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# Craniotomy for Hemorrhagic Cerebral Metastatic Choriocarcinoma: A Case Report and Literature Review

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## Abstract

Gestational trophoblastic neoplasia is a rare aggressive malignancy arising from abnormal placental trophoblastic proliferation. Central nervous system involvement is uncommon, and brain metastases typically present as mass phenomena, also with a high probability of presenting symptoms of intracranial hemorrhage or subdural hematoma. Risk stratification according to the FIGO scoring system guides treatment selection between single-agent and multi-agent chemotherapy.

A 36-year-old woman with prior hydatidiform molar pregnancy presented with focal motor seizures involving the left upper extremity, left hemiparesis and intense headache. MRI revealed an intra-axial mass in the right frontoparietal lobe, with perilesional vasogenic edema, mass effect, and midline shift. Intracranial disease progression occurred despite ongoing systemic chemotherapy and declining serum β-hCG levels. During hospitalization, the patient developed a tonic-clonic seizure lasting three minutes, followed by a postictal state with a Glasgow Coma Scale score of 8, prompting urgent neurosurgical intervention. After treatment, the patient remained seizure-free, demonstrated progressive neurological recovery, and continued outpatient chemotherapy.

This case highlights the aggressive neurovascular behavior of metastatic choriocarcinoma and the potential for rapid intracranial progression despite systemic therapy. Early neuroimaging surveillance and timely neurosurgical intervention may be critical in selected high-risk patients.

**Categories:** Pathology, Oncology, Neurosurgery
**Keywords:** beta-human chorionic gonadotropin (β-hcg), cerebral metastasis, choriocarcinoma, craniotomy, gestational trophoblastic neoplasia, intracranial hemorrhage

## Introduction

Choriocarcinoma (CC) is a rare, highly aggressive trophoblastic malignancy arising from placental cells, accounting for less than 1% of gynecologic malignancies and occurring in approximately 1-9 per 40,000 pregnancies [1-3].

Approximately 50% of cases result from the malignant transformation of a hydatidiform mole [3]; however, smaller proportions arise following ectopic pregnancies, spontaneous abortions, or even term pregnancies [1,4]. Pathologically, the molar tissue mass is characterised by abnormal, absent embryonic development and hydropic degeneration of chorionic villi. Although hydatidiform mole can persist locally or progress to an invasive mole or gestational CC, gestational trophoblastic neoplasia (GTN) can develop after any pregnancy [5].

Early recognition of suggestive signs and symptoms, particularly in women of reproductive age with a recent history of pregnancy, is essential for prompt diagnosis. Measurement of serum beta-human chorionic gonadotropin (β-hCG), alongside imaging studies such as computed tomography (CT) or magnetic resonance imaging (MRI), allows identification of the disease and assessment of distant metastatic spread [6]. This neoplasm is characterized by rapid hematogenous dissemination, most commonly involving the lungs, followed by the vagina, pelvis, and liver [1,7]. Brain metastases occur in approximately 10% of patients with GTN and represent one of the leading causes of disease-related mortality owing to their marked propensity for intracranial hemorrhage and acute neurological deterioration, which is associated with poor prognosis [2,3,8].

Disease staging is a critical step in assessing a patient's clinical status, guiding therapeutic strategies and determining prognosis. To establish accurate risk stratification, we utilize clinical examination, imaging studies and surgical findings in accordance with the International Federation of Gynecology and Obstetrics (FIGO) staging system, which classifies disease based on primary tumor location, anatomical extension and

the presence of distant metastases [9].

Metastatic choriocarcinoma constitutes an oncologic emergency; however, favorable outcomes may be achieved with prompt multidisciplinary management based primarily on intensive chemotherapy [5].

We present a case of right frontoparietal metastasis secondary to choriocarcinoma following a molar pregnancy and rapid spread.

