

Pathological transformation of rectal adenocarcinoma to choriocarcinoma in a male patient: A case report and review of the literature

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Abstract. Choriocarcinoma associated with poorly differentiated somatic cell carcinoma is a rare disease and there are limitations in its clinical management. The present study describes the case of a 62-year-old male patient with rectal adenocarcinoma; after undergoing three lines of standard colorectal cancer treatment, the rectal adenocarcinoma exhibited abnormal differentiation into choriocarcinoma with a notable increase in serum β -human chorionic gonadotropin (β -hCG). The patient underwent two cycles of EMP/CO chemotherapy (etoposide, methotrexate, cisplatin/cyclophosphamide and vincristine); the standard treatment regimen for choriocarcinoma is EMA/CO (etoposide, methotrexate, actinomycin D/cyclophosphamide and vincristine), whereas in the present case, cisplatin was used in place of actinomycin D. Subsequently, β -hCG levels exhibited a marked decrease. After the first cycle of chemotherapy, the β -hCG level decreased from >15,000 mIU/ml to 4,109 mIU/ml. Notably, the second cycle of chemotherapy was delayed due to fever and perianal abscess, after which the β -hCG level rose again to >15,000 mIU/ml; yet, after chemotherapy on days 1-2 of the second cycle, the β -hCG level decreased again to 11,900 mIU/ml. However, due to poor tolerance and rapid disease progression, the patient was unable to continue receiving treatment and ultimately died. Given the rarity and clinical intractability of this disease, the relevant literature was reviewed to offer insights into its mechanism, diagnosis and treatment. The current case report illustrates the pathological

transformation from rectal adenocarcinoma to choriocarcinoma in a male patient, which provides evidence for the abnormal differentiation of somatic cell carcinoma into choriocarcinoma. Concurrently, post-treatment β -hCG level changes validated the therapeutic efficacy of EMP/CO. Furthermore, given the promising advances in antiangiogenic and immunotherapy research, these therapeutic approaches warrant further exploration alongside chemotherapy.

Introduction

Choriocarcinoma is a highly vascularized and invasive tumor composed of cytotrophoblasts and syncytiotrophoblasts without the formation of chorionic villi, which can be classified into gestational choriocarcinoma (GC) and non-GC (NGC) based on its origin and pathogenesis. GC is a type of gestational trophoblastic neoplasia that can arise following any type of gestation and at any interval after conception. Approximately 60% of gestational trophoblastic neoplasia cases arise from a molar pregnancy, 30% follow a miscarriage, and the remaining 10% occur after an ectopic or term pregnancy (1). Compared with GC, which is associated with the paternal chromosome (2), NGC is derived exclusively from the patient's own germline. Clinically, NGC is rarer, more aggressive, less responsive to treatment and has a poorer prognosis (3). NGC can be further divided into tumors of germ cell origin and tumors of non-germ cell origin (4). NGC originating from germ cells can occur in gonadal or extragonadal tissues; the former is mostly mixed with other germ cell tumors and pure choriocarcinoma is less common (5-7); the latter is mostly found in midline structures, such as the pineal gland, mediastinum and retroperitoneum (8). The developmental mechanism of germ cell-derived NGC has not yet been clarified. It may originate from the aberrant migration of primordial germ cells during embryonic development, or from the metastasis of spontaneously regressing choriocarcinoma of the gonad (9). In addition, NGC of non-germ cell origin is associated with high-grade somatic cell carcinoma with clinical manifestations typically reflecting the primary tumor site. This entity is rare in clinical practice, and studies on its cytogenetic and molecular abnormalities are scarce (3,10,11).

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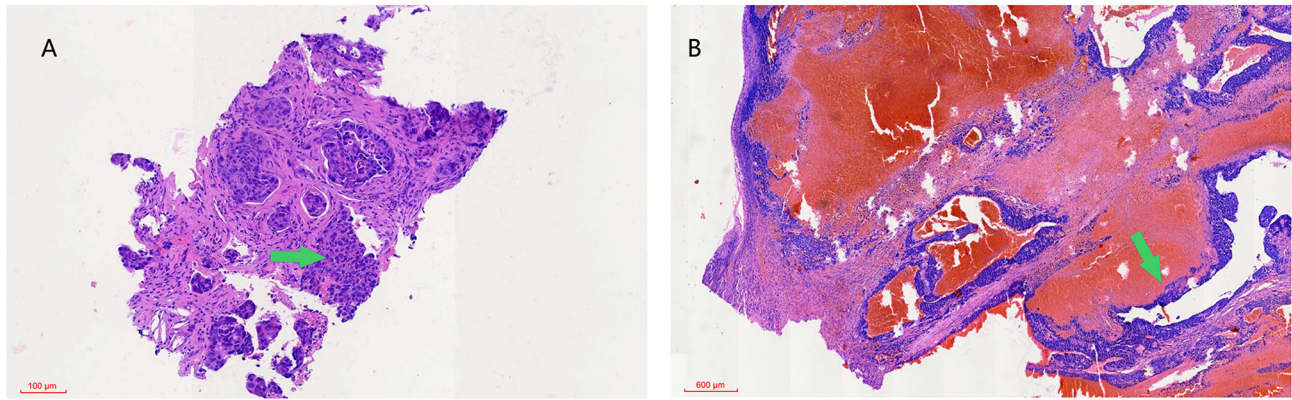


Figure 1. (A) Pelvic mass pathological staining in November 2023. The arrow indicates the tumor area (hematoxylin and eosin). (B) Liver tumor pathological staining in June 2024. The arrow indicates the tumor area (hematoxylin and eosin).

Given that clinical and preclinical studies on these types of choriocarcinoma are currently insufficient, future research is required into their molecular mechanisms and clinical diagnosis and treatment. The present study describes the rare case of a male patient with rectal adenocarcinoma that pathologically transformed into choriocarcinoma, providing insight into the pathogenesis and clinical management of this rare disease.

