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Invasive Breast Cancer Complicated by Colitis After TCbHP Chemotherapy: A Case Report

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Patient: Female, 53-year-old
Final Diagnosis: Breast cancer • colitis
Symptoms: Abdominal pain • diarrhea
Clinical Procedure: —
Specialty: Oncology • Surgery

Objective: Unusual clinical course

Background: Chemotherapy is a cornerstone of systemic treatment for breast cancer. Common adverse effects include nausea, vomiting, diarrhea, and myelosuppression; chemotherapy-induced colitis is rare, and its underlying mechanisms remain unclear. We aim to raise awareness of this uncommon but serious adverse event and describe a practical approach to regimen modification.

Case Report: A 53-year-old woman underwent left total mastectomy for invasive breast carcinoma (pT2N0M0, Stage IIA; ER [-]/PR [-]/HER2 [3+]). She received adjuvant chemotherapy with the TCbHP regimen (docetaxel, carboplatin, trastuzumab, and pertuzumab). One week after the first cycle, she developed abdominal pain and diarrhea. Colonoscopy revealed colitis. After dose reduction to 80% in the second cycle, abdominal pain recurred, and colonoscopy demonstrated colonic ulcers. The regimen was subsequently changed to THP (nab-paclitaxel, trastuzumab, and pertuzumab); symptoms then resolved, and follow-up colonoscopy showed ulcer healing.

Conclusions: Colitis associated with breast cancer chemotherapy is rare, presenting diagnostic and therapeutic challenges. Severe complications, including colonic necrosis, may develop if not promptly addressed. In the present case, timely intervention and regimen modification prior to the onset of intestinal necrosis appeared to prevent serious sequelae, including the need for intestinal resection. However, given the limitations of a single case report and the use of combination chemotherapy, a definitive causal relationship cannot be established.

Keywords: breast neoplasms • chemotherapy, adjuvant • colitis

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Introduction

Breast cancer is currently the most prevalent malignancy among women. Estimates for the United States comprised 310 720 new cases and 42 250 deaths in 2024 [1]. Breast cancer treatment involves a comprehensive approach, including surgery, chemotherapy, radiotherapy, endocrine therapy, targeted therapy, and immunotherapy. Chemotherapy remains a cornerstone of systemic treatment. Chemotherapy-induced colitis is defined as colonic inflammation caused by chemotherapeutic agents. Its incidence is low, and no cases of colitis associated with the TCbHP regimen (docetaxel, carboplatin, trastuzumab, and pertuzumab) have been reported to date [2,3]. Here, we describe a patient who presented with abdominal pain and diarrhea after the first cycle of adjuvant chemotherapy. Although these symptoms are common adverse effects of chemotherapy, they can lead to delays in diagnosis and treatment if not given sufficient attention. Chemotherapy-induced colitis in patients with breast cancer is rare but can produce serious complications, including colonic perforation and infectious peritonitis. Our detailed analysis of the present case indicated that rare adverse drug reactions require a high index of suspicion because even commonly used chemotherapeutic agents can cause life-threatening events. Here, we systematically evaluated causative agents in multi-agent chemotherapy; we focused on the TCbHP regimen, a 4-drug combination for which no associated colitis cases have previously been reported to our knowledge. Rapid identification of the most likely culprit agent(s) can facilitate early diagnosis, timely treatment, and planning of subsequent antitumor therapy.

Case Report

A 53-year-old woman was admitted to the hospital in early February 2023 because of a left breast mass discovered 3 months earlier. She had no history of colitis or colonic ulcers. Breast ultrasound revealed a solid hypoechoic lesion (28 × 13 × 21 mm) at the 1 to 2 o'clock position of the left breast, classified as BI-RADS 4C. Breast magnetic resonance imaging identified a nodular lesion (21 × 23 × 23 mm) at the same location, suggestive of malignancy (BI-RADS 5). No distant metastases were detected on contrast-enhanced computed tomography (CT) of the chest, abdomen, and pelvis, brain magnetic resonance imaging, or whole-body bone scintigraphy. Core needle biopsy confirmed high-grade ductal carcinoma in situ of the left breast with suspected focal invasion.

