




Recurrent Post-Abortion Gestational Trophoblastic Neoplasia Successfully Treated with TP/TE: A Case Report

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Introduction: Gestational trophoblastic neoplasia (GTN) is a highly curable malignancy, but cases arising after abortion are uncommon and often diagnosed late. We report a recurrent high-risk GTN following abortion that relapsed multiple times despite etoposide–methotrexate–actinomycin D/cyclophosphamide–vincristine (EMA/CO).

Case Report: A 26-year-old woman achieved initial remission with six cycles of EMA/CO but experienced several recurrences from 2021 to 2023. She presented again with rising β -hCG (16,925.8 mIU/mL) and an intrauterine mass. Paclitaxel/cisplatin alternating with paclitaxel/etoposide (TP/TE) was given for six cycles plus two consolidation cycles, resulting in complete remission. β -hCG normalized by January 2024 and remained undetectable for 12 months.

Conclusion: This case highlights diagnostic challenges of post-abortion GTN and shows that TP/TE is an effective and well-tolerated salvage option for recurrent or EMA/CO-resistant GTN. Long-term β -hCG surveillance is essential to ensure durable remission and assess fertility outcomes.

Keywords: gestational trophoblastic neoplasia, abortion, β -hCG, recurrence, salvage chemotherapy

Introduction

Gestational trophoblastic neoplasia (GTN) represents a malignant continuum of trophoblastic diseases, including invasive mole, choriocarcinoma, placental site trophoblastic tumor (PSTT), and epithelioid trophoblastic tumor (ETT).^{1,2} It is highly responsive to chemotherapy, with survival exceeding 90% even in high-risk disease.³ Serum β -hCG is the most reliable biomarker for diagnosis, monitoring treatment response, and detecting persistence or recurrence, and rising or plateauing levels after uterine evacuation or chemotherapy define treatment failure.^{4–6}

Although approximately half of GTN cases arise after molar pregnancy, about 25% develop following nonmolar gestations such as miscarriage or abortion.⁷ Women with a history of spontaneous abortion have a two- to threefold increased risk of subsequent trophoblastic disease.^{7,8} However, GTN following abortion remains uncommon, with an estimated incidence of 2–200 per 100,000 pregnancies, and often presents with nonspecific symptoms such as persistent bleeding, contributing to diagnostic delay.^{7,9} As a result, patients may present with higher-risk disease or face an increased likelihood of recurrence after treatment.

For high-risk GTN (FIGO score ≥ 7), etoposide–methotrexate–actinomycin D/cyclophosphamide–vincristine (EMA/CO) remains the standard first-line regimen, achieving survival rates of approximately 90%.^{6,10} Around 10% of patients, however, develop drug resistance or experience relapse and require salvage therapy.^{10,11} Etoposide–cisplatin alternating with EMA (EP/EMA) is an established salvage option but is associated with substantial toxicity, including high rates of neutropenia, thrombocytopenia, anemia, and renal impairment.^{10–12} Because of these limitations, paclitaxel-based regimens have gained interest. Emerging evidence from small case series and early clinical experiences suggests that

paclitaxel/cisplatin alternating with paclitaxel/etoposide (TP/TE) demonstrates meaningful activity and improved tolerability in relapsed or refractory GTN.^{12–14}

This case report describes a rare post-abortion GTN with multiple relapses following EMA/CO, ultimately achieving sustained remission with TP/TE. It underscores the diagnostic challenges of GTN after nonmolar pregnancy, the need for vigilant β -hCG surveillance even after remission, and the potential role of TP/TE as an effective and better-tolerated salvage option for recurrent high-risk GTN.

Case Report

A 26-year-old woman was diagnosed with recurrent gestational trophoblastic neoplasia (GTN) following abortion. Her clinical course began in June 2020 with an abortion managed by uterine evacuation, with histopathology confirming retained products of conception. Despite curettage, she continued to experience vaginal bleeding with a persistently positive urine pregnancy test (qualitative β -hCG). In September 2020, serum β -hCG was 73,870 mIU/mL and rose to >1,000,000 mIU/mL in October, the following month, leading to referral to a tertiary center.