Case report

In August 2022, due to anal discomfort and frequent bowel movements, a 62-year-old male patient was subjected to computed tomography (CT) of the abdomen at Renji Hospital, Shanghai Jiao Tong University School of Medicine (Shanghai, China), which revealed focal thickening of the rectal wall. Colonoscopy pathology showed high-grade intraepithelial neoplasia and carcinoma in some areas. At that time, the patient opted for traditional Chinese medicine and declined surgical or other conventional antitumor therapies. In November 2023, abdominal CT revealed notable thickening of the focal part of the rectal wall with multiple metastatic enlarged lymph nodes, which had markedly progressed from the previous scan. A CT-guided pelvic mass biopsy was performed, and the pathology revealed a poorly differentiated adenocarcinoma (paraffin-embedded; 4-μm thick; light microscopy). The tumor cells exhibited the following immunohistochemical tissue staining results: Villin(+), carcinoembryonic antigen (CEA)(+), CK7(-), CK20(+), Ki67(80%), NKX3.1(-), p63(-), GATA-3(-), InSM1(-) and human chorionic gonadotropin (HCG)(-) (Figs. 1A, 2 and 3). H&E staining was performed as follows: Samples were fixed in 10% neutral buffered formalin at room temperature for 12-48 h, and then stained with hematoxylin for 3-10 min and eosin for 1-3 min at room temperature. For immunohistochemistry, sections were blocked with 5% BSA (Shanghai Macklin Biochemical Co., Ltd.) at room temperature for 20 min. The primary antibodies included Villin, CEA, CK7, CK20, Ki67, NKX3.1, p63, GATA-3, InSM1, HCG (1:100; 4°C overnight/12-16 h or room temperature for 30 min; Beijing Zhongshan Golden Bridge Biotechnology Co., Ltd.). Sections were then incubated with HRP-conjugated goat anti-mouse/rabbit secondary antibody (ready-to-use; Fuzhou Maixin Biotechnology Development Co., Ltd.) at room temperature for 30 min. Notably, HCG was

added as a follow-up test. Genetic testing showed the presence of the KRAS p.G13D mutation (fluorescent PCR method performed according to the manufacturer's protocol; AmoyDx Human KRAS/NRAS/PIK3CA/BRAF Gene Mutation Detection Kit; Amoy Diagnostics Co., Ltd.). These findings suggested rectal cancer with extensive abdominopelvic lymph node metastasis. The patient underwent further tumor progression despite receiving two standard treatment protocols for colorectal cancer. Of these, first-line treatment was initiated in November 2023 for a total of five cycles: Bevacizumab, 7.5 mg/kg [intravenous guttate (ivgtt)] on day 1; oxaliplatin, 130 mg/m² (ivgtt) on day 1; and capecitabine, 1,000 mg/m² [oral administration (po)] twice a day on days 1-14 (once every 3 weeks). Second-line treatment was initiated in March 2024 for a total of three cycles: Bevacizumab 7.5 mg/kg (ivgtt) on day 1; irinotecan, 200 mg/m² (ivgtt) on day 1; and raltitrexed 3 mg/m² (ivgtt) on day 1 (once every 3 weeks). Following disease progression on second-line therapy, the patient had metastases in the liver, right lung and multiple lymph nodes throughout the body; therefore, they received treatment with oral trifluridine and tipiracil (TAS-102) chemotherapy [35 mg/m² (po) twice a day on days 1-5 (once every 2 weeks)] from June 2024.

A total of 6 days after starting treatment with TAS-102, the patient presented with dizziness and abdominal distension. Abdominal CT revealed that the metastasis in the liver was markedly larger than before and had broken through the hepatic envelope, with rupture and hemorrhage visible (Fig. 4). Emergency surgery was performed 1 day later. The postoperative pathology (paraffin-embedded tissues; 4-μm thick; light microscopy) was as follows: Liver tumor cells: CK19(+), AFP(-), SALL4(+), Ki67 (80%), CK8(+), VIM(-), p40(-), CK20(-), HCG(+), CD30(-), PLAP(+), OCT3/4(-), SATB2(-), GS(-), Inhibin(+), GATA3(+), CD10(+), p63(some cells +) and programmed death-ligand 1 (PD-L1) CPS 50 (Figs. 1B, 5 and 6). No mutation was detected in the KRAS gene test (PD-L1 and KRAS gene tests were not performed during the initial diagnosis and treatment and were added as a follow-up test). The immunohistochemistry results were consistent with choriocarcinoma, with no adenocarcinoma component. Postoperative measurement of serum β-hCG using a chemiluminescent assay, performed according to

