
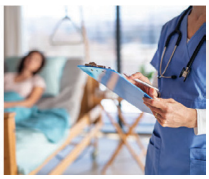

CASE REPORT

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Successful Live Birth in Partial Hydatidiform Mole: A Case Report

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ABSTRACT

Introduction: Partial hydatidiform mole (PHM) is a rare form of gestational trophoblastic disease characterized by abnormal trophoblastic proliferation accompanied by the presence of a fetus. Most cases end in miscarriage, while live births are extremely uncommon. **Case:** A 22-year-old primigravida diagnosed with PHM, intrauterine growth restriction (IUGR), and oligohydramnios underwent cesarean delivery at 35–37 weeks of gestation, resulting in a live female infant (1,750 g; Apgar scores 8/9). The enlarged placenta (1,180 g) was histopathologically confirmed as PHM. The newborn developed hypoglycemia, thrombocytopenia, and hyperbilirubinemia requiring intensive care, while the mother recovered uneventfully with declining β-hCG levels. **Discussion:** This case is remarkable as such an occurrence is extremely rare, as most PHM pregnancies end in spontaneous abortion during the first or second trimester due to chromosomal abnormalities and placental insufficiency. Postpartum follow-up revealed a gradual decline in β-hCG levels until they returned to normal, indicating the absence of persistent gestational trophoblastic disease. **Conclusion:** Pregnancies complicated by PHM require multidisciplinary care and close monitoring, including vigilance during the antenatal period, postpartum evaluation, and appropriate neonatal management to prevent and manage potential complications. Although rare and often associated with poor outcomes, PHM with live birth can achieve favorable results.

Keywords: Case report, intrauterine growth restriction, live birth, oligohydramnios, partial hydatidiform mole.

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INTRODUCTION

Gestational trophoblastic disease (GTD) is an abnormal pregnancy characterized by excessive trophoblastic proliferation, with benign to malignant biological potential. The most common form of GTD is hydatidiform mole, which is histopathologically classified as complete or partial.[1] In a complete mole, trophoblastic tissue shows diffuse proliferation without the presence of a fetus, whereas in a partial mole, fetal tissue is still present alongside an abnormal placenta. A partial mole is generally caused by dispermy, in which a single normal haploid ovum is fertilized by two spermatozoa, resulting in a triploid karyotype (69, XXY; 69, XXX; or 69, XYY). This triploid karyotype allows partial fetal development; however, growth is usually impaired due to chromosomal abnormalities and placental dysfunction.[2]

Globally, the incidence of hydatidiform mole is approximately 1–2 per 1,000 pregnancies. The rate is considerably higher in Southeast Asia, including Indonesia, where it has been reported to reach 10 per 1,000 pregnancies.[3] Geographic variation has also been observed: a study from Turkey reported a 3.17% prevalence in pregnancies, with partial moles accounting for approximately 2.28%.[4] Research from India found a predisposition to partial mole in about 3 per 1,000 pregnancies.[2] Epidemiologically, partial mole is less common than complete mole but remains clinically significant.[5]

Although a partial mole can involve some degree of fetal development, most pregnancies end in spontaneous abortion during the first or second trimester. When the fetus survives to a viable gestational age, obstetric complications such as intrauterine growth restriction (IUGR), oligohydramnios, preeclampsia, and preterm delivery are frequently observed.[1,6] The overall fetal prognosis is generally poor, with high perinatal morbidity and mortality rates.

Reports of live births in pregnancies complicated by partial mole are extremely rare, and similar publications from Indonesia are also limited. This case report is the first documented case in East Kalimantan of a live birth in a pregnancy complicated by partial hydatidiform mole accompanied by oligohydramnios and IUGR, successfully delivered via cesarean section at preterm gestation.

CASE

A 22-year-old female, $G_1P_0A_0$ (primigravida), presented at 30–32 weeks of gestation with severe nausea and vomiting. The patient had no history of allergies, chronic illnesses, contraceptive use, or previous obstetric complications. Her family history was unremarkable, with no similar conditions or known cases of GTD among close relatives.

An initial ultrasonographic examination performed at 14–16 weeks of gestation revealed a fetus accompanied by multiple echogenic areas resembling small vesicles within the placenta, consistent with a PHM (**Figure 1**). This finding served as the basis

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for the initial diagnosis of PHM in the current pregnancy.

