGR signaled fetal distress, requiring balance between gestational prolongation and maternal risk control. Hypoalbuminemia, ascites, elevated LDH, and uric acid reflected inflammation, protein loss, and endothelial dysfunction characteristic of severe preeclampsia.

Management prioritized maternal stabilization with Methyldopa and Nifedipine for hypertension control, magnesium sulfate for eclampsia prevention, and albumin infusion with fluid restriction for hypoalbuminemia and ascites. Corticosteroids promoted fetal lung maturity, while Doppler and biochemical monitoring guided timely cesarean delivery upon detecting a non-reassuring fetal heart rate.

Cesarean delivery revealed retroplacental hemorrhage, confirming placental abruption. Postoperatively, oxytocin, tranexamic acid, Ketorolac, and Ceftriaxone were administered. Both mother and premature infant improved, the latter after NICU care. This case highlights the importance of early detection, risk management, comprehensive monitoring, and multidisciplinary care, alongside preventive strategies like hypertension control and vigilant antenatal follow-up.

Placental abruption involves chronic and acute mechanisms. Chronic factors—thrombosis, inflammation, infection, and uteroplacental vasculopathy—cause hypoperfusion, abnormal spiral artery remodeling, infarction, and shallow trophoblast invasion, leading to hypoxia and increased vascular endothelial growth factor expression.[8] This cascade may deregulate the inflammatory response to decidual bleeding, thereby increasing its susceptibility to placental abruption and associated complications such as fetal growth restriction (FGR) and preeclampsia.[9,10]

The acute process often involves mechanical force or trauma, such as abdominal impact or rapid uterine decompression after amniotomy or delivery of the first twin. These triggers cause bleeding at the decidual-placental interface, where blood accumulates and disrupts placental attachment.[9,10] Vasospasm of small blood vessels further aggravates the bleeding. The inability of the uterus to contract sufficiently

due to distension contributes to continued bleeding.[11,12] Thrombin, a central mediator, promotes inflammation, endothelial injury, and extracellular matrix degradation while enhancing uterine contractions. Excessive thrombin release can lead to disseminated intravascular coagulation (DIC) and maternal morbidity.[13,14]

Placental abruption may be acute, with sudden bleeding, abdominal pain, uterine contractions, and possible fetal distress requiring emergency care, or chronic, with recurrent mild bleeding, persistent symptoms, and risks of fetal growth restriction and oligohydramnios. [15]

Bleeding severity in placental abruption depends on vessel size and location; central arterial bleeding causes acute, severe symptoms, while peripheral venous bleeding often presents with milder, gradual onset symptoms.[11,12] Ultrasound findings, although limited in sensitivity, can help diagnose abruption by identifying features such as retroplacental hematoma, placentomegaly, or intra-amniotic hematoma.[11] Placental abruption is diagnosed clinically by excluding other bleeding causes, notably placenta previa. Key signs are vaginal bleeding, abdominal pain, and uterine contractions. Ultrasound may support diagnosis but is limited by clot echogenicity and placental overlap.[13,15]

Management of placental abruption depends on severity and timing. In acute cases, priority is immediate maternal stabilization with IV fluids, blood products, and coagulation monitoring. Delivery is recommended to prevent harm, especially with fetal distress, and antenatal corticosteroids may be given in imminent preterm delivery. Chronic abruption requires close fetal surveillance through serial growth scans and non-stress testing, with delivery usually at 36–37 weeks unless complications arise. Hidden abruption demands high vigilance due to minimal or absent symptoms but carries high fetal death and coagulopathy risks. Management is complicated by occult bleeding, where visible blood loss may not match severity, and variable fetal heart rate patterns—abnormal in acute cases but sometimes normal in chronic cases. Histopathological placental examination can confirm pathophysiology, often showing decidual hemorrhage and trophoblastic necrosis. Timely diagnosis, individualized care, and ongoing monitoring are critical to improving maternal and fetal outcomes.[11]

New diagnostic tools such as thromboelastography (TEG) and rotational thromboelastometry (ROTEM) may aid in the rapid assessment of coagulation status, allowing for more targeted resuscitation efforts.