## Case Presentation

A 36-year-old woman with a history of hydatidiform molar pregnancy treated by uterine curettage one year earlier, without subsequent clinical follow-up because of financial constraints, presented nine months later with abnormal uterine bleeding. She was diagnosed with high-risk gestational choriocarcinoma, classified as FIGO stage III [9], with pulmonary and renal metastases. Serial serum β-hCG measurements throughout the clinical course are shown in Table *1*. Her initial serum β-hCG level was 664,362 mIU/mL, and induction chemotherapy with etoposide and cisplatin (EP regimen) was initiated. Despite an early biochemical response with serum β-hCG levels declining to 225,000 mIU/mL, after one month of treatment she developed focal motor seizures involving the left upper extremity, progressive left-sided hemiparesis, and impaired consciousness, requiring admission to an outside hospital, where repeat serum β-hCG testing revealed a rise to 368,000 mIU/mL.

| Laboratory parameter | Reference range | 10.18.24 | 12.6.25 | 01.12.26 | 03.10.26 | 04.02.26 | 05.15.26 |
|---|---|---|---|---|---|---|---|
| Serum β-hCG (mUI/mL) | <5.0* | 664,362 | 225,000 | 368,000 | 13,427 | 6,326 | 8,610 |

**TABLE 1: Serial serum β-hCG measurements throughout the patient's clinical course.**

* Reference range for non-pregnant women.

β-hCG: beta-human chorionic gonadotropin.

Three weeks later, she was transferred to our tertiary-care institution in the same city for definitive specialized treatment because of progressive headache, recurrent focal motor seizures, and persistent left-sided hemiparesis. Brain MRI and CT revealed a hemorrhagic right frontoparietal intra-axial lesion associated with extensive vasogenic edema and significant mass effect, highly suggestive of hemorrhagic cerebral metastasis (Figure *1*).