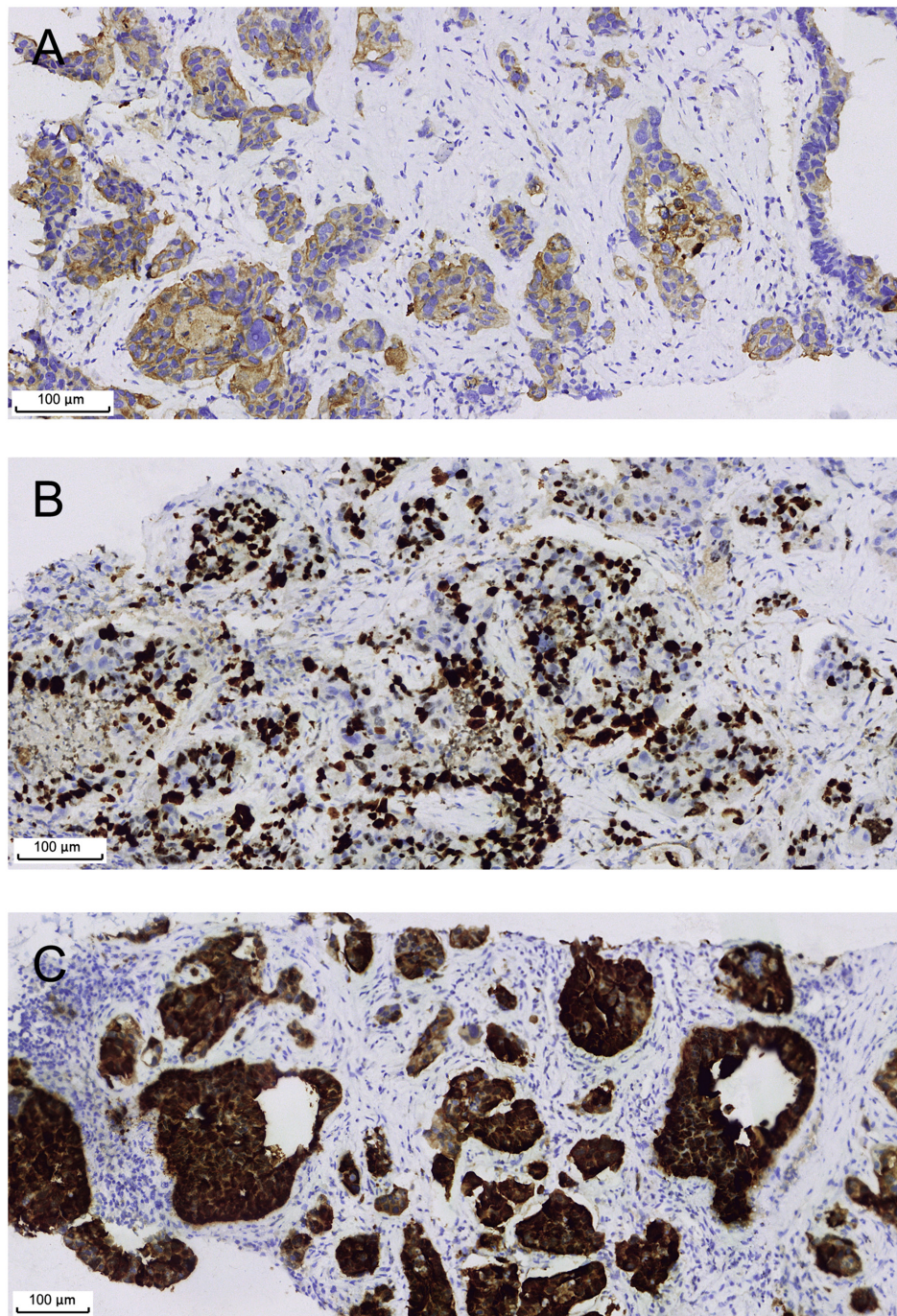


Figure 2. Immunohistochemistry images of positive staining indicative of adenocarcinoma pathology in November 2023. (A) CK20 (+); (B) Ki67 (80%); (C) Villin (+).

the manufacturer's protocol, was >15,000 mIU/ml (reference range, <5 mIU/ml; Beckman Coulter, Inc.). Thus, given the history of rectal cancer, transformation of rectal adenocarcinoma into choriocarcinoma was determined clinically. Rather than the standard EMA/CO (etoposide, methotrexate, actinomycin D/cyclophosphamide and vincristine) regimen, cisplatin therapy replaced actinomycin D due to accessibility. Two cycles of EMP/CO regimen chemotherapy were administered in August and September 2024, as follows: Etoposide, 100 mg/m² (ivgtt) on days 1-2; methotrexate, 100 mg/m² [intravenous (iv)] on day 1 + 200 mg/m² (continuous intravenous infusion over 12 h); cisplatin, 25 mg/m² (ivgtt) on days 1-2/cyclophosphamide

600 mg/m² (ivgtt) on day 8; vincristine, 2 mg (iv) on day 8; and leucovorin rescue therapy following methotrexate chemotherapy (once every 2 weeks). During this period, the second treatment was delayed due to a perianal abscess and recurrent fever. Serum β -hCG was monitored during chemotherapy and showed two decreases, namely 4,109 mIU/ml and 11,900 mIU/ml, after which it increased again to >15,000 mIU/ml (Fig. 7).

In November 2024, the patient checked into the Emergency Department at Renji Hospital, Shanghai Jiao Tong University School of Medicine, with a fever, and a CT scan suggested that the tumor had widely metastasized, involving multiple organs with obvious progression (Fig. 8). Furthermore, cranial CT

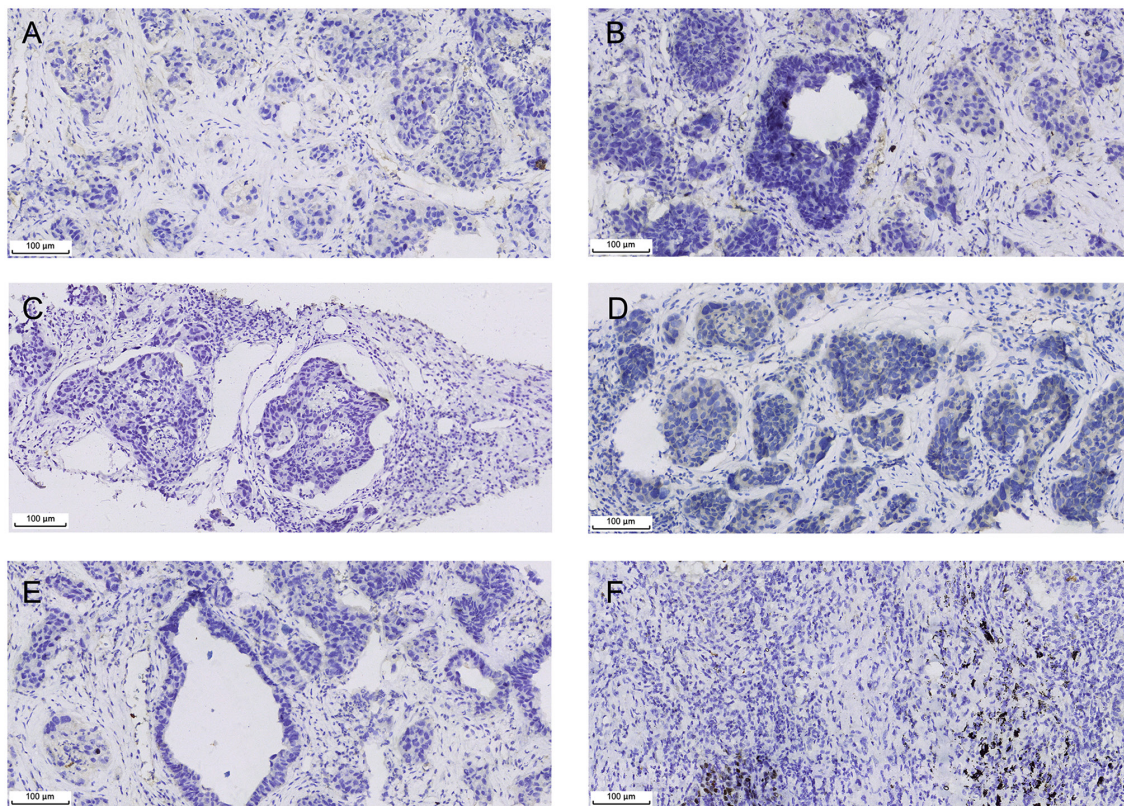


Figure 3. Immunohistochemistry images of negative staining indicative of adenocarcinoma pathology in November 2023. (A) CK7 (-); (B) GATA-3 (-); (C) human chorionic gonadotropin (-); (D) InSM1 (-); (E) NKX3.1 (-) and (F) P63 (-).