On assessment in November 2020, she complained vaginal bleeding without abdominal pain. Physical examination revealed a uterine fundal height midway between the symphysis and umbilicus. Transvaginal ultrasound demonstrated a uterus measuring 8.50×4.02 × 6.62 cm with two intrauterine masses infiltrating the myometrium (1.27 × 2.35 cm and 2.26×2.17 cm, Doppler +4). She was diagnosed with stage I GTN, FIGO score 8 (high risk), and received EMA/CO chemotherapy (4 cycles plus 2 consolidation cycles, December 2020–May 2021), achieving β -hCG <5 mIU/mL.

During surveillance, β -hCG rose again to 40.7 mIU/mL in December 2021, then she received another 2 cycles of EMA/CO plus 1 consolidation cycle (January–June 2022). By June 2022, β -hCG had declined to 1.75 mIU/mL. In July 2022, β -hCG increased again to 128.7 mIU/mL, and she was given 3 EMA/CO cycles plus 2 consolidation cycles, achieving remission (<5 mIU/mL, December 2022). However, in January 2023, levels rose again to 7 mIU/mL. Chest X-ray, head CT scan, and repeat β -hCG testing were performed, but the patient was lost to follow-up until August 2023.

When she returned in August 2023, she presented with vaginal spotting and provided imaging results from August 2023 showing a normal chest X-ray, a head CT scan without evidence of intracranial metastasis, neoplasm, or vascular malformation. The level β -hCG had increased to 16,925.8 mIU/mL in August 2023. Repeat evaluation revealed an abdominal ultrasound showing a mixed-echogenic uterine mass without calcification, suggestive of recurrent gestational trophoblastic neoplasia, with no evidence of metastasis (Figure 1A). The patient was then scheduled to receive TP/TE chemotherapy (Paclitaxel 135 mg/m² / Cisplatin 60 mg/m² alternating with Paclitaxel 135 mg/m² / Etoposide 150 mg/m²). The timeline of the disease is illustrated in Figure 1B.

The patient received six cycles of TP/TE followed by two consolidation cycles, resulting in a decline of β -hCG from 75,367.6 mIU/mL (September 2023) to 2.8 mIU/mL (January 2024). Twelve months of surveillance showed persistently normal β -hCG. At the latest follow-up, the patient had regular menstrual cycles and normal ovarian function. No markers of diminished ovarian reserve were noted; however, anti-Müllerian hormone (AMH) levels were not assessed due to limited availability at the time. Timeline of the patient's clinical course, including initial presentation, serial β -hCG trends, periods of loss to follow-up, and chemotherapeutic regimens administered from 2020 to 2024 was shown in Figure 2.

Discussion

GTN is a pregnancy-associated malignancy characterized by placental origin and the presence of paternal genetic material, conferring unique biologic behavior and marked chemosensitivity. However, GTN is able to evade both maternal and placental immune surveillance, permitting unchecked proliferation, invasion, and metastatic spread. These immune escape mechanisms are thought to underlie the persistence and progression of GTN.¹⁵

The pathophysiology is closely related to alterations within the placental villi. The villous structure is composed of distinct cellular compartments: trophoblasts (including cytotrophoblasts and syncytiotrophoblasts), mesenchymal elements with Hofbauer cells, and extravillous trophoblasts (EVTs). Cytotrophoblasts generate syncytiotrophoblasts, which produce β -hCG and interact directly with maternal blood. Hofbauer cells, in turn, contribute to vasculogenesis through VEGF secretion, while also mediating maternal-fetal immune interactions.^{16,17} EVT are of particular importance, as they share invasive, tumor-like behavior and are capable of infiltrating the decidua and maternal circulation.¹⁸ Normally, this