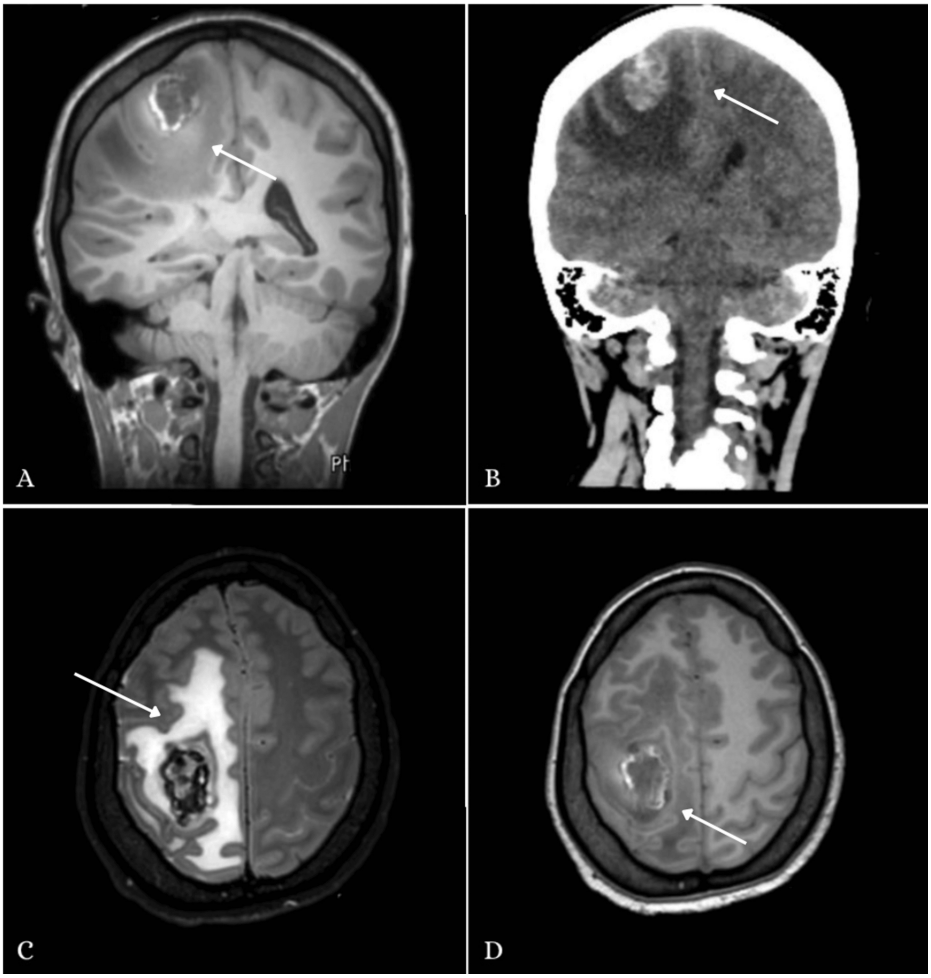



**FIGURE 1: Preoperative imaging**

(A) Coronal contrast-enhanced magnetic resonance imaging (MRI) showing a right frontoparietal intra-axial lesion with peripheral enhancement and extensive surrounding vasogenic edema. (B) Coronal computed tomography (CT) demonstrating the lesion with significant mass effect and midline shift. (C) Axial MRI further illustrates the heterogeneous lesion and associated edema. (D) Axial contrast-enhanced MRI showing ring enhancement and surrounding edema consistent with an aggressive intracranial metastatic process.

Given her progressive neurological deterioration despite systemic chemotherapy, recurrent seizures, and significant mass effect, the patient underwent an emergency right frontoparietal craniotomy with microsurgical gross-total resection of the lesion. Intraoperatively, a highly vascular hemorrhagic cortical tumor measuring approximately 2.5 × 5.0 cm with extensive necrotic areas was identified. Gross-total resection was achieved under microscopic visualization without intraoperative complications (Figure *2*).

