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From a suspected sinusoidal fetal heart rate pattern to postpartum choriocarcinoma: a case report of severe fetal anemia associated with fetomaternal hemorrhage

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Case Report

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Abstract

Background

Gestational choriocarcinoma may follow a nonmolar pregnancy. An occult placental lesion can disrupt fetal vessels, cause fetomaternal hemorrhage and severe fetal anemia, and escape routine placental sampling. Recognition is difficult when fetal compromise precedes maternal symptoms.

Case presentation:

A 35-year-old Chinese woman, gravida 2 para 1, presented at 34 weeks and 4 days of gestation after markedly decreased fetal movement for 1 day, with only one perceived movement that day. Cardiotocography was clinically interpreted as a Category III fetal heart rate tracing because of a suspected sinusoidal pattern. Fetal middle cerebral artery peak systolic velocity was 96.92 cm/s (1.94 multiples of the median). Emergency cesarean delivery was performed for nonreassuring fetal status and suspected severe fetal anemia. A 2060-g female infant was profoundly pale; total hemoglobin was 3.2 g/dL and hematocrit was 12.1%. Respiratory and circulatory support and partial exchange transfusion increased hemoglobin to 14.8 g/dL, and the infant was discharged in stable condition on day 16. A maternal acid-elution assay reported an HbF fraction of 7.01%, supporting fetomaternal hemorrhage. The friable placenta showed no trophoblastic tumor on initial examination or re-examination of retained blocks. Approximately 1 month postpartum, the mother developed recurrent vaginal bleeding. Ultrasonography demonstrated a vascular intrauterine lesion, and serum beta-human chorionic gonadotropin was 52,714 IU/L. Hysteroscopic and ultrasound-guided evacuation showed markedly atypical mixed trophoblastic proliferation without chorionic villi; morphology and immunohistochemistry supported gestational choriocarcinoma.

Conclusions

Severe unexplained fetal anemia with fetomaternal hemorrhage may be the first clinical manifestation of gestational choriocarcinoma. Immediate fetal assessment and delivery when indicated can enable neonatal survival. Targeted extensive placental sampling, maternal postpartum beta-human chorionic gonadotropin testing, and coordinated maternal-neonatal surveillance should be considered even when routine placental histology is negative.

Background

Gestational choriocarcinoma is a highly malignant gestational trophoblastic neoplasm composed of cytotrophoblasts, syncytiotrophoblasts and intermediate trophoblasts. It is characterized histologically by the absence of chorionic villi, extensive hemorrhage and necrosis, and a propensity for hematogenous dissemination [1, 2]. It may occur after a molar or nonmolar pregnancy. When the primary

lesion is located within the placenta, intraplacental choriocarcinoma is exceptionally rare and probably underdiagnosed.

A national gestational trophoblastic disease database and systematic review identified eight cases among 27,101 gestational trophoblastic disease registrations (0.03%) and summarized 62 published and database cases [3]. Half of the mothers were asymptomatic and 29% of placentas were macroscopically normal. Of 58 cases with reported fetal outcomes, 38 resulted in live births and 20 in intrauterine deaths; two neonatal deaths were also reported [3]. A systematic review focused on intraplacental choriocarcinoma with fetomaternal hemorrhage identified 19 cases and reported a perinatal mortality rate of 36.8% [4].

The present case is distinct because a preterm fetus first signaled the disease through markedly reduced movement, a suspected sinusoidal fetal heart rate pattern, a fetal middle cerebral artery peak systolic velocity (MCA-PSV) of 1.94 multiples of the median (MoM), and a hemoglobin concentration of 3.2 g/dL at birth. Urgent delivery and intensive neonatal treatment were successful. Maternal symptoms appeared only postpartum, and choriocarcinoma was confirmed in intrauterine tissue despite negative initial and repeat examination of the available placental blocks. This sequence illustrates both the diagnostic value of the fetal presentation and the limitations of routine placental sampling.