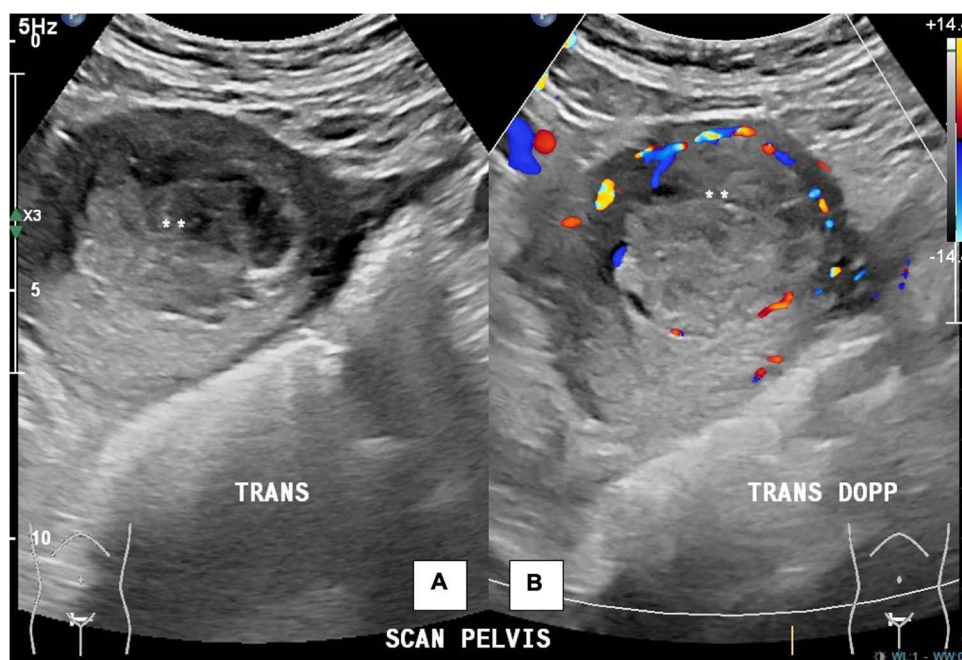


Figure 1 (A) Transverse pelvic ultrasonography showing a heterogeneous intrauterine mass with cystic spaces, measuring approximately 7.04×5.23 cm. (B) Sagittal Doppler pelvic ultrasonography showing a hypervascular intrauterine mass with prominent peripheral and intralesional blood flow signals.

invasion is tightly regulated by a balance of stimulatory and inhibitory signals in the myometrium. In GTN, disruption of this equilibrium drives uncontrolled trophoblast proliferation, invasion, and dissemination.^{16,19}

GTN most commonly develops following a hydatidiform mole; however, this case illustrates the occurrence of GTN after an abortion. Persistent vaginal bleeding after curettage should always prompt careful evaluation for the possibility of GTN. In this case, the patient underwent curettage for abortion but continued to experience abnormal vaginal bleeding, leading the physician to perform a quantitative β -hCG test. The markedly elevated β -hCG level raised suspicion of GTN, and the patient was subsequently referred to a tertiary care center for further management. She was diagnosed with GTN with a FIGO score of 8, categorized as high risk, and was therefore treated with EMA/CO combination chemotherapy. Table 1 summarizes reported cases of GTN following abortion.

Recurrent disease was evaluated using serial quantitative serum β -hCG measurements, clinical assessment, and imaging studies, including transvaginal and abdominal ultrasonography, chest radiography, and head CT when indicated. Importantly, alternative causes of β -hCG elevation—namely phantom hCG, quiescent GTN, and pituitary hCG—were considered during the evaluation of recurrence.

The marked rise in β -hCG to 16,925.8 mIU/mL occurring 7–8 months after completion of chemotherapy is inconsistent with phantom hCG, quiescent GTN, or pituitary hCG. Phantom hCG is characterized by low, non-progressive serum hCG levels without clinical or radiologic correlation and does not respond to chemotherapy. Quiescent GTN typically presents with persistently low and stable β -hCG levels (<200 mIU/mL), absence of symptoms, and no radiologic evidence of disease. Pituitary hCG occurs at very low levels in peri- or postmenopausal women. In contrast, this patient demonstrated progressive β -hCG elevation, clinical symptoms, radiologic evidence of uterine disease, and a prompt biochemical response to chemotherapy, confirming active recurrent GTN.

