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**Case Report**

# A twin pregnancy with partial hydatidiform mole, low-lying placenta and coexistent twin pregnancy with fatal obstetrics haemorrhage: a rare catastrophic case report

**Ruby Bhatia, Sheena Manktala, Vidushi Tewari*, Kashish Singla**

Department of Obstetrics and Gynecology, M. M. Institute of Medical Sciences and Research, Ambala, Haryana, India

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***Correspondence:**
Dr. Vidushi Tewari,
E-mail: vidushi.t.06@gmail.com



## ABSTRACT

The coexistence of twin gestation and partial hydatidiform mole (PHM) is an exceptionally rare obstetric condition, with fewer than 100 cases documented in world literature. A 28-year-old unbooked G4P3L2 woman presented to our tertiary care centre at 17 weeks and 4 days of gestation with a 15-day history of per vaginal bleeding and features of haemodynamic collapse (pulse rate 140 bpm; blood pressure 80/50 mmHg). Investigations revealed catastrophic anaemia (haemoglobin 3.1 g/dl), markedly elevated serum beta-hCG (>100,000 mIU/ml), coagulopathy (INR 2.83), and acute kidney injury (creatinine 1.31 mg/dl). Thyroid-stimulating hormone was suppressed at 0.005 uIU/ml, consistent with hCG-mediated thyrotoxicosis. Ultrasonography confirmed a diamniotic dichorionic twin gestation with an anterior placenta praevia displaying enlarged, heterogeneous echotexture covering the internal os. Emergency hysterotomy with B-Lynch compression sutures was performed. Intraoperative findings revealed vesicular products of conception alongside both male fetuses (each 182 g; Apgar scores zero). Placental biopsy demonstrated two discrete populations of villi irregularly shaped hydropic villi with cisterns, scalloping, and mild circumferential trophoblastic hyperplasia alongside normal tertiary villi confirming partial hydatidiform mole. No choriocarcinoma was identified. Despite aggressive resuscitation including inotropic support, mechanical ventilation, and multiple blood product transfusions, the patient developed refractory cardiac asystole and died approximately 14 hours postoperatively. This case underscores the lethal potential of PHM with twin gestation and placenta praevia, and highlights critical deficiencies in antenatal surveillance, referral systems, and resource availability that predispose unbooked patients to preventable maternal mortality. Enhanced first-trimester ultrasound protocols and routine beta-hCG measurement in twin pregnancies are urgently needed.

**Keywords:** Partial hydatidiform mole, Twin molar pregnancy, Placenta praevia, Massive obstetric haemorrhage, Maternal death, Hysterotomy, B-Lynch suture, Beta-Hcg, Coagulopathy

## INTRODUCTION

Hydatidiform mole coexisting with a viable twin fetus is an uncommon complication of pregnancy, estimated to occur in approximately 1 in 22,000 to 1 in 100,000 pregnancies.[1] The rarer subtype—partial hydatidiform mole (PHM) with concurrent twin gestation—is even more infrequently documented, with fewer than 100 cases reported in the indexed literature.[2] The diagnosis carries grave implications: elevated maternal risk of persistent gestational trophoblastic disease, haemorrhagic complications, thromboembolic events, and early-onset pre-eclampsia, alongside near-universal perinatal loss.[3] When superimposed upon placenta praevia in a haemodynamically compromised, unbooked patient with no access to prior antenatal care, the prognosis becomes catastrophic.