Case presentation

Antenatal course and emergency assessment

A 35-year-old Chinese woman, gravida 2 para 1, had previously delivered a healthy girl by cesarean section. Gestational age was established by first-trimester ultrasonography. Antenatal screening for trisomy 21 was low risk and oral glucose tolerance testing was normal. Fetal magnetic resonance imaging at 29 weeks and 4 days suggested possible bilateral pseudocysts adjacent to the frontal horns of the lateral ventricles. At 33 weeks, maternal protein S activity was 26.20%; anticardiolipin antibodies, antinuclear antibodies and anti-beta2-glycoprotein I antibodies were negative. She had chronic hepatitis B virus infection, with an HBV DNA level of 26 IU/mL approximately 1 month before delivery.

At 34 weeks and 4 days, she presented after a marked reduction in fetal movement for 1 day and reported only one perceived movement that day. Vital signs were stable, and there was no abdominal pain or vaginal bleeding. Cardiotocography was clinically interpreted by the treating team as a Category III fetal heart rate tracing because of a suspected sinusoidal pattern (Fig. 1A). Ultrasonography showed a singleton live fetus in cephalic presentation. Fetal middle cerebral artery peak systolic velocity (MCA-PSV) was 96.92 cm/s, corresponding to 1.94 MoM. Given the acute reduction in fetal movement, Category III fetal heart rate tracing and markedly elevated fetal MCA-PSV, the findings were considered indicative of nonreassuring fetal status with suspected severe fetal anemia. Emergency cesarean delivery was performed.

Delivery and neonatal management

A female infant weighing 2060 g and measuring 44 cm was delivered. Apgar scores were 7, 8 and 8 at 1, 5 and 10 minutes, respectively, with deductions mainly for muscle tone and color. The infant was profoundly pale with a weak cry (Fig. 1C). Positive-pressure ventilation was initiated with a T-piece resuscitator, followed by transfer to the neonatal intensive care unit. An arterial sample obtained immediately after birth showed pH 7.362 and total hemoglobin 3.2 g/dL. Repeat testing showed hemoglobin 32 g/L and hematocrit 12.1%, with metabolic acidosis and hyperlactatemia.

Because of extreme anemia and risk of circulatory collapse, the infant underwent endotracheal intubation, high-frequency oscillatory ventilation, umbilical arterial and venous catheterization, and partial exchange transfusion. She received 188 mL of group O RhD-positive washed red blood cells and 62 mL of group AB RhD-positive plasma. Total hemoglobin increased to 14.8 g/dL after exchange, and lactic acidosis gradually resolved without a transfusion reaction. During admission, respiratory distress, shock, thrombocytopenia, coagulopathy, risk of acute kidney injury, hypoalbuminemia and elevated markers of myocardial injury improved with supportive treatment. By day 16, she was breathing without respiratory support, taking full oral feeds and was discharged in stable condition.

The obstetric team considered fetomaternal hemorrhage the likely cause of the severe fetal anemia. A maternal acid-elution assay reported an HbF fraction of 7.01%, supporting this diagnosis.

Placental examination

The placenta delivered spontaneously during surgery and measured approximately 18 cm x 19 cm x 2 cm, with a weight of 480 g. It appeared grossly friable and fragmented easily (Fig. 1B). The placenta and membranes were complete, and intraoperative curettage showed no retained tissue. Initial histopathology showed a mature third-trimester placenta, perivillous fibrinoid deposition and sparse chronic inflammatory infiltration in the decidual and chorionic layers of the membranes. No significant umbilical cord abnormality or trophoblastic tumor was identified. Re-examination of the retained placental blocks also showed no trophoblastic neoplasm. The remaining placental tissue had already been disposed of, precluding additional sampling.

Postpartum presentation and diagnosis

Blood-stained lochia ceased approximately 10 days after cesarean delivery. At about 33 days postpartum, fresh vaginal bleeding recurred without an obvious precipitating event, initially approximating menstrual flow and then progressively increasing. On postpartum day 43, transvaginal ultrasonography showed a uterus measuring 62 mm x 66 mm x 60 mm, an endometrial thickness of approximately 16.2 mm, and a heterogeneous vascular lesion measuring 27 mm x 23 mm x 13 mm along the anterior uterine cavity, with a resistance index of 0.53. Serum beta-human chorionic

gonadotropin (beta-hCG) concentrations on postpartum days 44, 46 and 51 were 52,714, 48,306 and 49,593 IU/L, respectively.