Six days after admission, the patient underwent a left total mastectomy and sentinel lymph node biopsy under general anesthesia. Postoperative pathology confirmed grade III invasive ductal carcinoma of the left breast, without metastasis in the sentinel lymph nodes (0/8). Immunohistochemistry showed estrogen receptor (ER [-]), progesterone receptor (PR [-]), Ki-67 (25%), and human epidermal growth factor receptor 2 (HER2 [3+]). The tumor was staged as pT2N0M0 (Stage IIA). In early March 2023, the patient received the first cycle of adjuvant chemotherapy with the TCbHP regimen. One week later, she developed severe abdominal pain and diarrhea. Laboratory testing showed a neutrophil count of 0.87 × 10^9/L. She remained afebrile, with a maximum temperature of 37.1 °C. Abdominal CT showed multiple mesenteric lymph nodes (some enlarged) at the root of the mesentery, with mild blurring of the surrounding fat planes. No obvious abnormalities were identified in other abdominal organs.

Colonoscopy revealed diffuse mucosal erythema, edema, and scattered erosions in the ascending, transverse, descending, and sigmoid colon (**Figure 1**). Symptomatic treatment was initiated upon diagnosis of colitis. The patient's symptoms improved, and she was discharged after recovery. Following

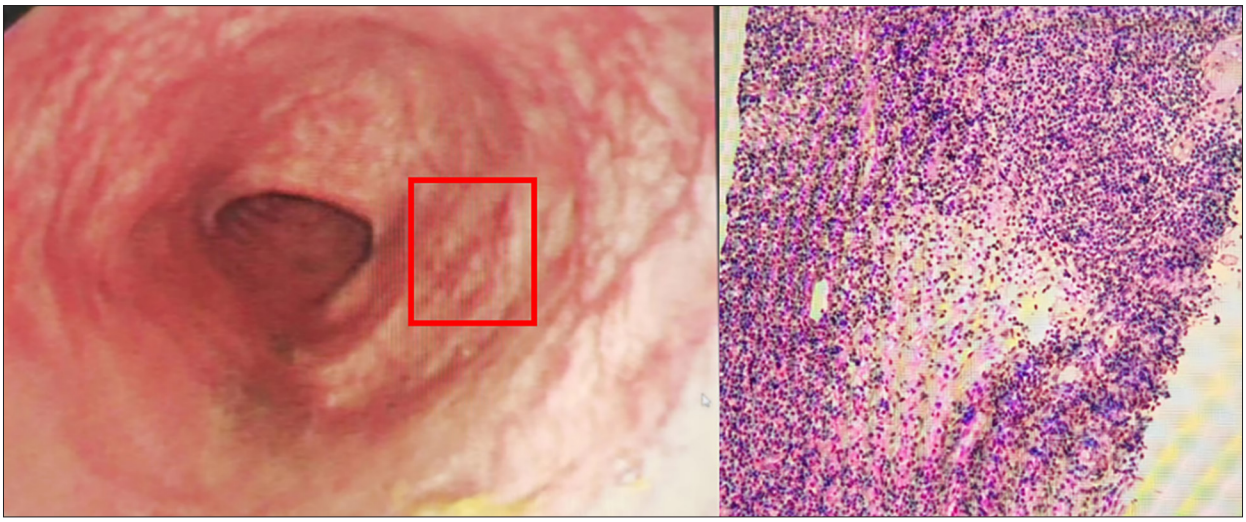

Figure 1. Colonoscopic and histopathological findings after the first cycle of chemotherapy.

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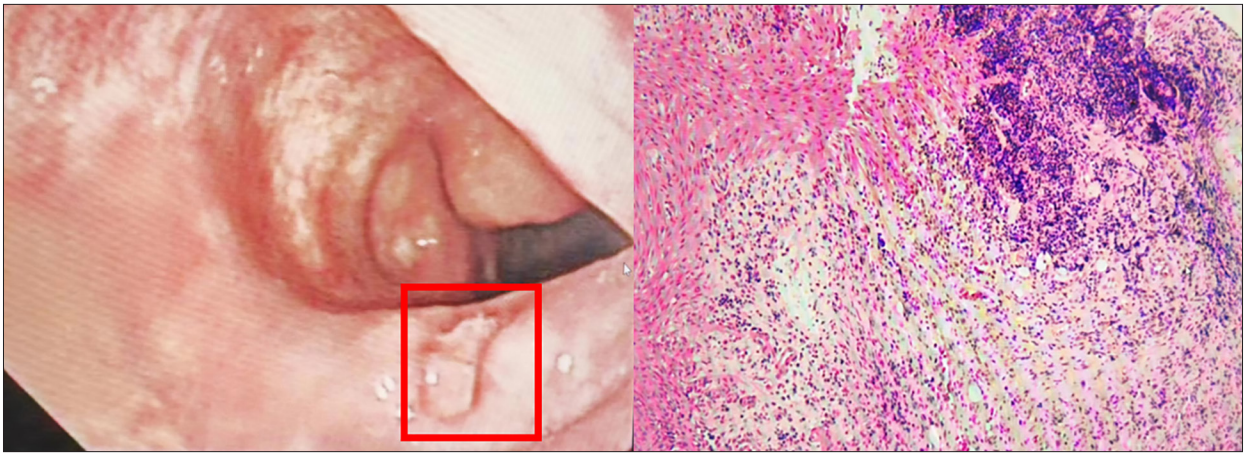