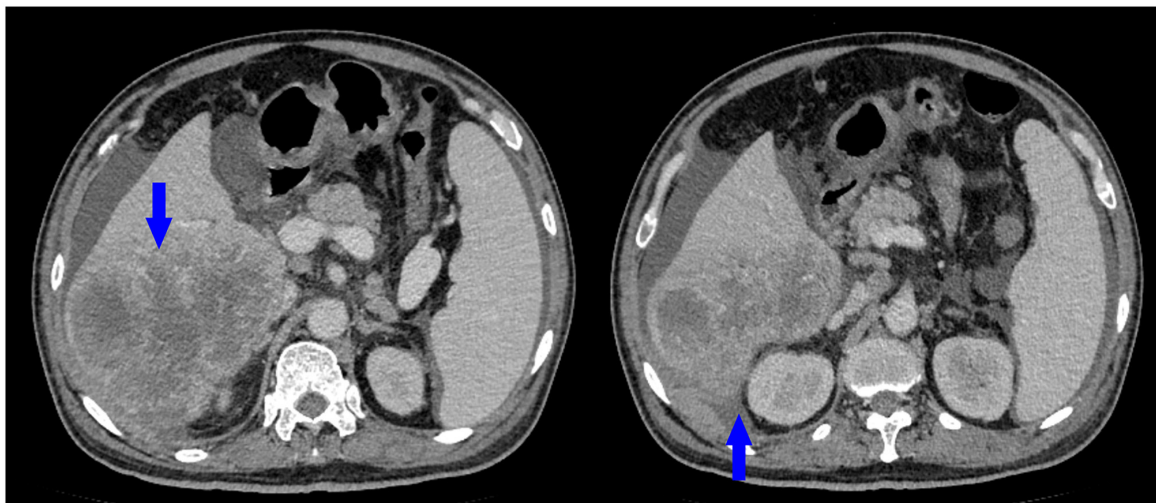


Figure 4. Computed tomography images of the ruptured liver metastasis with hemorrhage in June 2024. The arrows indicate the liver space-occupying lesion and liver rupture with hemorrhage.

showed multiple space-occupying lesions across the midline of the bilateral frontal lobes, which were considered metastasis with hemorrhagic changes (Fig. 8). Due to the poor general condition of the patient, the family decided against further aggressive antitumor therapy, and the patient died after being given optimal symptomatic supportive therapy (including anti-infective therapy, pain management and nutritional support). The timeline of the present case is illustrated in Fig. 9.

Discussion

Choriocarcinoma associated with high-grade somatic cell carcinoma is mainly found in the gastrointestinal tract, where it tends to exhibit biphasic tumor growth (12,13), such as in colorectal adenocarcinoma with choriocarcinoma differentiation (14) (Table I). Although the molecular mechanisms underlying the development of choriocarcinoma have not been fully elucidated, studies support the notion of the abnormal differentiation of