Hysteroscopy and ultrasound-guided uterine evacuation were performed on postpartum day 52. The uterine cavity was normally shaped, and approximately 3 cm of old tissue was attached to the anterior wall. The tissue was removed with forceps and an 8-mm suction cannula; repeat inspection showed no obvious residual tissue. Estimated blood loss was 2 mL. Histological examination showed markedly atypical mixed trophoblastic proliferation in a background of blood clot and secretory endometrium, without identifiable chorionic villi and with extensive hemorrhage and necrosis. Immunohistochemistry showed diffuse pancytokeratin AE1/AE3 and hCG expression, focal hPL, p63 and alpha-inhibin expression, and a Ki-67 hotspot index of approximately 60%. External pathological consultation additionally reported SALL4 positivity. These findings supported gestational choriocarcinoma.

Serum beta-hCG initially declined after evacuation but did not normalize and subsequently increased (Table 1; Fig. 2). External pathological review confirmed choriocarcinoma. Contrast-enhanced pelvic magnetic resonance imaging showed an irregular 1.7 cm x 1.5 cm lesion in the anterior wall of the lower uterine segment, with indistinct margins and heterogeneous enhancement. Chest computed tomography showed a 0.5-cm ground-glass nodule and a 0.6-cm solid nodule in the left upper lobe. Abdominal computed tomography showed several small hypoenhancing hepatic nodules, the largest measuring approximately 1.1 cm x 0.8 cm; subsequent assessment favored hemangiomas. Cranial computed tomography and contrast-enhanced magnetic resonance imaging showed no definite abnormality. The patient was referred to a specialist gestational trophoblastic disease center for definitive staging and treatment. The available records did not include the final chemotherapy regimen or treatment outcome. (Table 2)

Table 1
Postpartum serum beta-hCG kinetics

Postpartum day	beta-hCG (IU/L)	Clinical context
44	52,714	Evaluation of intrauterine lesion
46	48,306	Persistently elevated
51	49,593	Pre-evacuation plateau
53	23,927	First day after evacuation
59	3,557.2	Continued decline
66	3,066	Persistent elevation
69	1,190.39	Interim nadir
72	2,687	Subsequent increase

Table 2
Clinical timeline

Time	Key findings	Assessment and management
34 + 4 weeks	Markedly reduced fetal movement; Category III fetal heart rate tracing because of a suspected sinusoidal pattern; fetal MCA-PSV 96.92 cm/s (1.94 MoM)	Emergency cesarean delivery for nonreassuring fetal status and suspected severe fetal anemia
Birth	Female infant, 2060 g; profound pallor; total hemoglobin 3.2 g/dL; hematocrit 12.1%	Respiratory and circulatory support and partial exchange transfusion
Postpartum day 2	Maternal acid-elution assay reported HbF 7.01%	Fetomaternal hemorrhage supported
Postpartum day 16	Breathing without support and taking full oral feeds	Discharged in stable condition
Postpartum day 33	Fresh vaginal bleeding after lochia had ceased	Further evaluation initiated as bleeding increased
Postpartum days 43–51	Vascular intrauterine lesion; beta-hCG persistently approximately 48,000–53,000 IU/L	Persistent trophoblastic activity suspected
Postpartum day 52	Hysteroscopy and ultrasound-guided evacuation	Histology and immunophenotype supported gestational choriocarcinoma
Postpartum days 53–72	beta-hCG declined and then increased; uterine lesion and indeterminate pulmonary and hepatic nodules on imaging	Referral to specialist center for staging and treatment

Discussion and conclusions

Fetal anemia as the earliest clinical signal

MCA-PSV rises in fetal anemia because reduced blood viscosity and increased cardiac output increase cerebral arterial velocity. A threshold of 1.5 MoM is widely used to identify fetuses at risk of moderate-to-severe anemia [5, 6]. In this case, MCA-PSV was 1.94 MoM and neonatal total hemoglobin was only 3.2 g/dL, demonstrating close agreement between antenatal Doppler findings and extreme anemia after birth. A true sinusoidal fetal heart rate pattern is characterized by smooth, regular oscillations at three to five cycles per minute persisting for at least 20 minutes [7]. The available tracing was highly suggestive of a sinusoidal pattern and was clinically interpreted as a Category III fetal heart rate tracing.