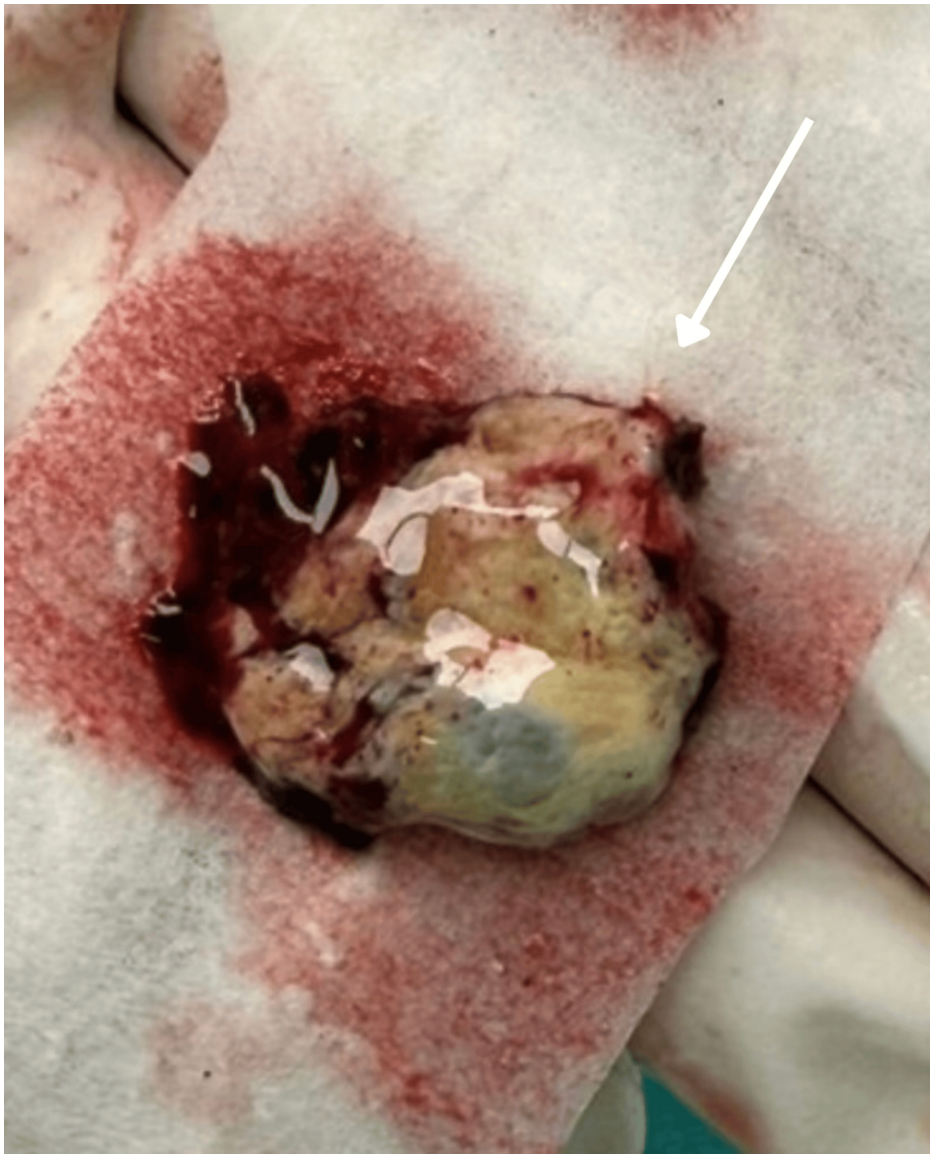


**FIGURE 2: Gross appearance of the resected cerebral metastatic lesion.**

Intraoperative specimen demonstrating a friable hemorrhagic mass with heterogeneous appearance.

Postoperative CT demonstrated gross-total resection of the lesion without evidence of residual or postoperative hemorrhage (Figure *3*).

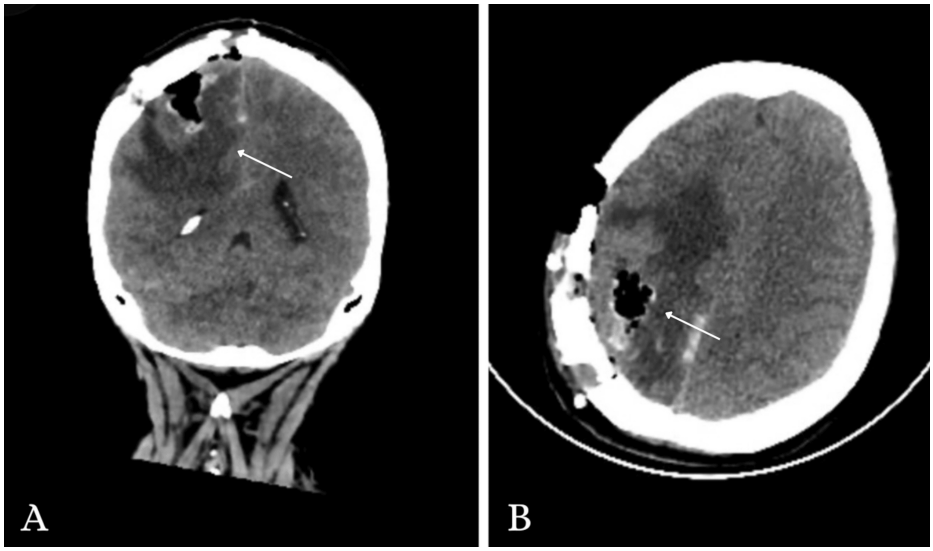



**FIGURE 3: Postoperative computed tomography following surgical resection.**

(A) Coronal CT demonstrating the postoperative resection cavity in the right frontoparietal region with reduction of mass effect. (B) Axial CT showing postoperative changes after craniotomy and tumor resection.

During the postoperative period, seizure prophylaxis was maintained with levetiracetam, and cerebral edema was treated with dexamethasone. Histopathological examination confirmed metastatic choriocarcinoma, demonstrating hemorrhagic trophoblastic proliferation with multinucleated syncytiotrophoblastic giant cells, extensive necrosis, and biphasic trophoblastic differentiation (Figure 4).