Upon hospital admission at 30–32 weeks of gestation, the patient's vital signs were within normal limits. She weighed 88 kg and measured 157 cm in height (BMI 35.7 kg/m²). Physical examination revealed a uterine fundal height appropriate for gestational age, with no signs of vaginal bleeding or uterine tenderness. Fetal heart tones were normal. A repeat ultrasonographic examination demonstrated heterogeneous echogenic areas within the placenta, suggestive of molar changes.

Initial laboratory evaluation revealed an elevated serum β-hCG level of 802.6 IU/L; in typical complete mole cases, β-hCG levels are generally much higher due to more extensive trophoblastic proliferation.[7] Routine hematologic tests showed leukocytosis (11,060/µL) and mild anemia with a hemoglobin level of 11.4 g/dL. Platelet count and coagulation profile were normal. Thyroid function tests showed FT_4 0.99 ng/dL and TSH 3.56 µIU/mL. Other biochemical parameters, including renal function, were within normal ranges, excluding systemic complications such as hyperthyroidism or preeclampsia.

The patient was closely monitored throughout pregnancy with serial ultrasonographic examinations. At 35–37 weeks of gestation, she returned for antenatal care, and ultrasound estimated a fetal weight of 1,790 grams with a decreased amniotic fluid volume (AFI 3 cm). Based on these findings, the patient was admitted for planned pregnancy termination. During hospitalization, cardiotocography (CTG) revealed signs of fetal distress, prompting an emergency cesarean section.

The cesarean section resulted in the delivery of a live female infant weighing 1,750 grams and measuring 45 cm in length, with Apgar scores of 8 and 9 at the first and fifth minutes, respectively. Macroscopically, the placenta appeared enlarged, weighing 1,180 grams, and exhibited vesicular changes characteristic of PHM **(Figure 2)**.

Histopathological examination of the placenta confirmed the diagnosis of a PHM. Microscopic evaluation revealed a mixture of normal villi and villi exhibiting hydropic degeneration, some showing fibrosis, along with characteristic diagnostic features such as scalloped villous borders, central cistern formation, and trophoblastic inclusions **(Figure 3)**.

The newborn was admitted to the neonatal intensive care unit for evaluation and management of metabolic and hematologic complications. Laboratory tests revealed severe hypoglycemia (random blood glucose, 15 mg/dL), severe thrombocytopenia (6,700/µL), hemoglobin level of 11.1 g/dL, and elevated indirect bilirubin of 4.2 mg/dL. The infant received intravenous glucose therapy to correct the hypoglycemia and was closely monitored for signs of bleeding or hyperbilirubinemia. Supportive neonatal care led to gradual clinical improvement.

The mother's postoperative condition remained stable without complications. Both the mother and the infant were discharged in good condition after 8 days, with recommendations for routine follow-up at the obstetrics and pediatrics outpatient clinics. A chest x-ray examination showed normal findings. Four days after the initial evaluation, the β-hCG level decreased to 38.33 IU/L, and one month postpartum, further dropped to 0.32 IU/L, confirming complete remission with no evidence of persistent trophoblastic disease or molar-related complications. **Figure 4** summarizes the treatment course.

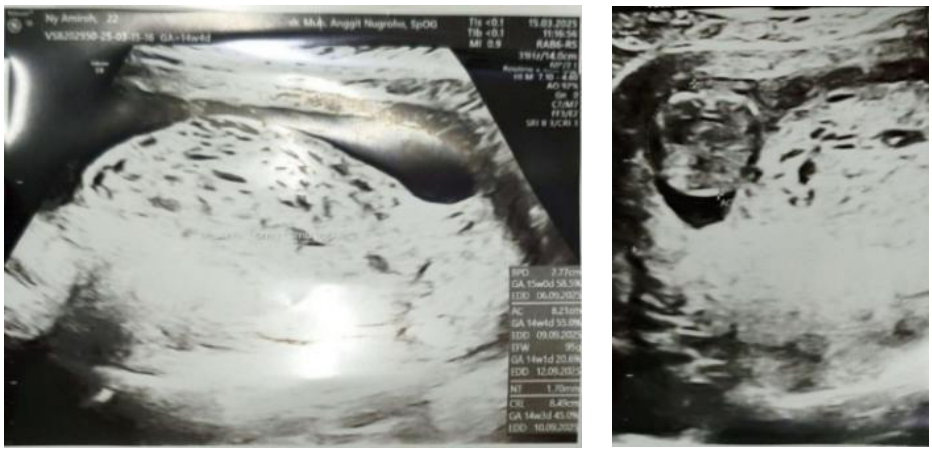

*Photo documentation by Anggia Mayangsari Wardhana.