Reduced fetal movement is a frequent presenting feature of clinically significant fetomaternal hemorrhage. Severe cases may be associated with sinusoidal fetal heart rate patterns, hydrops, fetal death or profound anemia at birth [8–10]. Here, the maternal HbF fraction of 7.01% on acid-elution testing, extreme neonatal anemia and absence of a clear alternative source of blood loss supported substantial fetomaternal hemorrhage. Rapid integration of fetal movement, cardiotocography and MCA

Doppler findings led to emergency delivery, while controlled partial exchange transfusion avoided abrupt volume loading and was followed by neonatal recovery.

Choriocarcinoma as a possible cause of fetomaternal hemorrhage

An updated PubMed search through September 2026 using combinations of “intraplacental choriocarcinoma,” “fetomaternal hemorrhage,” “fetal anemia,” and “neonatal anemia” identified systematic reviews and closely comparable cases [3, 4, 11–19]. Intraplacental choriocarcinoma can destroy chorionic villi and fetal vessels, permitting fetal erythrocytes to enter the maternal circulation. The 2023 systematic review by Stabile et al. identified 19 cases of intraplacental choriocarcinoma complicated by fetomaternal hemorrhage, while a 2025 review identified 35 cases and recurrent warning signs including decreased fetal movement, abnormal fetal heart rate patterns, abnormal MCA Doppler findings and fetal or neonatal anemia [4, 11].

Several individual reports closely resemble the present clinical sequence (Table 3). Peng et al. described a term infant with severe anemia whose mother developed major vaginal bleeding 2 weeks after cesarean delivery; intrauterine tissue confirmed choriocarcinoma and pulmonary metastases were detected [12]. Hookins and Vatsayan and Monteiro et al. reported intraplacental choriocarcinoma associated with massive fetomaternal hemorrhage after reduced fetal movement, with disseminated intravascular coagulopathy in one mother and fetal death in the other [13, 14]. Farmer reported a late-preterm infant with hemoglobin 2.4 g/dL, a maternal acid-elution test of 7%, partial exchange transfusion and subsequent maternal choriocarcinoma [15]. Jin et al. published a term case in this journal in which decreased fetal movement, fetal distress and fetomaternal hemorrhage led to placental histological confirmation [16]. Tsuge et al. reported severe neonatal anemia of 3.2 g/dL, initially negative placental examination and postpartum maternal hemorrhage; placental re-examination subsequently demonstrated intraplacental choriocarcinoma [17].

Table 3
Published evidence most relevant to the present case

Report	Case scope	Key findings	Relevance
Jiao et al., 2016 [3]	62 intraplacental choriocarcinoma cases	50% of mothers asymptomatic; 29% of placentas grossly normal; substantial fetal mortality	Routine gross examination and limited sampling can miss focal disease
Stabile et al., 2023 [4]	19 cases with fetomaternal hemorrhage	Perinatal mortality 36.8%; recurrent fetal warning signs	Fetomaternal hemorrhage can be the presenting clue
Wang et al., 2025 [11]	35 cases with spontaneous fetomaternal hemorrhage	Reduced movement, abnormal fetal monitoring, abnormal MCA Doppler and severe anemia recurred	Supports a coordinated maternal-placental-neonatal pathway
Farmer, 2024 [15]	Late-preterm infant and maternal choriocarcinoma	Neonatal hemoglobin 2.4 g/dL; maternal acid-elution result 7%; partial exchange transfusion	Closest neonatal rescue phenotype
Jin et al., 2025 [16]	Term intraplacental choriocarcinoma	Reduced fetal movement, fetal distress and fetomaternal hemorrhage; placental pathology positive	Direct placental confirmation contrasts with the present sampling-negative case
Tsuge et al., 2026 [17]	Postpartum hemorrhage with intraplacental choriocarcinoma	Neonatal hemoglobin 3.2 g/dL; initial placental examination negative; re-examination positive	Shows why prompt retention and re-sampling of placenta matter
Present case	Preterm fetal anemia with postpartum uterine choriocarcinoma	Suspected sinusoidal fetal heart rate pattern; fetal MCA-PSV 1.94 MoM; neonatal hemoglobin 3.2 g/dL; placental blocks negative; neonatal survival	Fetal physiology can trigger successful rescue before maternal disease becomes apparent