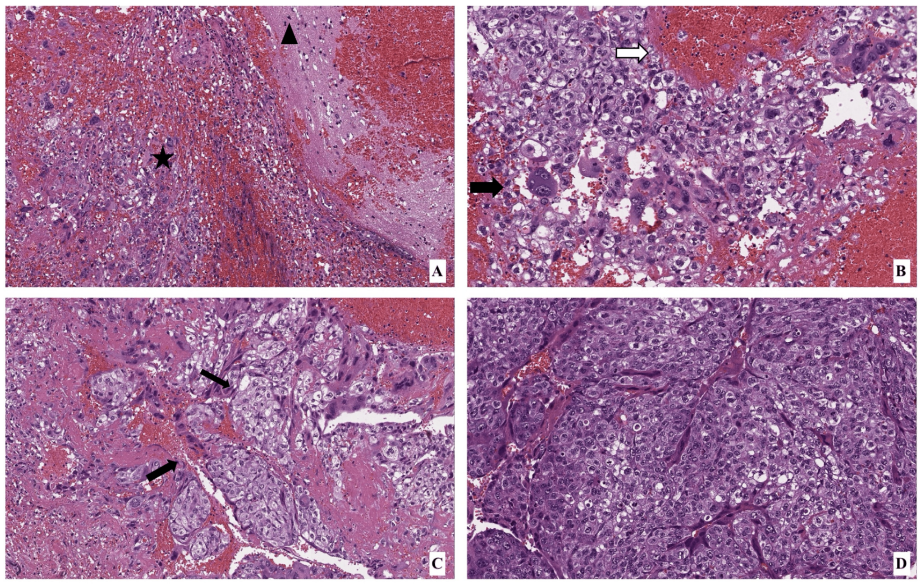



**FIGURE 4: Histopathological findings of the metastatic cerebral lesion (hematoxylin and eosin stain).**

Representative photomicrographs of the metastatic brain lesion diagnostic of choriocarcinoma, all obtained by digital slide scanning at an equivalent magnification of 20×. (A) Transition zone between preserved glial parenchyma and infiltrating hemorrhagic trophoblastic proliferation (triangle). Hemorrhagic trophoblastic proliferation (star). (B) Proliferation of pleomorphic trophoblastic cells with marked nuclear atypia and multinucleated giant cells consistent with syncytiotrophoblasts (black arrow) within a markedly hemorrhagic background (white arrow). (C) Viable tumor nests (arrows) adjacent to extensive areas of necrosis and hemorrhage. (D) Characteristic biphasic pattern composed of mononuclear cytotrophoblastic cells and multinucleated syncytiotrophoblastic cells arranged in solid aggregates, diagnostic of metastatic choriocarcinoma.

The patient remained in the neurocritical care unit with a favorable neurological course, although expressive aphasia and persistent left-sided hemiparesis were noted and no recurrent seizures were documented while receiving levetiracetam prophylaxis.

On postoperative day 14, she was discharged from the Neurosurgery service for continuation of oncologic management, where neurological examination demonstrated bilaterally equal 3-mm pupils reactive to light, no cranial nerve deficits, persistent left-sided hemiparesis (MRC grade 2/5) [10], generalized hyperreflexia and bilateral extensor plantar responses.

On the day following transfer from the Neurosurgery service, expressive aphasia had completely resolved. Residual neurological deficits were limited to left-sided hemiparesis with associated hyperreflexia. Induction chemotherapy was resumed, by which time the serum β-hCG level had declined to 13,427 mIU/mL. Upon completion of the induction chemotherapy, treatment was escalated with etoposide, methotrexate, actinomycin D, etoposide and cisplatin regimen (EMA-EP) according to the institutional protocol. A comprehensive timeline of the patient's clinical course, neurosurgical intervention, serial serum β-hCG measurements and oncologic treatment is presented in Figure *5*. At the most recent follow-up, she remained seizure-free, demonstrated progressive neurological recovery, and continued outpatient chemotherapy.