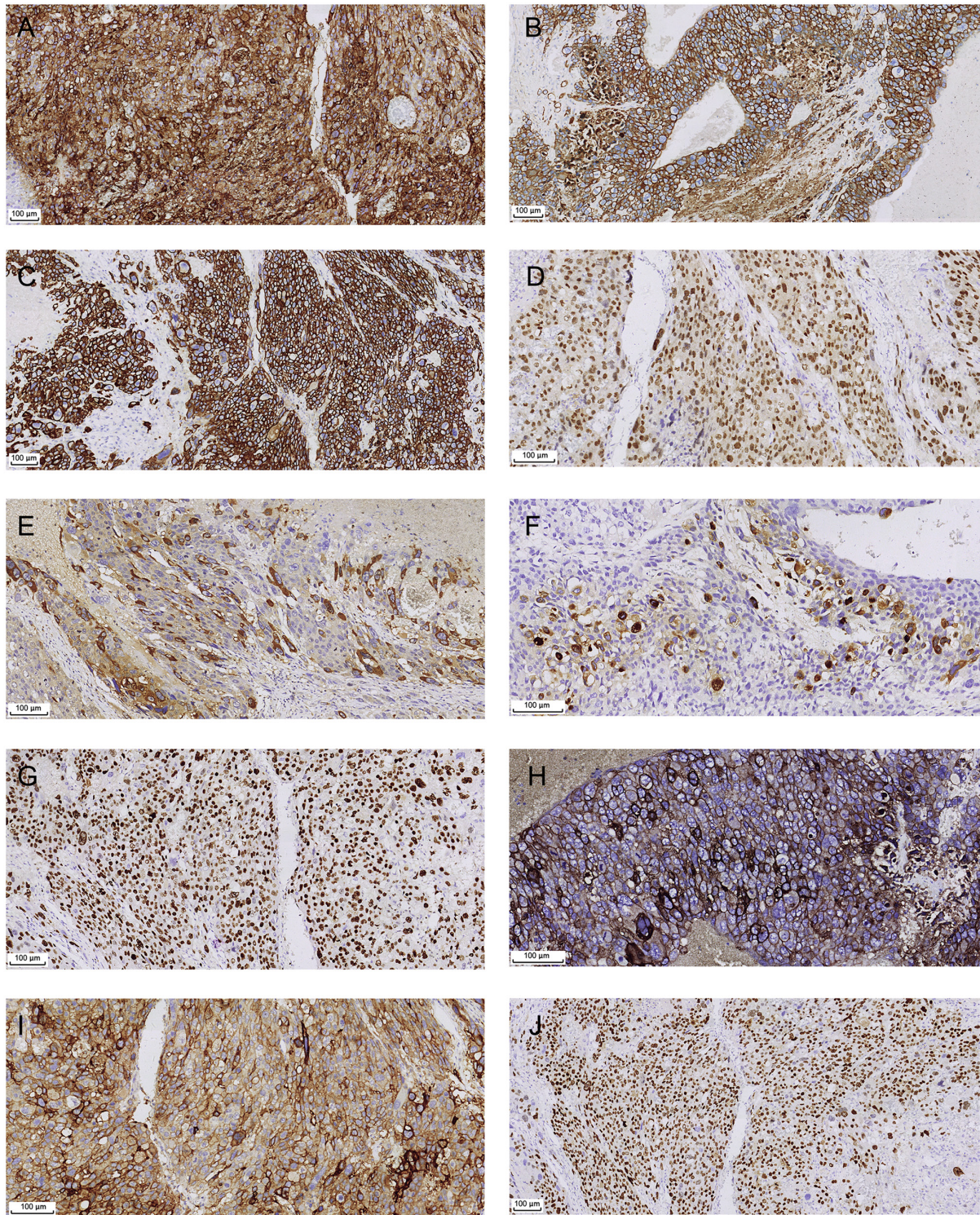


Figure 5. Immunohistochemistry images of positive staining indicative of choriocarcinoma pathology in June 2024. (A) CD10 (+); (B) CK8 (+); (C) CK19 (+); (D) GATA3 (+); (E) human chorionic gonadotropin (+); (F) Inhibin (+); (G) Ki67 (80%); (H) programmed death-ligand 1 22C3 (combined positive score 50); (I) PLAP (+) and (J) SALL4 (+).

somatic cell carcinoma (15,16). In view of this, the present case report illustrates the pathological transformation from rectal adenocarcinoma to choriocarcinoma in a male patient, providing evidence for the abnormal differentiation of somatic cell carcinoma into choriocarcinoma from a clinical perspective (17). In addition, some cases have shown only pure choriocarcinoma without a somatic cell carcinoma component, which may be due

to the more aggressive nature of the fast-growing choriocarcinoma component (18). For example, in the current case report, the pathological specimen obtained after surgery for ruptured liver metastasis did not exhibit any adenocarcinoma component; only choriocarcinoma was observed.

NGC is not associated with any form of gestation and is therefore genetically related only to the patient. In 2017,

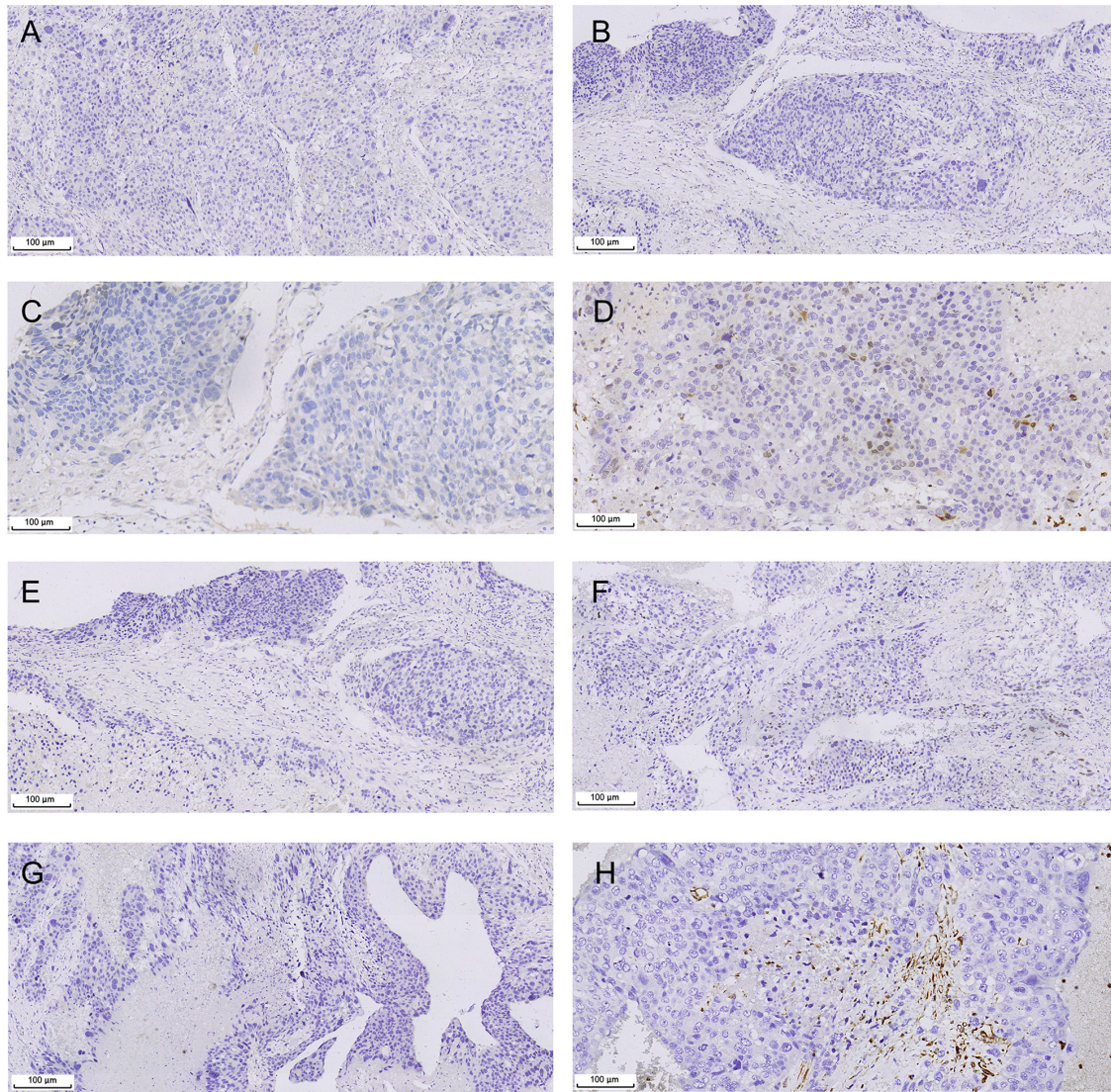


Figure 6. Immunohistochemistry images of negative staining indicative of choriocarcinoma pathology in June 2024. (A) AFP (-); (B) CD30 (-); (C) CK20 (-); (D) GS (-); (E) OCT3/4 (-); (F) P40 (-); (G) SATB2 (-) and (H) VIM (-).