Negative placental histology and diagnostic boundaries

The clinical mechanism strongly suggests that an occult placental trophoblastic lesion was present during pregnancy. Nevertheless, choriocarcinoma was demonstrated only in postpartum intrauterine tissue, while the original placental examination and re-examination of retained blocks were negative. The present case therefore should not be labeled pathologically proven intraplacental choriocarcinoma. This distinction is important because focal intraplacental lesions may measure only a few millimeters and resemble infarction or hematoma, and nearly one third of affected placentas have been reported as grossly normal [3].

When severe unexplained fetal anemia or fetomaternal hemorrhage is recognized, all placental tissue and blocks should be retained. Obstetricians and pathologists should jointly target friable, hemorrhagic, infarct-like or otherwise abnormal areas for extensive sampling. Immunohistochemistry may include

pancytokeratin, hCG, hPL, p63, GATA3, SALL4 and Ki-67. If lesions are found at more than one site, short tandem repeat genotyping can identify paternal alleles, support gestational origin and help establish whether tumors share a common origin [20]. Genomic and methylation analysis can provide additional evidence regarding trophoblastic origin and tumor biology [21].

Maternal and neonatal surveillance

The interval from cesarean delivery to maternal diagnostic evaluation was approximately 6 weeks, similar to the median maternal diagnostic interval reported in a systematic review of concurrent maternal and infantile gestational trophoblastic neoplasia [22]. Maternal and neonatal evaluation should proceed in parallel when choriocarcinoma is suspected: quantitative maternal beta-hCG and staging assessment; extensive placental sampling with tissue retention; and quantitative neonatal beta-hCG, blood counts, and pediatric oncology-directed imaging and longitudinal follow-up. Concurrent maternal and infantile choriocarcinoma is extremely rare but may be disseminated when recognized [22].

Long-term maternal follow-up is also important. Liu et al. reported 14 patients with pathologically confirmed intraplacental choriocarcinoma; none relapsed during a median follow-up of 45.5 months [23]. Most reported patients treated in the chemotherapy era achieved durable remission, although evidence concerning subsequent pregnancy remains limited [3, 23].

Postpartum bleeding and beta-hCG kinetics

The differential diagnosis of postpartum vaginal bleeding with a hypervascular intrauterine lesion and elevated beta-hCG includes retained products of conception, subinvolution of the placental site, uterine arteriovenous malformation, a new pregnancy and gestational trophoblastic neoplasia. Choriocarcinoma is often hemorrhagic, necrotic and highly vascular; repeated blind curettage can increase the risk of hemorrhage or uterine perforation. In this patient, hysteroscopy and ultrasound guidance enabled tissue acquisition with minimal blood loss. The absence of chorionic villi, markedly atypical mixed trophoblastic proliferation, immunophenotype and high Ki-67 index supported choriocarcinoma [1, 2].

After evacuation, beta-hCG declined from 49,593 to 1,190.39 IU/L and then increased to 2,687 IU/L. The initial decline did not exclude choriocarcinoma; removal of part of the tumor burden may have been followed by persistent secretion from residual or metastatic disease. Final staging depends on specialist review of the original imaging, including the number, size and nature of the pulmonary and hepatic lesions.

Limitations

This report has three principal limitations. First, no trophoblastic tumor was found in the retained placental blocks and the remaining placenta was unavailable for additional sampling, so the proposed placental origin of the fetomaternal hemorrhage remains a clinicopathological inference. Second, the available cardiotocography image did not permit independent confirmation of every temporal criterion for a true sinusoidal pattern; it is therefore described as a suspected sinusoidal pattern. Third, the

available records ended after referral and did not include definitive maternal stage, chemotherapy or long-term maternal and neonatal outcomes.