PHM is characterised pathologically by the presence of two discrete populations of chorionic villi hydropic, cistern-forming villi and morphologically normal tertiary villi accompanied by mild trophoblastic hyperplasia and a triploid or diploid karyotype, most commonly arising from dispermy.[4] The coexistence of PHM with a twin gestation creates a diagnostic and therapeutic dilemma: beta-hCG concentrations are markedly elevated, often disproportionate to gestational age; ultrasound findings of placental heterogeneity and cystic change may be misinterpreted as bleeding or degenerative placental change; and the haemorrhagic propensity of molar tissue in the context of an actively bleeding placenta praevia is potentially lethal.[5]

Maternal death from obstetric haemorrhage remains a leading cause of preventable mortality globally, accounting for approximately 27% of all maternal deaths, with the majority occurring in low- and middle-income countries.[6] Late or absent antenatal care, delayed referral, and limited access to blood products and intensive care disproportionately amplify this risk. The present case exemplifies the intersection of a rare obstetric pathology with systemic healthcare access barriers, and is reported to contribute to the body of literature on this rare entity, to advocate for timely diagnosis, and to draw attention to preventable maternal mortality.

## CASE REPORT

### *Clinical history and initial assessment*

A 28-year-old unbooked woman (G4P3L2) was referred to the obstetrics emergency department of our tertiary care institution from a District Women's Hospital with a history of amenorrhoea of four months' duration and per vaginal bleeding of 15 days. Her obstetric history included two uncomplicated normal vaginal deliveries at term (P1: female, 4 years; P2: male, 2 years) and one prior twin pregnancy at 7 months of gestation, both fetuses being female, with the first expiring within 12 hours of birth and the second within 24 hours of birth for undetermined reasons.

The current pregnancy was reported to be spontaneously conceived. She had received one unit of packed red blood cells (PRBC) at the referring hospital three days prior to transfer. Her medical and surgical history was unremarkable; she denied any history of hypertension, diabetes mellitus, asthma, epilepsy, hepatic disease, or thyroid disorder.

On arrival, the patient was critically unwell and was bleeding actively. Clinical examination demonstrated marked pallor, pedal oedema (++). Vital signs were as follows: pulse rate 140 bpm; blood pressure 80/50 mmHg, Afebrile, oxygen saturation 96% on room air; respiratory rate 26 breaths per minute. Cardiovascular examination S1, S2 present with no murmurs. Chest auscultation was clear bilaterally with equal air entry. Abdominal examination demonstrated a uterus of 26 weeks' size more than period of gestation, non-tender, non-tense, with multiple fetal parts palpable; fetal heart sounds of both twins could not be localised.

### *Investigations*

Laboratory investigations (Table 1) demonstrated severe micronormocytic anaemia (haemoglobin 3.1 g/dl; PCV 10.3%), leukocytosis (TLC 29.4×103/cumm) with marked neutrophilia (93%), normal platelet count, significantly elevated serum beta-hCG (>100,000 mIU/ml), and coagulopathy with prolonged prothrombin time (INR 2.83; PT patient 30.6 seconds vs. control 10.8 seconds). Renal function revealed creatinine 1.31 mg/dl and blood urea 39.3 mg/dl, consistent with acute kidney injury (AKI).

Hepatic function demonstrated elevated alkaline phosphatase (257 U/l), hypoalbuminaemia (1.5 g/dl), and hypoproteinaemia (3.2 g/dl). Random blood sugar was 331 mg/dl, likely stress-related. Serum electrolytes revealed hypernatraemia (155 mEq/l) and hyperkalaemia (5.1 mEq/l). Thyroid stimulating hormone (TSH) was suppressed at 0.005 uIU/ ml, consistent with hCG-mediated thyroid stimulation.

### *Ultrasonographic and radiological findings*

First trimester ultrasonography performed at 13 weeks and 6 days (Figure 1) at the referring centre confirmed two live intrauterine fetuses: fetus A on the right side of the maternal spine, with a crown-rump length (CRL) of approximately 75 mm corresponding to 13 weeks and 6 days (Hadlock), with cardiac activity present, posterior placenta grade I, and adequate amniotic fluid; and fetus B on the left side of the maternal spine, with a CRL of approximately 67 mm corresponding to 13 weeks and 1 day (Hadlock), with cardiac activity present. Critically, the placenta of fetus B was described as anterior, enlarged, and heterogeneous in echotexture, covering the internal os (low lying placenta placenta praevia), with a radiological impression of placenta praevia for the second anterior placenta. The overall ultrasound impression was diamniotic dichorionic twin pregnancy.