Mello *et al* (10) performed genomic profiling of NGC and identified the only significant chromosome copy number change to be 21p11, potentially implicating this locus in disease development. As for NGC associated with poorly differentiated somatic cell carcinoma, its rarity makes it difficult to obtain clear breakthroughs in both clinical and preclinical studies. Based on the current findings, it is believed that this type of choriocarcinoma originates from the abnormal differentiation of somatic cell carcinoma. Krulewski and Cohen (19) revealed that, at the epidemiologic level, primary gastric choriocarcinoma had a similar mean age at onset, male-to-female ratio and geographic distribution to primary gastric adenocarcinoma. Secondly, Skinner and Whitehead (20) and Campo *et al* (21) reported varying levels of β -hCG expression in colorectal adenocarcinoma cells as early as 1981 and 1987, respectively, and β -hCG expression may be associated with poor prognostic factors, such as tumor aggressiveness and lymph node metastasis. Similarly, in 1986, Lind and Haghighi (22) observed positive staining for CEA in colonic choriocarcinoma cells. Moreover, in 2001, Liu *et al* (23) detected copy number gains in chromosomes 1p34-36, 12, 17,

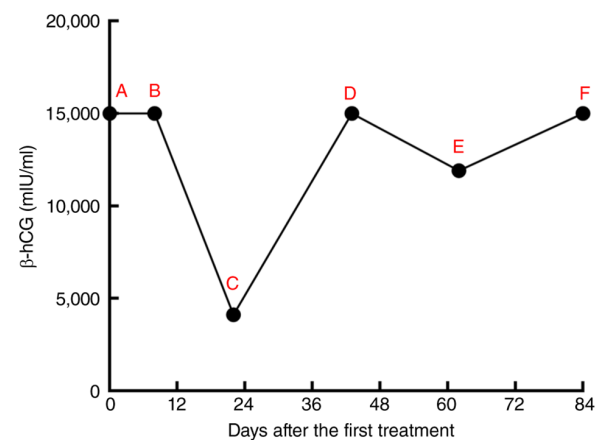


Figure 7. β -hCG trend during treatment. The actual values presented as A, B, D and F are >15,000 mIU/ml, but they are all shown as 15,000 mIU/ml in the figure. β -hCG levels were detected at the following timepoints: A, EMP/CO treatment cycle 1, days 1-2; B, EMP/CO treatment cycle 1 day 8; C, symptoms of perianal abscess and fever, chemotherapy was halted; D, EMP/CO cycle 2, days 1-2; E, EMP/CO cycle 2, day 8; F, disease progression and death. β -hCG, β -human chorionic gonadotrophin.

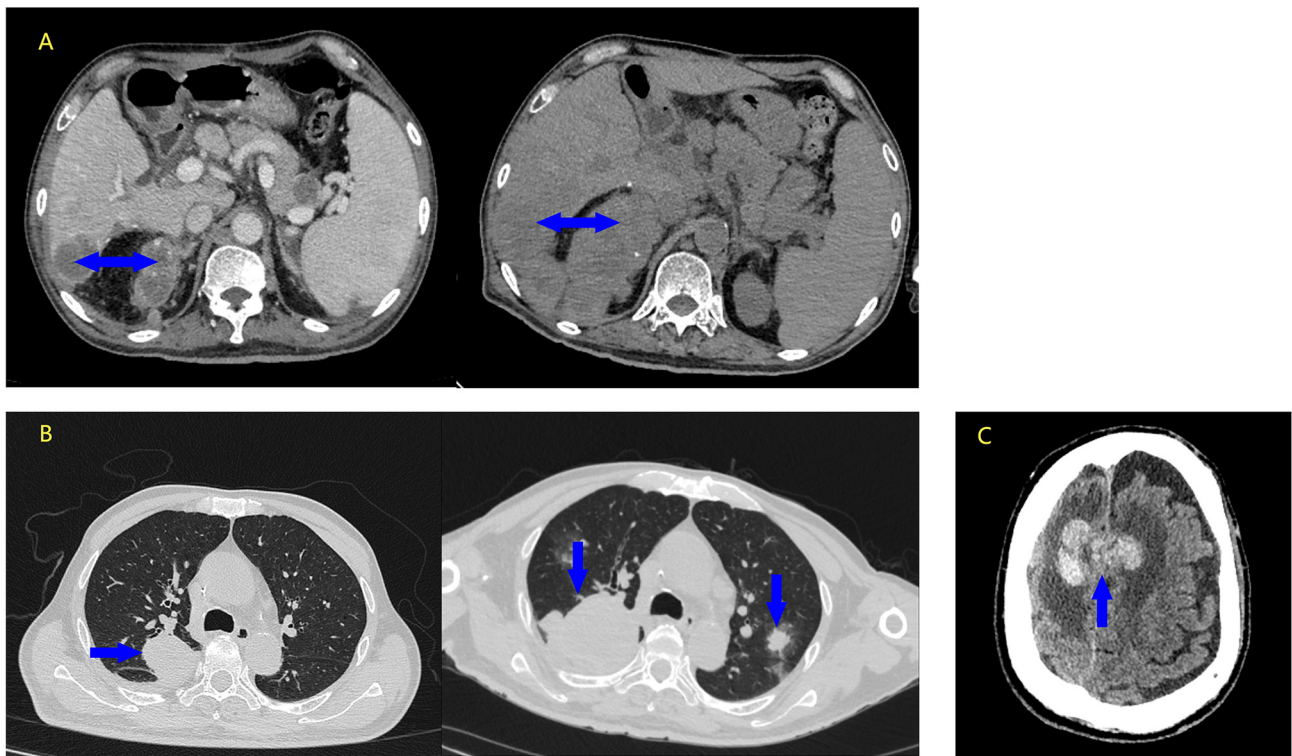


Figure 8. (A) Comparative imaging of abdominal lesions in August and November 2024. Arrows indicate the liver and adrenal lesions. (B) Comparative imaging of lung lesions in August and November 2024. Arrows indicate the lung lesions. (C) Intracranial metastasis with hemorrhage in November 2024. The arrow indicates the intracranial lesions.