Figure 2. Colonoscopic and histopathological findings after the second cycle of chemotherapy.

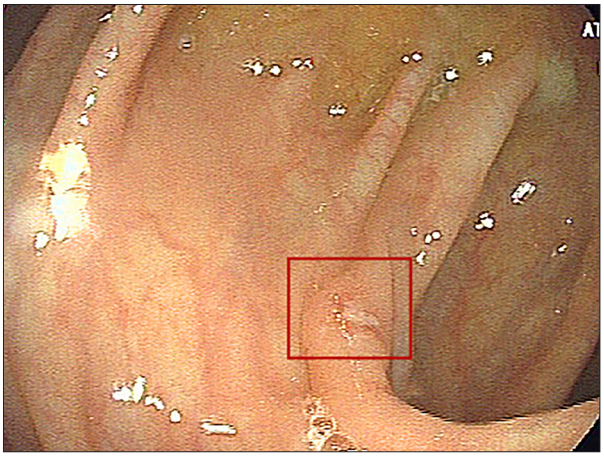

Figure 3. Follow-up colonoscopy 3 months after completion of chemotherapy.

multidisciplinary consultation, the TCbHP regimen was continued at 80% of the standard dose for the second cycle, which was administered in late March 2023 [4]. This decision reflected a careful risk-benefit assessment. Concerning benefits, the patient's HER2-positive (3+) status and Ki-67 index (25%) indicated a high risk of recurrence, supporting aggressive adjuvant therapy. Concerning risks, the colitis had resolved with supportive care and colonoscopy showed no deep ulcers or perforation; dose reduction of docetaxel and carboplatin was expected to reduce gastrointestinal toxicity. Thus, cautious dose reduction was favored over discontinuation of the regimen.

However, severe abdominal pain and diarrhea recurred 1 week after treatment. Laboratory testing showed a neutrophil count of 0.35×10^9/L. Repeat colonoscopy identified a 0.6 × 1.2 cm longitudinal ulcer with fibrinous exudate in the ascending colon (**Figure 2**). Histopathology confirmed chronic active colitis with crypt abscesses, inflammatory granulation tissue, and ulceration. Stool studies, including culture and *Clostridioides difficile* toxin assays, revealed negative findings.

Due to persistent colitis after the second cycle, a structured clinical decision was made to switch to THP (weekly nab-paclitaxel, trastuzumab, and pertuzumab). The key clinical question was whether anti-HER2 therapy could be continued while replacing the suspected causative chemotherapeutic agents. This decision was based on 4 considerations: (1) the temporal relationship implicated docetaxel and/or carboplatin; (2) no cases of colitis associated with trastuzumab or pertuzumab had been reported; (3) the National Comprehensive Cancer Network Guidelines list THP as an alternative regimen; and (4) nab-paclitaxel is an alternative taxane. Accordingly, TCbHP was replaced with THP while anti-HER2 therapy was continued.

The patient completed 6 cycles without further gastrointestinal complications. Follow-up colonoscopy in August 2023 demonstrated ulcer healing (**Figure 3**). By March 2024, she had completed 17 cycles of targeted therapy, and comprehensive re-evaluation in April 2025 showed no evidence of tumor recurrence.