Placental abruption is a variable condition requiring prompt recognition and individualized management to reduce maternal and fetal morbidity and mortality. Research should aim to enhance diagnostic accuracy and develop tailored strategies, particularly for chronic and occult cases. In this case, the patient had a retroplacental hematoma, the most common type, located between the basal plate and myometrium, elevating the placenta into the amniotic cavity. This finding aligns with the pathophysiology of placental infarction, involving spiral artery occlusion by thrombus, excessive fibrin deposition around placental villi, and impaired fetal circulation caused by fetal thrombotic vasculopathy, as confirmed during surgery.[5,7]

Retroplacental hemorrhage with >5% placental calcification and infarction is strongly linked to severe preeclampsia, occurring in up to 39% of such cases, highlighting its role in adverse maternal–fetal outcomes.[8] Thrombophilia and antiphospholipid syndrome can worsen placental disease. Although absent here, intraplacental hematoma is notable for its rarity and strong link to adverse outcomes, including preeclampsia, IUGR, and fetal death.[16,17] Placental lesions, like retroplacental or intraplacental hematomas, appear hypoechogenic with a regular shape

and hyperechogenic rim on ultrasound. MRI can enhance diagnostic accuracy in suspected intraplacental hematoma cases.[11,18]

The clinical implications of placental abruption depend on the extent and location of separation. Placental abruption can be total or partial, and marginal or central. Classification of placental abruption is based on the following clinical findings:[11]

Class 0: Asymptomatic

This solution is often discovered retrospectively through the discovery of a blood clot on the side of the placenta that has been delivered by the mother. There are no visible symptoms during pregnancy.

Class 1: Mild

In mild abruption, vaginal bleeding may be absent or only slight. The uterus may be slightly tender, but maternal blood pressure and heart rate remain within normal limits. There are no signs of fetal distress.

Class 2: Moderate

In these cases, vaginal bleeding may be absent to moderate in amount. There is significant uterine tenderness with tetanic contractions. Maternal vital signs show changes, such as maternal tachycardia or orthostatic changes in blood pressure. Fetal distress is seen, and there are changes in the blood clotting profile, such as hypofibrinogenemia.

Class 3: Severe

Severe abruption is characterized by heavy or absent vaginal bleeding, but the uterus feels hard like a board on palpation. Maternal shock often occurs, accompanied by changes in the blood clotting profile such as hypofibrinogenemia and coagulopathy. In these cases, fetal death almost always occurs.

A classification of 0 or 1 is usually associated with partial or marginal separation, while a classification of 2 or 3 tends to be associated with complete or central separation. Understanding the mechanism and severity of placental abruption is essential to determine the appropriate management strategy to reduce the risk to the mother and fetus.

The patient had Class 2 (Moderate) Placental Abruption, evidenced by retroplacental bleeding during cesarean, tetanic uterus, severe preeclampsia, oligohydramnios, and placental calcification. IUGR and fetal distress, shown by low Apgar score, further supported the diagnosis and highlighted significant maternal-fetal complications.

In this case, placental abruption likely stemmed from chronic hypertension and severe preeclampsia, causing vascular endothelial damage and chronic placental insufficiency. A history of recurrent abortion and prior abruption indicated predisposition to placental dysfunction, further worsened by systemic endothelial dysfunction, heightening maternal and fetal complication risks.

Maternal outcomes of Class 3 placental abruption can include shock and coagulopathy. Although maternal shock was not explicitly mentioned in this patient, the severe preeclampsia she experienced suggests serious systemic compromise.[10] Postoperative management with antihypertensive therapy, seizure prophylaxis using magnesium sulfate, and broad-spectrum antibiotics are important steps to prevent further complications. The impact on the fetus is significant, as seen from the presence of IUGR, oligohydramnios, and low birth weight (1055 g). Severe placental separation compromises oxygen and nutrient supply, leading to fetal hypoxia that contributes to preterm birth and neonatal intensive care in the NICU. In this case, emergency cesarean section at 33 weeks and 4 days of gestation was the right step to save the lives of the mother and fetus.

The management strategy for this patient emphasizes the importance of timely intervention in a pregnancy complicated by placental pathology. The decision to perform an emergency cesarean section at 33 weeks 4 days is critical to reduce the risk of further maternal and fetal damage. Low molecular weight heparin (LMWH) and aspirin are often recommended for placental infarction seen

on ultrasound; however, in this case, the focus remains on stabilizing the maternal and fetal condition through delivery.[12]

Postoperative management with oxytocin, magnesium sulfate, and antihypertensives addressed uterine atony, seizure risk, and hypertension. The premature neonate (1055 g) recovered in the NICU, benefiting from antenatal corticosteroids. This case underscores multidisciplinary care for placental hematoma in preeclampsia and chronic hypertension, with timely delivery as the key intervention. Further research is needed for targeted diagnosis and treatment.[4]

Preterm delivery was indicated due to nonreassuring fetal heart rate and worsening maternal status from severe preeclampsia and placental abruption. Post-cesarean, the premature infant recovered reasonably (Apgar 6, 7, 8) but required NICU admission and respiratory support, reflecting elevated morbidity risk in infants of hypertensive mothers.