Conclusions

Gestational choriocarcinoma associated with fetomaternal hemorrhage is exceptionally rare. In this case, severe fetal anemia was the first clinical manifestation, and integration of reduced fetal movement, abnormal cardiotocography and MCA-PSV elevation prompted emergency delivery and successful neonatal rescue. Unexplained fetomaternal hemorrhage should prompt consideration of an occult placental trophoblastic lesion, maternal postpartum beta-hCG testing, targeted extensive placental sampling and coordinated neonatal surveillance. Negative routine placental histology should not end the diagnostic pathway when postpartum bleeding or abnormal beta-hCG kinetics subsequently emerge.

Abbreviations

beta-hCG
beta-human chorionic gonadotropin
FMH
fetomaternal hemorrhage
hCG
human chorionic gonadotropin
hPL
human placental lactogen
MCA-PSV
fetal middle cerebral artery peak systolic velocity
MoM
multiples of the median

Declarations

Ethics approval and consent to participate

Not applicable. This article reports a single clinical case and does not constitute a research study.

Consent for publication

Written informed consent was obtained from the patient for publication of her clinical details and for publication of the neonatal clinical details and images as the infant's legal guardian. A copy of the signed consent form is available for review by the Editor-in-Chief on request.

Competing interests

The authors declare that they have no competing interests.

Generative artificial intelligence statement

The authors used OpenAI Codex to assist with English-language editing, manuscript organization and formatting. The authors independently reviewed and verified all clinical facts, medical interpretations and references and take full responsibility for the final content.

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Author Contribution

"S.Q. and Y.S. wrote the main manuscript text. S.Q. and Z.G. prepared figures and clinical data. All authors reviewed the manuscript."

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Data Availability

All data supporting the conclusions of this article are included within the article. Additional de-identified clinical information may be available from the corresponding author on reasonable request, subject to patient privacy and institutional requirements.

References

1. Ngan HYS, Seckl MJ, Berkowitz RS, et al. Diagnosis and management of gestational trophoblastic disease: 2025 update. Int J Gynaecol Obstet. 2025;171(Suppl 1):78–86. 10.1002/ijgo.70275.
2. Lok C, van Trommel N, Braicu EI, et al. Practical guidelines for the treatment of gestational trophoblastic disease: collaboration of the EOTTD-ESGO-GCIG-ISSTD. J Clin Oncol. 2025;43(18):2119–28. 10.1200/JCO-24-02326.
3. Jiao L, Ghorani E, Sebire NJ, Seckl MJ. Intraplacental choriocarcinoma: systematic review and management guidance. Gynecol Oncol. 2016;141(3):624–31. 10.1016/j.ygyno.2016.03.026.
4. Stabile G, Gentile RM, Carlucci S, et al. Maternal and fetal outcomes of intraplacental choriocarcinoma complicated by fetomaternal hemorrhage: a systematic review. J Matern Fetal

Neonatal Med. 2023;36(2):2285238. 10.1080/14767058.2023.2285238.