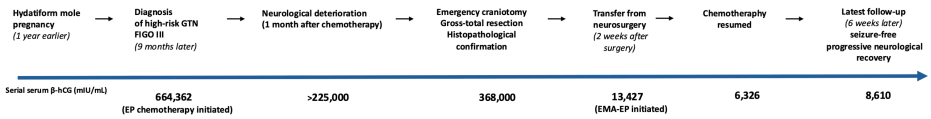



**FIGURE 5: Chronological clinical timeline illustrating disease progression, neurosurgical intervention, serial serum β-hCG levels and oncologic treatment.**

GTN: gestational trophoblastic neoplasia; β-hCG: beta-human chorionic gonadotropin; EP: etoposide and cisplatin; EMA-EP: etoposide, methotrexate, actinomycin D, etoposide and cisplatin.

## Discussion

Brain metastases from gestational choriocarcinoma are uncommon but represent a life-threatening manifestation of gestational trophoblastic neoplasia because of their propensity for intracranial hemorrhage, rapid neurological deterioration, and elevated intracranial pressure. Clinical manifestations commonly include seizures, focal neurological deficits, altered mental status, and acute hemorrhagic stroke, which may necessitate emergency neurosurgical evaluation [2,3,5].

Our patient presented with worsening headache, recurrent focal motor seizures, and progressive left-sided hemiparesis secondary to a large hemorrhagic right frontoparietal intra-axial lesion with significant mass effect. To contextualize these findings, we compared our case with previously published reports of hemorrhagic cerebral metastases from gestational choriocarcinoma managed with neurosurgical intervention (Table *2*). Across the reviewed literature, seizures and focal neurological deficits were recurring clinical manifestations, frequently accompanied by intracranial hemorrhage and mass effect. Although some reports describe favorable outcomes following surgical resection combined with systemic chemotherapy [4,11,12], the optimal role and timing of neurosurgical intervention within multimodal treatment remain unstandardized [2,3]. Mandybur reported substantial mortality among patients with intracranial hemorrhage secondary to metastatic tumors, highlighting the potentially devastating consequences of this presentation [13].

| Author (journal, year) | Study design | Age/Sex | Clinical Presentation | Surgery | Chemotherapy | Outcome |
|---|---|---|---|---|---|---|
| Mandybur [13], American Academy of Neurology, 1977 | Case series | 47/F; 17/F; 32/F | Hemorrhagic cerebral metastases with acute neurological deterioration | Craniotomy in selected patients and hematoma evacuation | Methotrexate in 1 patient after craniotomy | Poor prognosis overall; prolonged survival in one surgically treated patient |
| Dadlani et al. [4], Journal of Cancer Research and Therapeutics, 2010 | Case report | 26/F | Acute neurological deterioration with hemorrhagic cerebral metastasis and hemiparesis | Craniotomy with gross tumor decompression/resection | Post-operative EMA-CO | Neurological improvement with long-term remission |
| Semple et al. [12], International Journal of Gynecological Cancer, 2004 | Two-case report | 25/F; 32/F | Progressive neurological deterioration, hemorrhagic lesions, edema, and mass effect | Craniotomy and tumor debulking | EMA-CO ± cisplatin + WBRT; intracranial progression during chemotherapy in 1 patient | Resolution of hemiparesis and cranial nerve deficits |
| Fujiwara et al. [11], Journal of Neurosurgery, 1992 | Case report | 26/F | Hemiparesis, seizures and progressive consciousness deterioration secondary to intracerebral hemorrhage | Frontoparietal craniotomy with hematoma evacuation and aneurysm resection | Postoperative EMA-CO chemotherapy | Minimal residual upper extremity weakness |
| Zhang et al. [14], Journal of International Medical Research, 2021 | Case report | 55/F | Acute left hemiparesis and intracerebral hemorrhage | Craniotomy and hematoma evacuation and metastatic tumor resection | EP induction before and after surgery | Complete neurological recovery |
| Present Case | Case report | 36/F | Seizures, progressive hemiparesis, and hemorrhagic frontoparietal mass effect despite initiation of chemotherapy | Right frontoparietal craniotomy with gross total resection | Etoposide prior to hemorrhagic progression | Residual left hemiparesis with partial postoperative neurological recovery |

**TABLE 2: Selected PubMed/MEDLINE-indexed neurosurgical reports of hemorrhagic cerebral metastatic choriocarcinoma requiring surgical intervention.**

EMA-CO: etoposide, methotrexate, actinomycin D, cyclophosphamide, and vincristine; EP: etoposide and cisplatin; WBRT: whole-brain radiotherapy.