The treatment for GTN is chemotherapy, with the regimen selected based on stage and prognostic scoring. According to the 2000 FIGO system, patients with a score ≤ 6 are classified as low risk, while those with a score ≥ 7 are considered high risk. High-risk GTN, as in the present case, is primarily managed with combination chemotherapy such as EMA/CO, which is continued until β -hCG normalization. The EMA/CO regimen consists of etoposide, actinomycin D, and methotrexate on days 1–2, followed by cyclophosphamide and vincristine on day 8. An alternative protocol is EMA/EP, in which etoposide and cisplatin are administered on day 8 in place of cyclophosphamide and vincristine.²⁵ According to

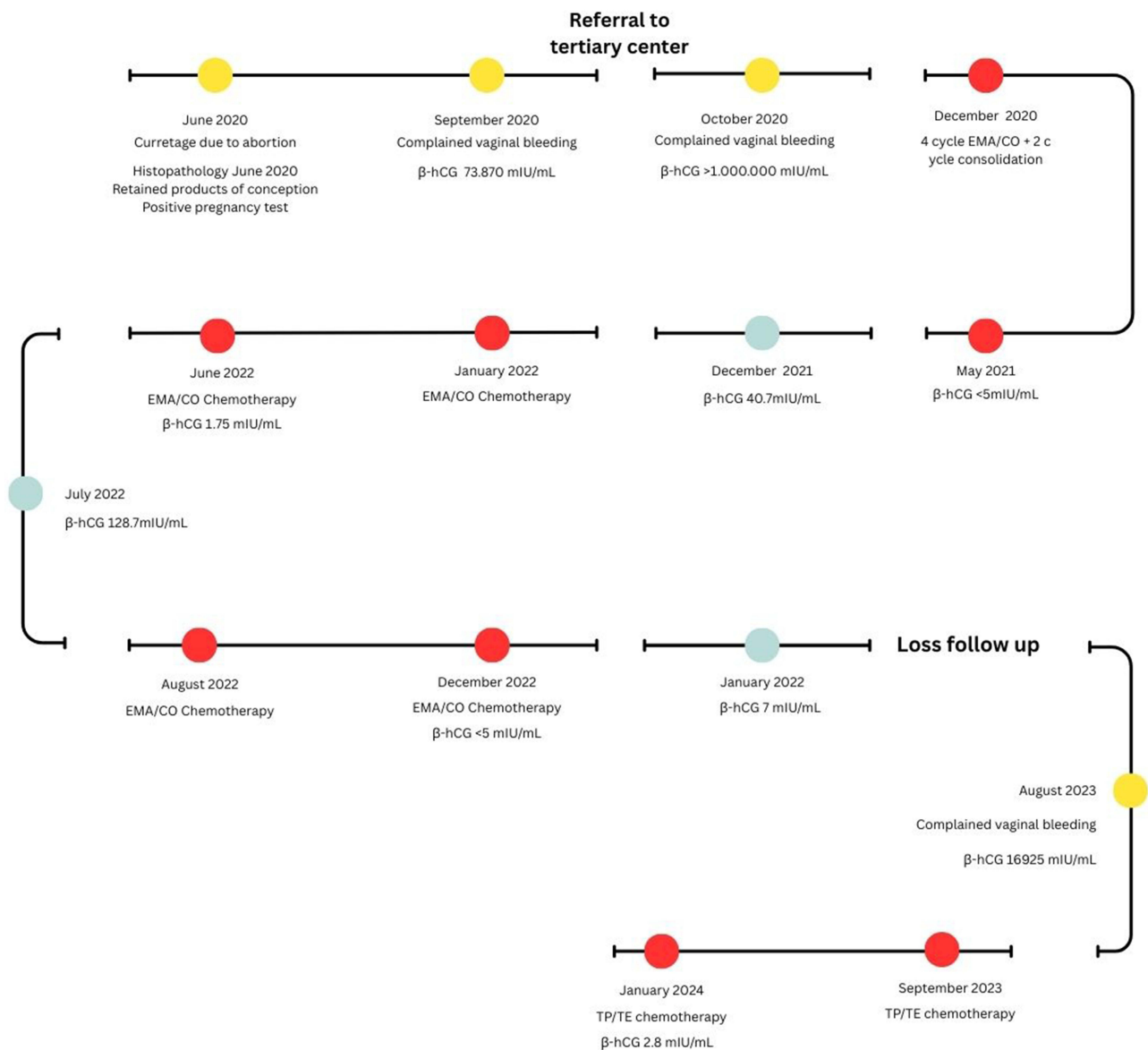


Figure 2 Timeline of the patient's clinical course, including initial presentation, serial β -hCG trends, periods of loss to follow-up, and chemotherapeutic regimens administered from 2020 to 2024. Yellow markers indicate clinical presentations or diagnostic events; red markers represent chemotherapy treatments; blue markers represent follow-up evaluations and β -hCG monitoring.