The important aspect of this case is the performance of tubal sterilization during cesarean section. Tubal sterilization is an elective procedure that provides permanent contraception. As emphasized by professional organizations such as the American College of Obstetricians and Gynecologists (ACOG), the Royal College of Obstetricians and Gynecologists (RCOG), and the International Federation of Gynecology and Obstetrics (FIGO), the only indication for this procedure is the patient's informed desire to eliminate future fertility.[19] Sterilization requires informed, voluntary consent, consideration of alternatives, and alignment with reproductive goals; high-risk conditions alone don't mandate it.[7,9]

In this case, tubal sterilization was chosen during cesarean after counseling, representing an informed decision. Globally, it is a widely used, effective, permanent, hormone-free, and low-maintenance contraception for those who no longer desire children.[9] This case underscores the importance of thorough counseling, early detection, and prompt management in high-risk pregnancies with hypertension and preeclampsia. Grade 3 placental abruption highlights the value of multidisciplinary care to minimize complications, enhance maternal-fetal outcomes, and improve overall prognosis through close monitoring and timely intervention.

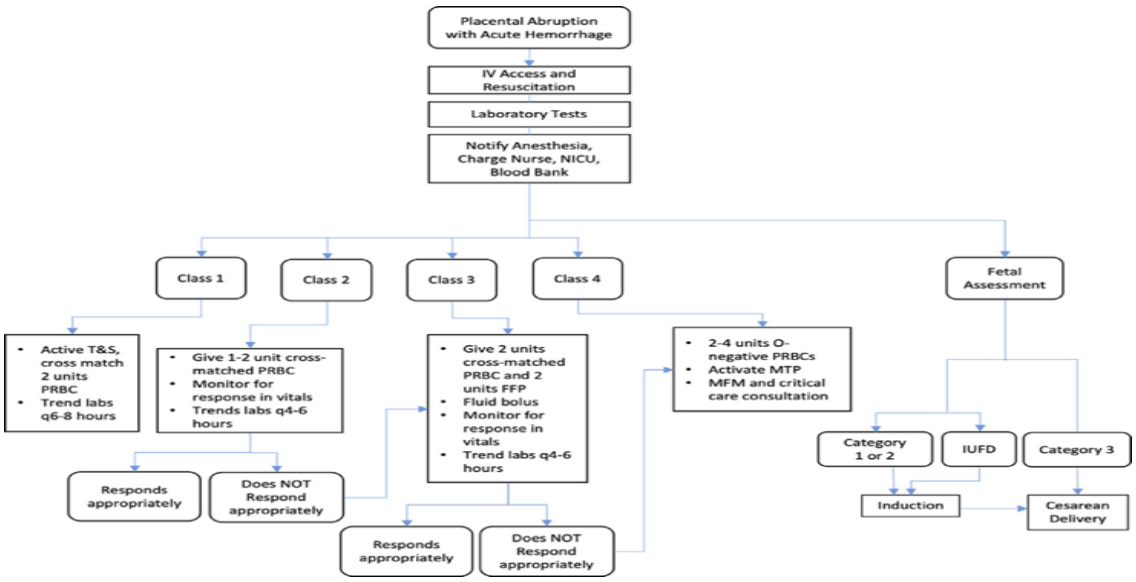

Figure 4. The algorithm for the management of placental abruption with acute hemorrhage uses the Advanced Trauma Life Support classification for hypovolemic shock, which has not been validated in pregnancy

Conclusion

This case of placental abruption underscores the combined impact of chronic hypertension and severe preeclampsia in driving adverse maternal–fetal outcomes. Early recognition, maternal stabilization, continuous fetal monitoring, and timely cesarean delivery were pivotal in reducing complications. Retroplacental hemorrhage, severe oligohydramnios, and IUGR indicated significant placental compromise from endothelial and vascular damage. Management balanced maternal and fetal needs while addressing hypoalbuminemia and coagulopathy. Postoperative recovery, NICU support for the premature neonate, and patient counseling for sterilization reflected a comprehensive care approach. Future priorities include enhancing diagnostic precision, strengthening antenatal surveillance, and developing individualized treatment strategies for complex placental pathologies.

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