### *Operative management*

Given the haemodynamically unstable condition with massive haemorrhage. The patient was taken up for emergency hysterotomy on 19 March 2025. A transverse uterine incision was made. The placenta was found to be covering the internal os completely and was showing grape like vesicles with two male fetuses (twin A and twin B), each with a birth weight of 182 g, delivered at 10:00 AM on 19 March 2025. Both infants were still born. The uterus appeared atonic and flabby throughout; oxytocin and tranexamic acid were administered intravenously without sustained response. B-Lynch compression sutures were applied bilaterally. Complete haemostasis was achieved; uterine incision was closed in double layers.

Intraoperatively, 3 units of PRBC and 4FFP were transfused along with initiation of inotropic support. Urine output via catheter measured approximately 400 ml intraoperatively.

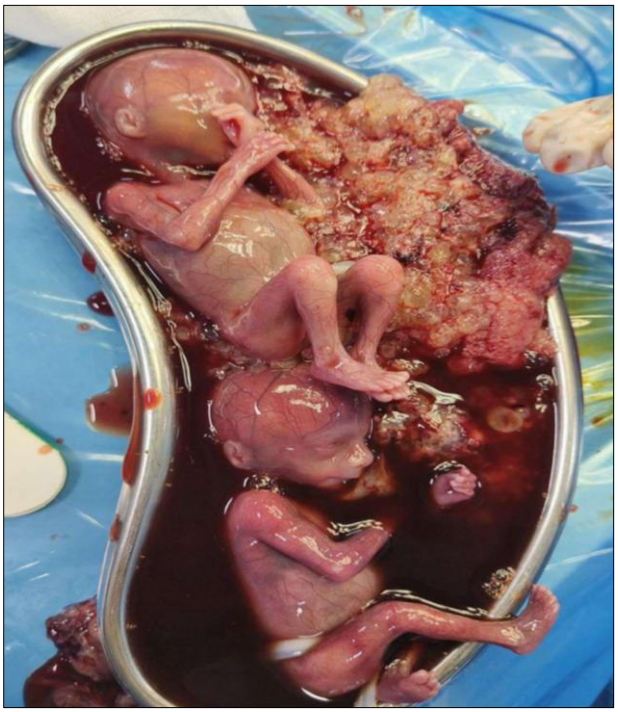

**Figure 1: Intraoperative photograph following emergency hysterotomy demonstrating both male fetuses (twin A and twin B) delivered at 17 weeks 4 days of gestation, each weighing 182 g, with Apgar scores of zero at one and five minutes, consistent with previable fetal demise. Note the absence of maceration, indicating acute intraoperative or immediately perioperative fetal expiry.**

***Postoperative course and maternal outcome***

Postoperatively, the patient was transferred to the respiratory intensive care unit (RICU) and placed on mechanical ventilatory and inotropic support. Postoperatively, 1-unit PRBC and 2 units of fresh frozen plasma (FFP) were transfused. Despite these measures, the patient's haemodynamic status continued to deteriorate, necessitating escalation to triple inotropic support. A multidisciplinary team comprising obstetrics, critical care medicine, nephrology, and haematology was convened. At 12:00 AM on 20 March 2025 approximately 14 hours postoperatively the patient developed cardiac asystole. Five cycles of cardiopulmonary resuscitation (CPR) were administered without return of spontaneous circulation. The patient was declared deceased at 12:45 AM on 20 March 2025.

The certified cause of death was: primary cause irreversible hypovolaemic shock secondary to partial hydatidiform mole with massive haemorrhage; secondary cause very severe anaemia with severe metabolic acidosis; tertiary cause acute kidney injury.