the NCCN Clinical Practice Guidelines, once β -hCG levels have normalized, treatment should be continued with consolidation therapy. This consists of 2 cycles of single-agent chemotherapy for low-risk GTN, and 2–3 additional cycles of multi-agent chemotherapy, such as EMA/CO or EMA/EP, for high-risk disease.²⁶

Although GTN is highly chemosensitive, approximately 25% of patients with high-risk disease may experience persistence or recurrence following first-line therapy, or relapse after initial remission, resulting in residual disease. This phenomenon is thought to be related to an imbalance between stimulatory and inhibitory mechanisms during the cellular phase, even after treatment. GTN employs several immune evasion strategies, such as: (1) imitating immune tolerance mechanisms by lacking HLA class II expression, thereby escaping T-cell recognition and cytolysis; (2) resisting natural killer (NK) cell-mediated cytotoxicity; (3) downregulating conventional activation markers—including CD69 (NK cells), CD71 (macrophages), CD134 (T cells), CD3/HLA-DR (T cells)—as well as the apoptosis marker CD95 (Fas) on lymphocytes; and (4) overexpressing hCG receptors and increasing hCG secretion, which enhances local

Table 1 Summarizes of Reported Cases of GTN Following Abortion

Author, Year	Age (Years)	Diagnosis After Abortion	Type of GTN	Therapy Given
Thomakos, 2010 ²⁰	39	Abortion	<ul style="list-style-type: none"> Repeat D&C (evacuation of complete hydatidiform mole) Combination chemotherapy with BEP for 4 cycles Exploratory laparotomy with: <ul style="list-style-type: none"> Splenectomy Retroperitoneal tamponade due to massive hemorrhage 	Did not survive
Zhang, 2014 ²¹	44	Induced Abortion	<ul style="list-style-type: none"> Exploratory laparotomy with excision of tumor on the left broad ligament Hysterectomy Combination chemotherapy with EMA-CO regimen for 6 cycles 	Survived
Neupane, 2016 ²²	17	Incomplete Abortion	<ul style="list-style-type: none"> Misoprostol per vaginal and MVA for incomplete abortion Oral antibiotics (Doxycycline) post-MVA Laparotomy to repair uterine perforation Single-agent chemotherapy with MTX and Leucovorin rescue for invasive mole 	Survived
Gülhan, 2017 ²³	39	Missed Abortion	<ul style="list-style-type: none"> Single-agent chemotherapy with MTX and folinic acid (Leucovorin) rescue according to departmental protocol: <ul style="list-style-type: none"> MTX 50 mg IM every 48 hours × 4 doses Folinic acid 15 mg orally 30 hours after each MTX injection Repeated every 15 days until β-hCG level normalized Two additional cycles given as consolidation (total 6 cycles) Oral contraceptives administered during treatment 	Survived
Jaiswar, 2022 ²⁴	26	Incomplete Abortion	<ul style="list-style-type: none"> Single-agent chemotherapy with MTX 1 mg/kg on days 1, 3, 5, and 7 Folinic acid rescue 15 mg orally on days 2, 4, 6, and 8 Cycles repeated every 14 days until β-hCG level normalized After β-hCG normalization, 2 consolidation cycles were administered MTX dose during consolidation was reduced by 25% due to hepatotoxicity 	Survived

immunosuppression at the uterine site and prevents malignant trophoblast detection by the maternal immune system. Several clinical risk factors contribute to this phenomenon.