In contrast, other published cases describe neurological recovery and long-term remission following prompt surgical intervention and multi-agent chemotherapy, including EMA-CO or EP protocols [4,11,12,14]. Collectively, these findings support considering surgery as an adjunct to systemic treatment in carefully selected patients with hemorrhagic lesions and neurological compromise, although differences in clinical presentation, surgical approach, and chemotherapy regimens limit direct comparisons across cases.

A particularly noteworthy finding in our patient was symptomatic intracranial progression despite a substantial decline in serum β-hCG levels following the initiation of systemic chemotherapy. Serum β-hCG remains a cornerstone biomarker for the diagnosis, risk stratification, and therapeutic monitoring of gestational trophoblastic neoplasia [3,6]. However, as observed in our patient and described in a subset of cases by Semple et al. [12], intracranial clinical or radiological progression may occasionally occur despite an ongoing systemic biochemical response. This apparent dissociation suggests that favorable serum β-hCG trends may not invariably reflect intracranial disease control.

The underlying mechanism remains uncertain. However, the highly vascular nature of choriocarcinoma and its propensity for vascular invasion may contribute to intracranial hemorrhage. Reported mechanisms include tumor-associated vessel wall erosion and pseudoaneurysm formation [15]. Although these mechanisms provide a plausible explanation for hemorrhagic progression despite a favorable systemic biochemical response, they cannot be established as the cause of progression in our patient. This observation highlights the importance of maintaining a high index of suspicion when new or progressive neurological symptoms develop during treatment despite declining serum β-hCG levels. In such cases, follow-up neuroimaging could be considered to reassess intracranial disease status.

Although multi-agent chemotherapy remains central to the treatment of gestational trophoblastic neoplasia with brain metastases because of the chemosensitivity of this disease, neurosurgical intervention is generally limited to selected cases requiring symptomatic palliation or relief of intracranial pressure and hemorrhage [2,3,6]. Surgery may be warranted in patients with symptomatic hemorrhagic metastases, significant mass effect, refractory seizures, progressive neurological deterioration, or impending cerebral herniation, particularly when urgent decompression is necessary to address neurological compromise [2,3,12].

Our patient met several of these criteria, including a large hemorrhagic metastasis, recurrent seizures, progressive hemiparesis, and marked mass effect. These findings supported the decision to perform urgent microsurgical resection, which achieved decompression and was followed by complete resolution of expressive aphasia and progressive neurological recovery. This favorable course illustrates the potential role of urgent neurosurgical intervention as part of multimodal management in selected patients with hemorrhagic brain metastases and significant neurological compromise.

## Conclusions

As a single-case report, our findings cannot be generalized to all patients. Nevertheless, this report contributes to the limited literature describing neurosurgical management of hemorrhagic cerebral metastases from choriocarcinoma.

This case highlights the aggressive neurovascular behavior of metastatic choriocarcinoma and its potential for rapid intracranial progression despite ongoing systemic therapy. In patients with elevated serum β-hCG levels and a recognized risk of metastatic disease, vigilant clinical follow-up is essential. New or worsening neurological symptoms could prompt careful evaluation and consideration of timely neuroimaging, even when serum β-hCG levels are declining. Early recognition of neurological deterioration may facilitate timely multidisciplinary management, including neurosurgical intervention when indicated, and help reduce neurological morbidity while allowing continuation of oncologic treatment.

## Additional Information

### Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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**Supervision:** Lizzy Y. Lázaro Ramirez, Jesús A. Morales-Gómez, Angel Martínez Ponce De Leon, Nilsa D. Martinez-Rosales

### Disclosures

**Human subjects:** Informed consent for treatment and open access publication was obtained or waived by all participants in this study. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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