***Histopathological findings***

Placental tissue biopsy (HPR No. 1650/25; Department of Obstetrics and Gynaecology, Ward W38/RICU/RICU-1) revealed a specimen measuring 1×16.5×4 cm with attached umbilical cord measuring 2 cm in length. Numerous grape-like vesicles with clear fluid were noted on gross examination. Microscopically, sections demonstrated two discrete populations of villi: irregularly shaped, large hydropic villi with cisterns and scalloping, and mild circumferential trophoblastic hyperplasia; and morphologically normal tertiary villi. No fetal erythrocytes or fetal tissue parts were identified within the villi. Histopathological impression: features consistent with partial hydatidiform mole (Figure 2).

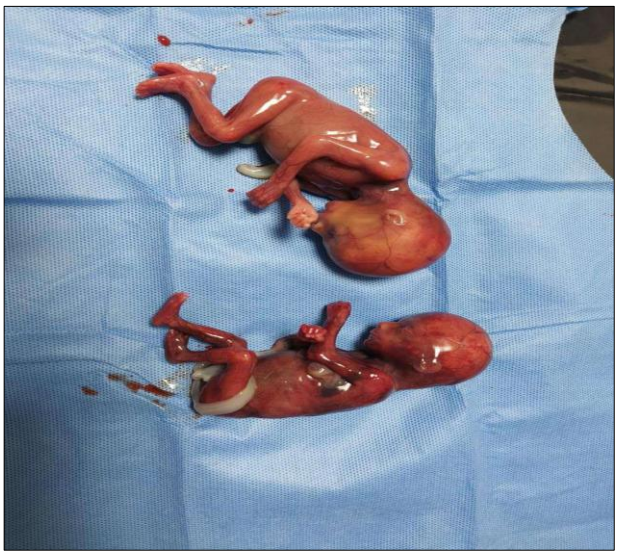

**Figure 2: Close-up intraoperative photograph depicting both evacuated fetuses alongside the hydropic molar placental tissue, demonstrating the characteristic vesicular (grape-like) products of conception admixed with the fetal products, consistent with partial hydatidiform mole in twin gestation. The vesicular tissue is clearly distinguishable from the normal fetal structures.**

## DISCUSSION

The present case represents an exceptionally rare and fatal convergence of partial hydatidiform mole, twin gestation, low lying placenta (placenta praevia), and massive obstetric haemorrhage in an unbooked patient with no prior antenatal care. To the best of our knowledge, a case combining all four of these entities with a maternal death outcome has not been previously described in the indexed literature, underscoring its singularity and the importance of its documentation.

***Epidemiology and pathogenesis of partial molar twin pregnancy***

Complete hydatidiform mole coexisting with a normal twin is more frequently reported than PHM with twin gestation.[2] PHM is characterised by diandric triploidy (typically 69, XXY or 69, XXX) arising from fertilisation of a single ovum by two spermatozoa, resulting in a partial molar placenta alongside a triploid, often malformed fetus.[4] In the subset of PHM with normal co-twin, the molar component typically involves one placenta in a dichorionic gestation, while the co-twin may have a structurally normal diploid karyotype and normal placenta.[3] In the present case, the gross hydropic vesicles, the markedly elevated beta-hCG (>100,000 mIU/ml), and the histological features of two villous populations with trophoblastic hyperplasia are consistent with this diagnosis, pending confirmatory p57 IHC and genotyping as recommended by the pathologist.

***Diagnostic challenges and the role of first-trimester ultrasound***

The first-trimester ultrasound report in the present case documented an enlarged, heterogeneous anterior placenta covering the os in the context of live twin gestation but did not explicitly raise the possibility of gestational trophoblastic disease (GTD). This is a recognised diagnostic pitfall: the sonographic features of PHM — echogenic placenta with multiple cystic spaces, enlarged placenta, and elevated beta-hCG — can be subtle in the first trimester, particularly in the context of multi-fetal gestation where placental heterogeneity may be attributed to haematoma or normal variation.[7] Sensitivity of ultrasound for PHM diagnosis ranges from 17–29% in the first trimester.[8] Serum beta-hCG quantification is an indispensable adjunct; values disproportionately elevated for gestational age should prompt dedicated placental evaluation and consideration of GTD. In this case, the absence of beta-hCG measurement at the referring facility and the absence of formal antenatal care represent critical missed diagnostic opportunities.