5. Mari G, Deter RL, Carpenter RL, et al. Noninvasive diagnosis by Doppler ultrasonography of fetal anemia due to maternal red-cell alloimmunization. N Engl J Med. 2000;342(1):9–14. 10.1056/NEJM200001063420102.
6. Martinez-Portilla RJ, Lopez-Felix J, Hawkins-Villarreal A, et al. Performance of fetal middle cerebral artery peak systolic velocity for prediction of anemia in untransfused and transfused fetuses: systematic review and meta-analysis. Ultrasound Obstet Gynecol. 2019;54(6):722–31. 10.1002/uog.20273.
7. Macones GA, Hankins GDV, Spong CY, Hauth J, Moore T. The 2008 National Institute of Child Health and Human Development workshop report on electronic fetal monitoring: update on definitions, interpretation, and research guidelines. J Obstet Gynecol Neonatal Nurs. 2008;37(5):510–5. 10.1111/j.1552-6909.2008.00284.x.
8. Wylie BJ, D'Alton ME. Fetomaternal hemorrhage. Obstet Gynecol. 2010;115(5):1039–51. 10.1097/AOG.0b013e3181da7929.
9. Treisser A, Vige P, Maria B, Lebrun F, Sureau C. Sinusoidal fetal heart rate pattern in severe fetal anemia from feto-maternal transfusion. Int J Gynaecol Obstet. 1982;20(3):211–7. 10.1016/0020-7292(82)90074-1.
10. Sankaran D, Giusto E, Lim MJ, Valdez R, Lakshminrusimha S. Fetomaternal hemorrhage: pathogenesis, diagnosis, and clinical management. J Perinatol. 2026. 10.1038/s41372-025-02548-y.
11. Wang L, Li X, Bai D, et al. Intraplacental choriocarcinoma and spontaneous fetomaternal hemorrhage: uncovering diagnostic clues in a challenging case. Front Oncol. 2025;15:1600200. 10.3389/fonc.2025.1600200.
12. Peng HH, Ng ZP, Tang YH, Chua AAA, Huang KG. Term pregnancy with choriocarcinoma presenting as severe fetal anemia and postpartum hemorrhage. Taiwan J Obstet Gynecol. 2016;55(3):430–3. 10.1016/j.tjog.2016.04.021.
13. Hookins B, Vatsayan A. Intraplacental choriocarcinoma and fetomaternal haemorrhage and maternal disseminated intravascular coagulopathy in a term pregnancy: a case report. Case Rep Womens Health. 2020;27:e00216. 10.1016/j.crwh.2020.e00216.
14. Monteiro S, Burling M, Doyle H. Late diagnosis of intraplacental choriocarcinoma co-existing with fetomaternal haemorrhage causing fetal demise: a case report. Case Rep Womens Health. 2021;31:e00341. 10.1016/j.crwh.2021.e00341.
15. Farmer M. Fetomaternal hemorrhage and choriocarcinoma: a case report. Adv Neonatal Care. 2024;24(5):417–23. 10.1097/ANC.0000000000001192.
16. Jin Y, Zhang S, Wang X, Wei S. Intraplacental choriocarcinoma in a term pregnancy with massive fetomaternal hemorrhage: a case report. BMC Pregnancy Childbirth. 2025;25(1):975. 10.1186/s12884-025-08104-y.
17. Tsuge S, Yoshida K, Shirasaki M, et al. Intraplacental choriocarcinoma presenting with postpartum hemorrhage requiring uterine artery embolization: a case report. J Obstet Gynaecol Res.

2026;52(8):e70463. 10.1111/jog.70463.

18. Simoes M, Vale G, Lacerda C, Pais P, Pignatelli D. Fetomaternal hemorrhage: a clue to intraplacental choriocarcinoma and neonatal malignancy. J Matern Fetal Neonatal Med. 2022;35(25):6615–7. 10.1080/14767058.2021.1918665.
19. She Q, Cheng Z, El-Chaar D, Luo F, Guo X, Wen SW. Intraplacental choriocarcinoma coexisting with fetomaternal hemorrhage: case report, chemotherapy management, and literature review. Med (Baltim). 2018;97(14):e9977. 10.1097/MD.0000000000009977.
20. Fisher RA, Savage PM, MacDermott C, et al. The impact of molecular genetic diagnosis on the management of women with hCG-producing malignancies. Gynecol Oncol. 2007;107(3):413–9. 10.1016/j.ygyno.2007.07.081.
21. Savage P, Monk D, Hernandez Mora JR, et al. A case of intraplacental gestational choriocarcinoma characterised by the methylation pattern of the early placenta and an absence of driver mutations. BMC Cancer. 2019;19:744. 10.1186/s12885-019-5906-8.
22. Mangla M, Rahiman EA, Kaur H, Kanikaram P. Gestational trophoblastic neoplasia with concurrent metastasis to the mother and child: a systematic literature review. J Turk Ger Gynecol Assoc. 2023;24(3):206–19. 10.4274/jtgga.galenos.2023.2023-5-2.
23. Liu Y, Song X, Zhang H, et al. Long-term outcome and fertility results of intraplacental choriocarcinoma: a retrospective study of 14 patients and literature review. Orphanet J Rare Dis. 2024;19:214. 10.1186/s13023-024-03199-6.