Higher total FIGO scores are strongly associated with chemotherapy resistance, largely reflecting a greater tumor burden (eg, markedly elevated β -hCG levels, larger tumor size, multiple metastases) rather than independent prognostic factors. Inadequate primary therapy and poor treatment adherence also play critical roles in resistance development.^{27,28} Additionally, Doppler ultrasound studies have suggested that a low uterine artery pulsatility index before chemotherapy indicates increased tumor vascularization and a higher risk of methotrexate resistance.²⁸ In the present case, the patient's markedly high FIGO score, extremely elevated pretreatment β -hCG concentration (>1,000,000 mIU/mL), and treatment interruption contributed to the risk of relapse, necessitating salvage chemotherapy.²⁷

Salvage chemotherapy options for recurrent GTN include Paclitaxel/Cisplatin–Paclitaxel/Etoposide (TP/TE), a regimen initially developed for germ cell tumors. More recently, case reports have described its benefit in two untreated GTN patients. In a prior cohort of 24 patients receiving TP/TE, 16 had refractory or relapsed disease after earlier therapy. Within this subgroup, survival was relatively low (45%), reflecting the fact that most had undergone two or more previous regimens, including six patients who had failed platinum-based treatment (five after EP/EMA and one after BEP). Conversely, survival outcomes were more favorable (75%) in eight patients who were switched to TP/TE because of toxicity from their prior regimens, including six previously treated with EP/EMA. Notably, compared with EMA/CO, TP/TE was associated with a more tolerable toxicity profile, despite its use in patients who had generally received more intensive prior therapy. Severe neutropenia (grade 3–4) occurred in 42% and thrombocytopenia in 13% of TP/TE patients, compared with 68% and 40% among those receiving EP/EMA. Importantly, no TP/TE patients

developed neutropenic sepsis or required treatment delays, whereas 88% of EP/EMA patients experienced delays, partly due to neutropenic sepsis.^{12,14}

Nonetheless, two key uncertainties remain regarding TP/TE use: first, the long-term survival outcomes, given the relatively short follow-up in available series; and second, its potential impact on fertility. While animal studies and limited case reports indicate that pregnancy is possible after paclitaxel exposure, further data are required.^{12,14,29} Consequently, in cases of recurrent high-risk GTN, combination regimens such as TP/TE or EP/EMA may be considered.

This patient initially presented with FIGO stage I disease but was categorized as high risk, with a FIGO score of 8. She was started on multi-agent chemotherapy with the EMA/CO regimen. Despite this, recurrence occurred, and salvage therapy with TP/TE was initiated in September 2023. The patient had complete response. Overall survival following chemotherapy in GTN is excellent, approaching 98%, but outcomes vary according to the risk of drug resistance determined by the FIGO score. Low-risk patients (scores 0–6) are treated with methotrexate and folinic acid, achieving nearly 100% survival, although up to one-third require second-line treatment with either intravenous actinomycin D alone or combination regimens such as EMA/CO.⁸ In contrast, patients with high-risk disease (score ≥ 7) are treated with EMA/CO as first-line therapy, with survival rates of approximately 90%.^{12,14,29,30} Nevertheless, more than 10% of these patients experience treatment failure. For women who relapse after EMA/CO, prior studies suggest that cisplatin can be incorporated with etoposide (EP) alternating weekly with EMA, sometimes alongside surgery, resulting in salvage rates of around 75%. However, this regimen is associated with substantial toxicity, as 40% of patients experience grade 3–4 adverse effects, including neutropenia (68%), thrombocytopenia (40%), anemia (21%), renal impairment (41%), and other complications. Consequently, there remains a need for salvage regimens with greater efficacy and a more favorable toxicity profile.^{12,14,29,30}

If patients fail to respond to TP/TE, several studies have reported alternative salvage options, including gemcitabine-based regimens and high-dose chemotherapy (HDC) with stem cell rescue, which may be combined with surgical procedures such as hysterectomy. Patients who achieve a response to TP/TE generally show better outcomes with subsequent therapies.^{8,11,14} Because these patients are classified as high risk, the standard first-line treatment is EMA/CO. Serial quantitative β -hCG monitoring plays a central role in identifying drug resistance, recurrence, and therapeutic response.

All GTN patients require quantitative serum β -hCG surveillance for 12 months following treatment. The recommended schedule is every two weeks during the first three months, monthly for the next three months, and every two months over the final six months. In addition, chest radiography is suggested at 6 and 12 months if respiratory symptoms are present.²⁵ In patients with stage IV disease, follow-up is extended to 24 months due to the higher risk of late recurrence.