Figure 1. Ultrasonographic findings showing heterogeneous echogenic areas suggestive of PHM.

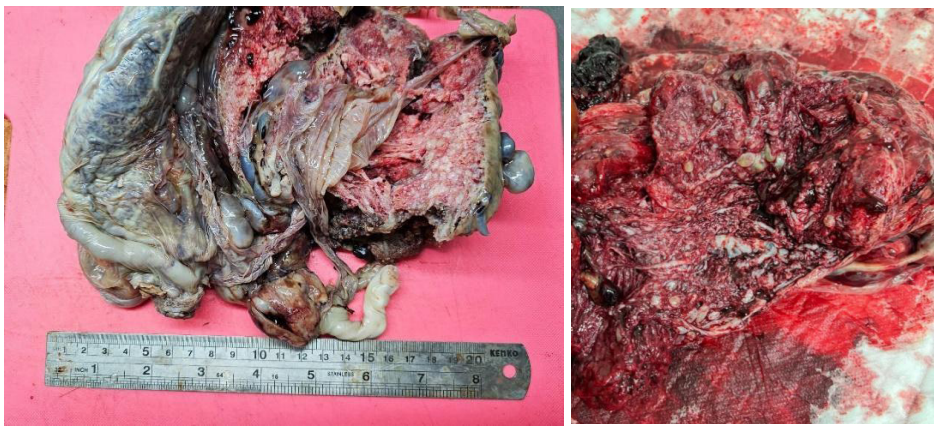

*Photo documentation by Anggia Mayangsari Wardhana.

Figure 2. Gross specimen of the placenta weighing 1,180 grams with molar changes.

DISCUSSION

PHM is a rare form of GTD characterized by abnormal trophoblastic proliferation and the coexistence of two villous populations, normal and hydropic villi.[8] This biphasic morphology distinguishes PHM from complete hydatidiform mole (CHM), which exhibits diffuse villous edema without fetal tissue. The pathogenesis of PHM is typically attributed to dispermic fertilization of a