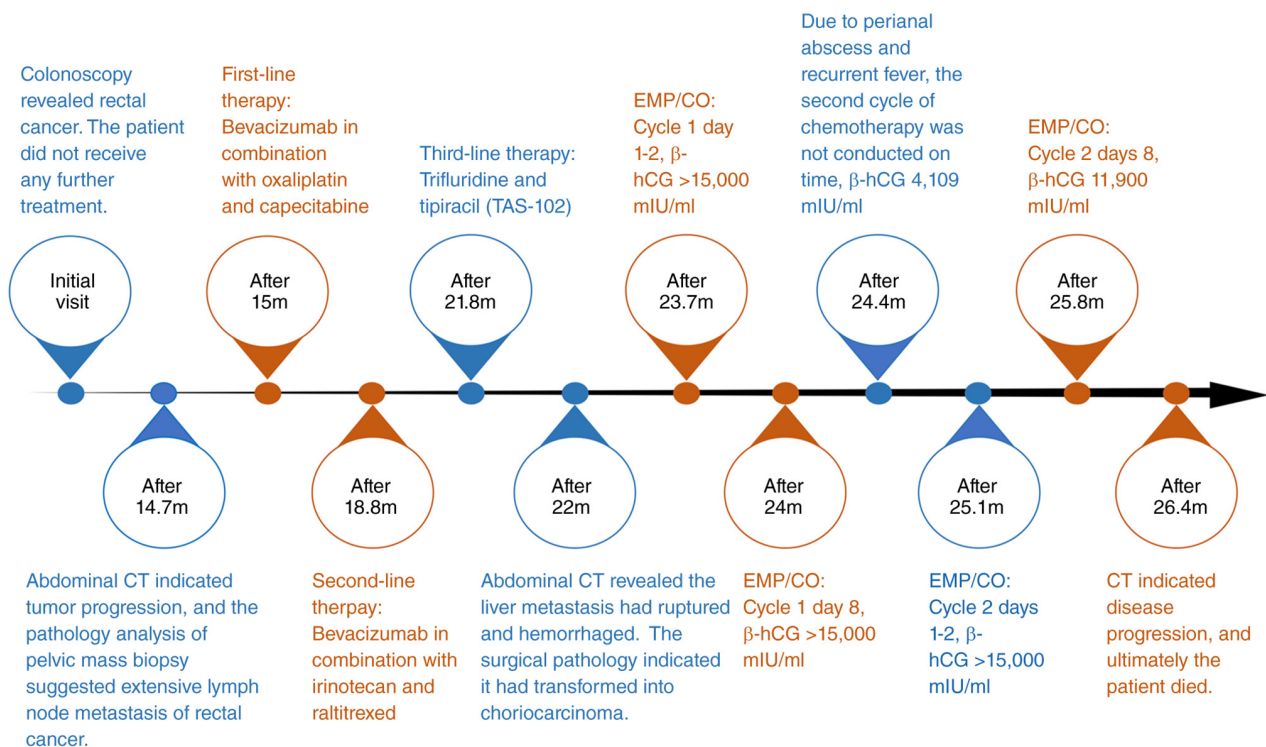


Figure 9. A schematic timeline from the patient's initial visit to eventual death. β -hCG, β -human chorionic gonadotrophin; CT, computed tomography; m, months.

18p and 20, as well as losses in chromosome 18q, in a case of primary gastric choriocarcinoma, in which, with the exception of chromosome 12, the other chromosomal alterations were

highly consistent with those of primary gastric adenocarcinoma. Moreover, the investigators microscopically identified minute adenocarcinoma components and transitional cells exhibiting

Table I. Case reports of primary choriocarcinoma of the gastrointestinal tract.

First author, year	Primary site	Sex	Age, years	Treatment	β-hCG response	Overall survival	(Refs.)
Liu <i>et al</i> , 2001	Stomach	Male	66	Surgery	Not available	7 days	(23)
Mardi <i>et al</i> , 2014	Rectum	Female	54	Surgery; 5-FU	Postoperative: 4,568 mIU/ml	50 days	(12)
Mitselou, 2020	Sigmoid colon	Female	61	Surgery; chemotherapy	Postoperative: 112,000 mIU/ml	2 months	(15)
Verbeek <i>et al</i> , 2004	Rectum	Female	54	Surgery; EPC	Maximum value: 102,000 mIU/ml; post-treatment minimum value: Negative	8 months	(13)
Zhong and Yang, 2024	Rectum	Female	60	FAEV and bevacizumab; EMA/CO; PD-1 inhibitor and bevacizumab	Maximum value: 38,643 mIU/ml; post-treatment minimum value: >500 mIU/ml	8 months	(17)
Hirotsu <i>et al</i> , 2019	Stomach	Male	78	Surgery; EMA/CO	Maximum value: 120 ng/ml; post-treatment: Not available	10 months	(31)
Liu <i>et al</i> , 2001	Stomach	Male	55	Not available	Not available	12 months	(23)
Harada <i>et al</i> , 2012	Sigmoid colon	Female	58	Surgery; EMA; 5-FU and CDDP	Maximum value: 2,420 mIU/ml; post-treatment minimum value: Negative	5 years	(16)
Takahashi <i>et al</i> , 2013	Stomach	Female	65	Surgery; EMA/CO; RFA	Maximum value: 12,000 mIU/ml; post-treatment minimum value: Negative	9 years	(18)
Jiang <i>et al</i> , 2013	Ascending colon	Male	36	Surgery; BEP	Minimum value: 3.38 mIU/ml; post-treatment maximum value: 10,000 mIU/ml	Not available	(32)

β-hCG, β-human chorionic gonadotrophin; 5-FU, 5-fluorouracil; EPC, etoposide, cisplatin and ifosfamide; FAEV, fluorouracil, actinomycin D, etoposide and vincristine; EMA/CO, etoposide, methotrexate, actinomycin D/cyclophosphamide and vincristine; PD-1, programmed cell death protein 1; CDDP, cisplatin; RFA, radiofrequency ablation; BEP, bleomycin, etoposide and cisplatin.

features of both adenocarcinoma and choriocarcinoma (23), findings consistent with the microscopic observations reported by Gorczyca and Woyke (24), suggesting abnormal differentiation in the neoplastic cells. The aforementioned studies provide evidence of homology between somatic cell carcinoma and choriocarcinoma; however, further studies are needed to prove the cause of such abnormal differentiation and its rarity.