***Placenta praevia in the context of molar tissue***

Placenta praevia — defined as placental implantation overlying or within 2 cm of the internal cervical os — occurs in approximately 0.5% of pregnancies and is a well-recognised cause of antepartum haemorrhage.[9] In the setting of molar placental tissue, the haemorrhagic risk is compounded by the inherent vascularity and friability of trophoblastic tissue, abnormal uterine invasion, and the propensity for coagulopathy secondary to trophoblast-derived procoagulant activity.[5] Combination of praevia and molar tissue in the present case created a haemorrhagic substrate that proved irreversible, precipitating the coagulopathy (INR 2.83), metabolic acidosis, and refractory haemodynamic instability that ultimately led to maternal death.

***Perioperative management and B-Lynch suture***

Emergency hysterotomy was the only viable surgical option given the gestational age, haemodynamic instability, and need for definitive control of the haemorrhaging praevia. Uterine atony following evacuation of products, compounded by the contractile dysfunction associated with the grossly enlarged and abnormal uterus, necessitated B-Lynch compression suturing — a procedure first described by B-Lynch et al in 1997 and now widely employed for refractory postpartum haemorrhage.[10] administration of uterotonics (oxytocin) and antifibrinolytics (tranexamic acid) was consistent with current WHO and FIGO guidelines for management of postpartum haemorrhage.[11] Despite achieving intraoperative haemostasis, the cumulative physiological insult — prolonged shock, severe anaemia, coagulopathy, AKI, and metabolic acidosis — exceeded the patient's physiological reserve and resuscitative capacity.

***Suppressed TSH and beta hCG-mediated thyrotoxicosis***

The markedly suppressed TSH (0.005 uIU/ml) observed in this patient is consistent with hCG-mediated subclinical or overt thyrotoxicosis, a recognised complication of gestational trophoblastic disease due to structural homology between beta-hCG and thyroid-stimulating hormone.[12] In the acute setting, this finding may have contributed to the tachycardia and haemodynamic instability, further compounding the clinical picture. Clinicians managing suspected molar pregnancies should routinely include TSH measurement in their biochemical assessment.[3,6]

***Systemic barriers and preventable maternal death***

Maternal mortality from obstetric haemorrhage in India, while declining, remains substantially higher than in high-income settings, with systemic factors including unbooked status, ANC USG, delayed referral, limited blood bank capacity, and absence of point-of-care coagulation management contributing disproportionately to adverse outcomes.[13] In the present case, the patient had received only one unit of PRBC prior to referral despite a haemoglobin consistent with near-fatal anaemia. The 15-day duration of haemorrhage prior to definitive care represents a critical failure in the referral chain. Population-level education regarding danger signs in pregnancy, mandatory first-trimester ultrasound with beta-hCG correlation, and strengthened inter-facility referral protocols are imperative systemic interventions.

## CONCLUSION

We present a fatal case of twin pregnancy complicated by partial hydatidiform mole, anterior placenta praevia, and massive obstetric haemorrhage in an unbooked, critically anaemic patient, resulting in the demise of both fetuses and the mother. This case is, to the best of our knowledge, among the most clinically severe presentations of this rare

obstetric entity documented in the indexed literature. It reinforces several critical clinical lessons: the necessity of first-trimester beta-hCG measurement and placental sonographic assessment in all twin pregnancies; the imperative of early hospital-based antenatal care for unbooked patients; the vital importance of timely and adequate blood product availability in centres managing high-risk obstetric cases; and the potentially fatal consequences of delayed recognition and referral in the setting of molar disease. We hope this case contributes to the growing literature on gestational trophoblastic disease in the context of multi-fetal pregnancy and stimulates institutional and policy-level dialogue on the prevention of such tragedies.

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