Figures

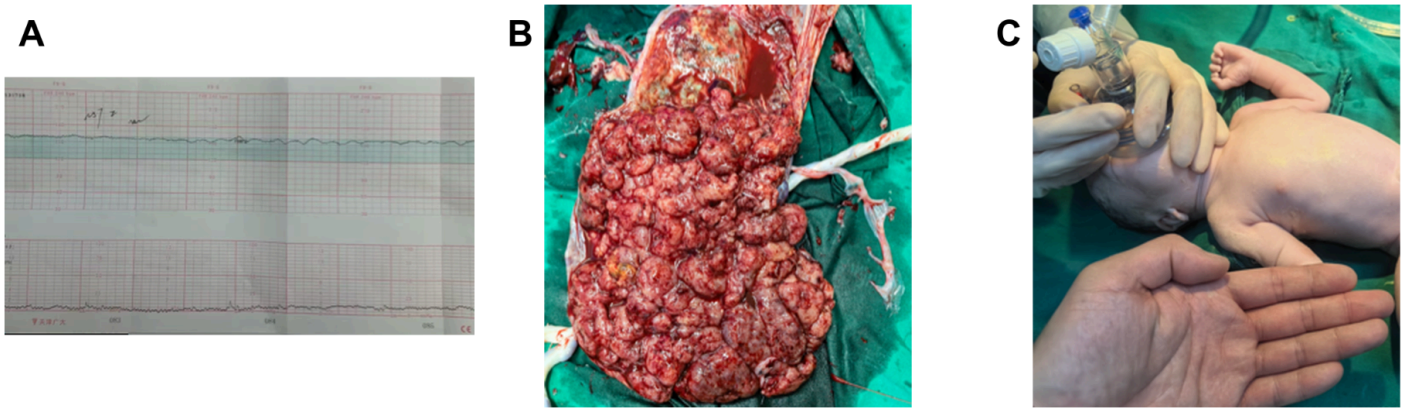


Figure 1

Perinatal clinical findings

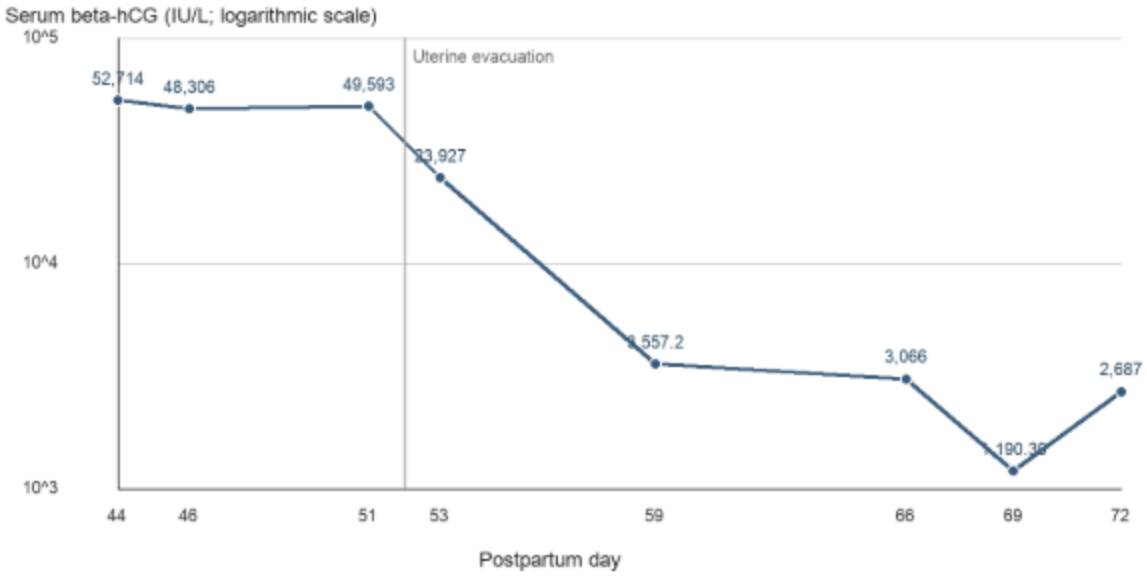


Figure 2

Postpartum serum beta-hCG kinetics