Newlands (25) revealed that only 9% of patients with gestational trophoblastic neoplasia died from the disease, compared with a mortality rate of 84% in patients with non-gestational origins. In particular, NGC associated with somatic cell carcinoma, while being rare, is often predicted to be characterized by low clinical target response rates and short remission periods, with more aggressive clinical progression and poorer prognosis.

The majority of patients tend to die within 12-18 months of surgery due to advanced disease stage and the lack of effective treatment (26). As reported in the present case, most patients with NGC ultimately derive limited clinical benefit from chemotherapy. However, due to the absence of standardized treatment protocols, surgery and chemotherapy remain primary therapeutic approaches for NGC, with chemotherapy regimens typically referenced to those for other germ cell tumors, such as EMA/CO (8) and BEP (bleomycin, etoposide and cisplatin regimen) (27), or other chemotherapy regimens that use this class of drugs as a cornerstone. Cisplatin, a foundational chemotherapeutic agent for such diseases, is classified alongside actinomycin D as a cell-cycle-nonspecific drug. Cisplatin forms DNA cross-links that disrupt DNA structure, halting replication and transcription, whereas actinomycin D intercalates into DNA and inhibits RNA polymerase, thereby blocking RNA transcription. Both agents can activate pathways such as p53 to initiate the intrinsic apoptotic pathway (28-30). In the present case, because actinomycin D was unavailable, cisplatin was substituted, which slightly reduced the risk of myelosuppression, but increased the incidence of gastrointestinal adverse reactions and nephrotoxicity. In terms of efficacy, after pathological confirmation of choriocarcinoma transformation in the present case, the patient exhibited a marked decline in β -hCG levels following the first chemotherapy cycle. However, treatment was delayed due to a perianal abscess and fever, resulting in a subsequent β -hCG resurgence. Due to the highly aggressive nature of the disease, the poor general condition of the patient, the suboptimal response to chemotherapy and potential chemoresistance, disease progression and mortality ultimately occurred. β -hCG is secreted by trophoblast cells, is largely unaffected by inflammation or fever, and can be used as a straightforward indicator to monitor the efficacy of cancer treatment (31,32). In this case, changes in β -hCG indicate that the treatment is not completely ineffective. However, despite the efficacy of the regimen, maximizing therapeutic benefits in patients with poor physical fitness is a focus of future diagnosis and treatment of this disease.

With the current treatment dilemma, effective treatment options need to be further explored in addition to conventional chemotherapy. Given the highly vascularized nature of choriocarcinoma, antiangiogenic therapy may be of benefit. Notably, while anti-vascular endothelial growth factor (VEGF) upregulates endoglin expression, endoglin blockade conversely enhances VEGF expression; this reciprocal relationship suggests that dual blockade of endoglin and VEGF may yield unexpected therapeutic efficacy (33,34). This treatment modality has been attempted in a patient with GC after multiple lines of multidrug resistance and achieved durable remission for >28 months (35), implying that this type of targeted therapy regimen may be considered a novel treatment option for patients with NGC in the future, potentially overcoming chemotherapy resistance to improve clinical outcomes. In 2019, Lu *et al* (36) revealed that PD-L1 was strongly expressed in most trophoblastic tumors, including NGC associated with somatic cell carcinoma. As in the present case, the subsequent PD-L1 test on the liver metastasis was positive, suggesting that immune checkpoint blockade may be an effective therapeutic option for NGC, warranting further exploration in clinical and preclinical studies. On the one hand, given that anti-VEGF therapy and immune checkpoint inhibitor therapy are not

conventional first-line options, they were not selected as the initial treatment regimen in the current case. Subsequently, due to the rapid disease progression, the patient became ineligible for further antitumor therapy, thereby precluding the opportunity to attempt alternative medications. On the other hand, the patient in the present case had received long-term bevacizumab (anti-VEGF therapy) during prior treatment for rectal adenocarcinoma. However, the exact timing of pathological transformation to choriocarcinoma could not be determined and the possibility of pre-existing bevacizumab resistance could not be ruled out. With regard to immune checkpoint inhibitors, a PD-L1 test was not performed prior to treatment; however, had positive PD-L1 expression been detected before therapy, a different treatment regimen might have been chosen, potentially leading to better outcomes. This, to some extent, emphasizes the importance of prospectively evaluating PD-L1 in this type of disease in the future. To date, only a minority of patients with somatic carcinoma-associated NGC have demonstrated favorable clinical outcomes in reported cases. Most of them underwent complete resection at an early stage and the patients were generally in good condition, making them eligible for a variety of treatment options and regimens (16,18). Consequently, future efforts should focus on actively exploring novel therapeutic modalities for this type of NGC.

The present case report illustrates the transformation from rectal adenocarcinoma to choriocarcinoma, encompassing subsequent treatment, monitoring and prognosis. However, it has certain limitations: Firstly, two immunohistochemical staining figures were unavailable for this case, therefore, all presented results are derived from the original pathology reports; secondly, as a single case, it lacks generalizability, and the non-standard chemotherapy regimen used may have impacted treatment efficacy; thirdly, genomic confirmation of the clonal relationship is lacking, resulting in an incomplete case presentation. Therefore, future research is warranted to further elucidate the mechanisms underlying this disease transformation and optimize treatment selection, ultimately aiming for notable breakthroughs and improved clinical outcomes.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

YL, KW and HL completed conception and design. ZP, XZha and SR obtained medical images and pathology figures. XZho, XM and QW analyzed and interpreted the data. YL and HL confirm the authenticity of all the raw data. All authors read and approved the final manuscript.

Ethics approval and consent to participate

Not applicable.

Patient consent for publication

Written informed consent was obtained from the patient before their death for publication of the present case report.

Competing interests

The authors declare that they have no competing interests.

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