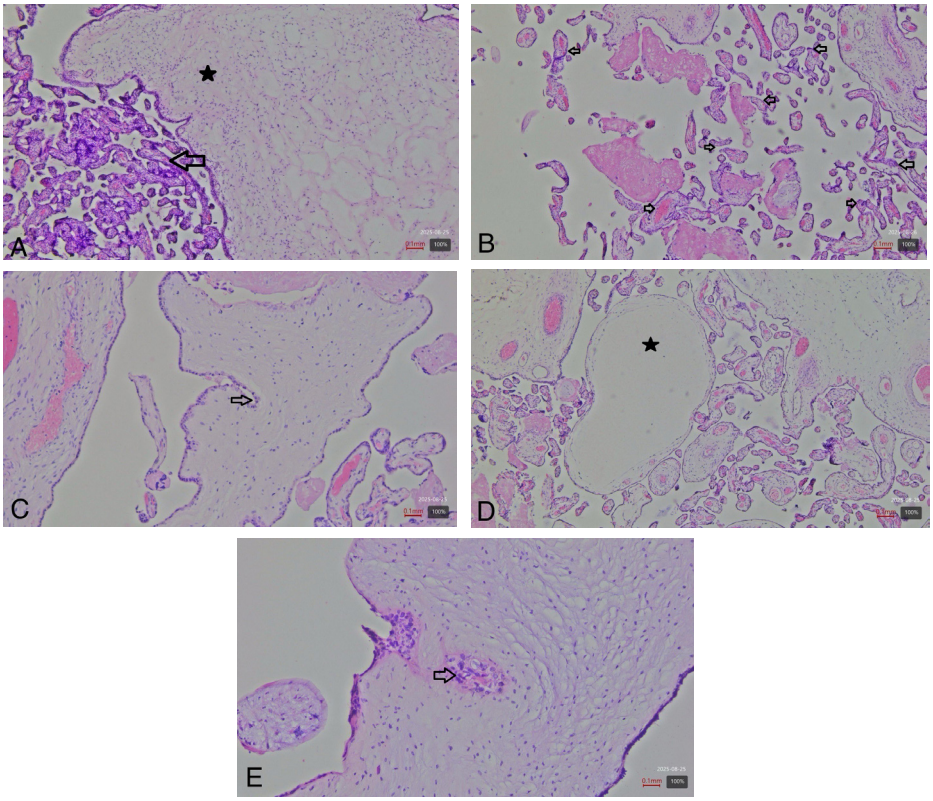

*Photo documentation by Anggia Mayangsari Wardhana.

Figure 3. Histopathological examination of the placenta. (A) two populations of villi: normal (arrow) and hydropic (asterisk); (B) fibrotic villi (arrow); (C) scalloped villous borders (arrow); (D) central cistern formation (asterisk); (E) trophoblastic inclusions (arrow).

single haploid ovum, resulting in a triploid karyotype (69, XXY/XXX/XYY).[9,10] The addition of an extra paternal genome leads to overexpression of trophoblastic genes, excessive yet disorganized proliferation, and the formation of an abnormal placenta that disrupts maternal–fetal exchange.

Epidemiologically, PHM occurs in approximately 3 per 1,000 pregnancies, whereas CHM affects about 1–3 per 1,000 pregnancies.[10] Approximately 60% of PHM cases are initially misdiagnosed as incomplete or missed abortion.[11] Unlike CHM, PHM often presents with only mildly elevated β-hCG levels, a uterus of normal or slightly enlarged size, and, in some cases, a viable fetus.[12] These subtle clinical findings frequently delay diagnosis, making histopathological examination essential for confirmation.[8]

Most PHM pregnancies end in early miscarriage due to severe genetic incompatibility and profound placental dysfunction.[1,13] However, in rare instances, the presence of partially normal villi allows limited fetoplacental exchange, enabling the pregnancy to continue into the mid or late trimester. Abnormal hydropic villi, stromal edema, and cistern formation markedly reduce the effective surface area for oxygen and nutrient transfer, leading to placental