Monitoring should continue until β -hCG levels remain undetectable for 12 consecutive months, during that, time effective contraception must be maintained. Patients should also be assessed for complications such as hemorrhage or metastatic disease involving the pelvis, abdomen, liver, or brain. Imaging modalities including pelvic and abdominal ultrasound, chest radiography, CT, or MRI may be employed as clinically indicated. During the surveillance phase, pregnancy is contraindicated, and only barrier methods or the calendar method are advised, whereas intrauterine devices and injectable contraceptives should be avoided.²⁵

The incidence of secondary infertility following chemotherapy for GTN is relatively low, around 7%, indicating that most women who wish to conceive are able to achieve pregnancy.^{31,32} A large multicenter study involving 2,657 women demonstrated reassuring reproductive outcomes: the majority achieved live births, with rates of miscarriage, stillbirth, and congenital malformations comparable to the general population. Importantly, no significant differences were observed between single-agent and multi-agent regimens in terms of fertility or pregnancy outcomes. Current recommendations advise a 12-month follow-up period before attempting conception in stage I–III disease and 24 months in stage IV, to allow accurate β -hCG monitoring and reduce risks of adverse pregnancy outcomes. Pregnancies occurring within 6 months of treatment completion carry a higher risk of miscarriage, stillbirth, or recurrent molar pregnancy.^{31,32} In the present case, the patient achieved normal β -hCG levels during 12 months of surveillance post-chemotherapy; however, she has not yet conceived.

This case highlights several clinically important points. First, GTN can arise after abortion and should be considered in patients with persistent vaginal bleeding and elevated β -hCG following nonmolar pregnancy. Second, recurrent high-risk GTN may require alternative salvage chemotherapy strategies, particularly after failure of standard EMA/CO therapy. In this

context, TP/TE demonstrated effective disease control with acceptable tolerability. Third, this case underscores the central role of serial quantitative β -hCG monitoring in detecting recurrence early and guiding timely therapeutic interventions.

This report has several limitations. First, as a single case report, the findings may not be generalizable to all patients with recurrent GTN. Second, the follow-up duration is insufficient to fully evaluate long-term survival and fertility outcomes following TP/TE therapy. Third, limited molecular profiling and imaging data precluded deeper exploration of the biological mechanisms underlying chemotherapy resistance and disease recurrence. Larger studies with longer follow-up are needed to better define the role of TP/TE in recurrent high-risk GTN.

Implications

This case highlights several important clinical implications. First, gestational trophoblastic neoplasia can occur after spontaneous abortion, emphasizing the need for early recognition and routine serial quantitative β -hCG monitoring to detect recurrence promptly. Second, in patients with recurrent high-risk GTN after standard EMA/CO therapy, TP/TE may serve as an effective and better-tolerated salvage regimen, supporting its consideration in treatment algorithms. Finally, long-term surveillance remains essential to ensure durable remission and to evaluate reproductive outcomes in women of reproductive age. This case supports vigilant post-abortion follow-up and individualized escalation of therapy in recurrent GTN.

Conclusion

Spontaneous abortion may be followed by gestational trophoblastic neoplasia, underscoring the importance of serial β -hCG monitoring. In recurrent disease, timely modification or escalation of chemotherapy, including the use of alternative salvage regimens such as TP/TE, is crucial to achieve sustained remission.

Registration of Research Studies

Registration of research is not applicable in our case.

Abbreviations

BEP, Bleomycin–Etoposide–Cisplatin; β -hCG, Beta–human Chorionic Gonadotropin; D&C, Dilation and Curettage; EMA–CO, Etoposide–Methotrexate–Actinomycin D–Cyclophosphamide–Vincristine; IM, Intramuscular; MVA, Manual Vacuum Aspiration; MTX, Methotrexate; OCP, Oral Contraceptive Pills.

Ethical Approval

This study adhered to the principles of the Declaration of Helsinki and institutional guidelines for case reports. Written informed consent was obtained from the patient for publication of anonymized clinical details and accompanying images.

Informed Consent Patient Statement

The authors confirm that written informed consent has been obtained from the involved patient. The patient has been informed about the details of the case and has provided approval for the information to be published in this case report.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare that they have no competing interests in this work.

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