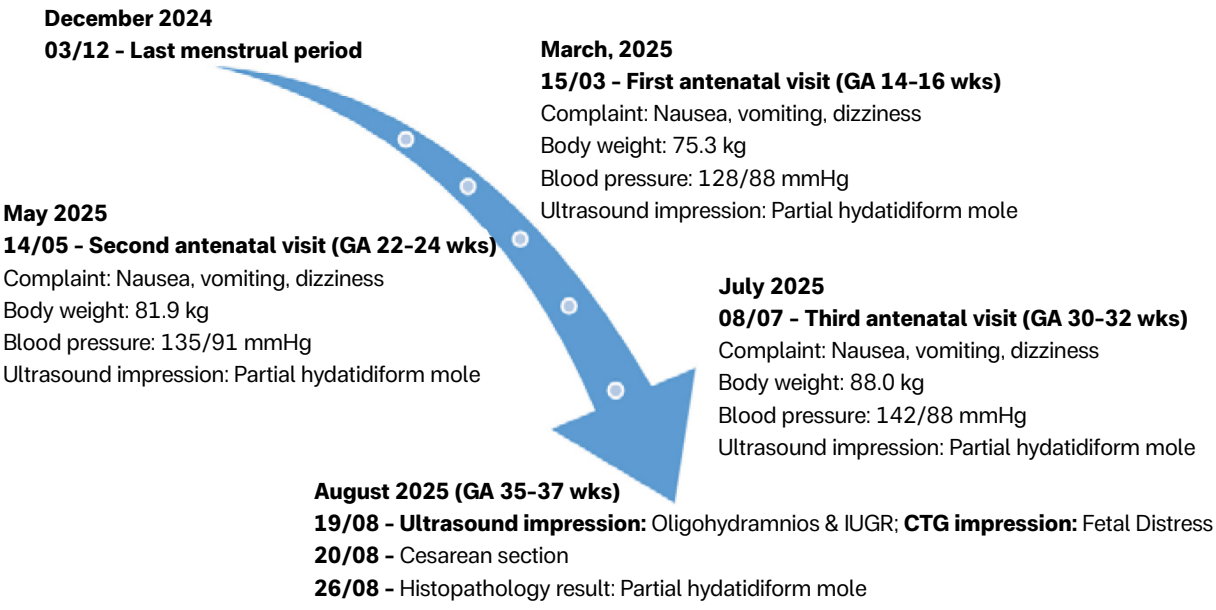

Figure 4. Timeline of the reported case.

Abbreviations: GA: Gestational age; KG: Kilogram; mmHg: millimeters of mercury; IUGR: Intrauterine growth disorder; CTG: Cardiotocography.


insufficiency, chronic fetal hypoxia, and oligohydramnios secondary to decreased fetal urine production.[8,14] This mechanism explains the frequent association of PHM with IUGR and neonatal morbidity.[3]

The present case is noteworthy because the pregnancy progressed to 35–37 weeks of gestation with a live-born infant, despite severe oligohydramnios, IUGR, fetal distress, and placentomegaly (1,180 g). The relatively low maternal β-hCG level (802.6 IU/L) reflects limited trophoblastic proliferation and accounts for the absence of complications such as hyperthyroidism or preeclampsia, which are commonly observed in CHM.[12,15] β-hCG levels in PHM are lower than in CHM due to a smaller amount of proliferating trophoblastic tissue. At the same time, some villi retain normal differentiation, resulting in reduced hCG secretory activity. Furthermore, the persistence of fetal tissue helps maintains endocrine balance within the placenta, thereby preventing excessive hCG production.[7,12]

Histopathological examination confirmed the diagnosis of PHM, showing normal and hydropic villi with scalloped borders, cistern formation, and trophoblastic inclusions, hallmark features of this condition.[8] It is important to note that in PHM, serum β-hCG levels are often only mildly elevated or even appear "normal" for gestational age; therefore, a single β-hCG value (such as 802.6 IU/L in this patient) cannot serve as a reliable diagnostic indicator, unlike in CHM where levels are typically markedly elevated. Diagnosis of PHM should rely on a correlation of ultrasonographic findings, histopathological confirmation of the placenta, and serial postpartum β-hCG monitoring.[7,12]

Neonatal complications such as thrombocytopenia,[16] hypoglycemia,[17,18] and hyperbilirubinemia[19] are likely due to chronic intrauterine hypoxia and impaired transplacental metabolism. Despite these complications, neonatal survival was achieved through vigilant antenatal surveillance, timely cesarean delivery, and multidisciplinary neonatal care.[3,5,20] This case highlights that although PHM is generally associated with poor prognosis, favorable outcomes can still be achieved through optimal maternal–fetal monitoring and preparedness for neonatal management. Long-term follow-up, including β-hCG regression monitoring and assessment of the child's neurodevelopmental progress, remains essential.

CONCLUSION

PHM with live birth is an extremely rare condition, often accompanied by both maternal and neonatal complications. This case represents the first reported instance in East Kalimantan, detected at 14–16 weeks of gestation and successfully progressing to late preterm with a live-born infant despite IUGR, oligohydramnios, and fetal distress. Both the mother and the infant recovered well after delivery. This favorable outcome highlights the importance of early detection, accurate histopathological diagnosis, vigilant maternal–fetal monitoring, and multidisciplinary perinatal management. Reporting such rare cases contributes to improved clinical understanding and may aid future decision-making in managing high-risk pregnancies.

Consent for Publication

Informed consent for publication of this case report and the accompanying clinical images was obtained from the patient.

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Author Contributions

AMW contributed to the conception and design of the case report, patient management and data collection, acquisition and interpretation of clinical data, preparation of the figures and clinical documentation, literature review, drafting and critical revision of the manuscript, and approval of the final version of the manuscript.

Conflict of Interest

The author declares no conflicts of interest.

Declaration on the Use of Artificial Intelligence (AI)

The author declares that no artificial intelligence tools were used in the conduct of this study or preparation of this manuscript.

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