Discussion

Alternative causes of colitis were systematically excluded. The patient had no prior history suggestive of inflammatory bowel disease. Cytomegalovirus (CMV) tissue testing (immunohistochemistry or polymerase chain reaction) was not performed on colonic biopsy specimens, which represents a limitation of the present case. Although the rapid clinical improvement without antiviral therapy made active CMV colitis less likely, CMV infection could not be definitively excluded. CT imaging showed no intestinal wall thickening. Histopathological findings demonstrated inflammation throughout the colon, rather than localization to the cecum. The patient had moderate neutropenia (absolute neutrophil count 0.87×10^9/L, Grade 2) at symptom onset. Neutropenic enterocolitis typically occurs in the context of profound neutropenia (Grade 4, absolute neutrophil count $< 0.5 \times 10^9$/L) and fever. Although colonic

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Table 1. Diagnostic criteria and clinical decision-making for suspected chemotherapy-associated colitis.

Step	Component	Key points
1	**Clinical suspicion**	Abdominal pain, diarrhea, with or without fever and neutropenia after recent chemotherapy
2	**Exclusion of alternative causes**	Negative stool cultures, negative *Clostridioides difficile* toxin assay findings, and exclusion of inflammatory bowel disease and cytomegalovirus infection (with tissue testing if indicated)
3	**Diagnostic evaluation**	Computed tomography imaging (bowel wall thickening, fat stranding); colonoscopy (mucosal erythema, erosions, ulceration); biopsy (active inflammation and exclusion of chronic changes)
4	**Causality assessment**	Temporal relationship, rechallenge (if applicable), response to drug withdrawal, and Naranjo score (interpreted cautiously in multi-agent regimens)
5	**Management decision**	Grade 1-2: symptomatic treatment with or without dose reduction; Grade 3-4: withhold suspected agent and switch to an alternative regimen if feasible

involvement alone does not exclude neutropenic enterocolitis, the absence of these features—combined with the lack of a requirement for broad-spectrum antibiotics and the absence of transmural necrosis—made neutropenic enterocolitis less likely, although it could not be definitively ruled out. Colonic biopsy findings were consistent with active colitis but showed no evidence of chronic architectural changes, such as crypt distortion or basal plasmacytosis, that are characteristic of inflammatory bowel disease. Furthermore, there were no histological features suggestive of ischemic or pseudomembranous colitis. However, histopathological findings of acute colitis without chronic changes are nonspecific and do not independently confirm a drug-related etiology. Such findings should be interpreted only as supportive evidence in conjunction with the temporal relationship, exclusion of alternative causes, and clinical response to treatment modification.

The diagnosis of chemotherapy-associated colitis in patients with breast cancer is based on a history of antineoplastic drug exposure and the presence of typical symptoms, including abdominal pain and diarrhea. Supporting findings can include severe neutropenia on laboratory testing; symmetric bowel wall thickening, intraluminal fluid, or pericolic inflammation on abdominal ultrasound or CT; and free intraperitoneal air in cases of perforation. Colonoscopy may demonstrate mucosal erythema, edema, scattered erosions, or ulceration. Mesenteric artery thrombosis may also be identified on angiography [5] (**Table 1**). Chemotherapy-induced colitis is rare. Cytotoxic agents that have been associated with colitis include cytarabine, vincristine, erythromycin, paclitaxel, docetaxel, 5-fluorouracil, vinorelbine, carboplatin, and cisplatin. To date, no cases of colitis attributable to trastuzumab or pertuzumab have been reported.

Several mechanisms have been proposed for taxane-associated colitis. Available evidence suggests that chemotherapeutic agents can induce vascular endothelial injury and small-vessel vasospasm. Platinum-based drugs may further promote microvascular thrombosis; colitis can result from the combined effects of these processes [6,7]. Additionally, taxanes cause mitotic arrest and apoptosis in gastrointestinal mucosal cells, and docetaxel has been shown to disrupt intestinal barrier function [8,9]. Other mechanisms, including microbiome disruption and immune-mediated inflammation, have been proposed; however, there is a lack of direct evidence to support these pathways in taxane-associated colitis, and further investigation is required [10]. The adverse effects of individual agents in the TCbHP regimen may also provide insight into the etiology of colitis. Docetaxel primarily causes myelosuppression, alopecia, gastrointestinal adverse effects (nausea, vomiting, and diarrhea), fluid retention, and hypersensitivity reactions. Severe complications include heart failure and rare cases of neutropenic enterocolitis with lower gastrointestinal bleeding [11]. Carboplatin commonly causes myelosuppression, gastrointestinal disturbances (nausea, vomiting, and diarrhea), hepatorenal toxicity, hypersensitivity reactions, electrolyte abnormalities, and alopecia. Platinum-associated colitis has been reported only rarely [12]. We used the Naranjo algorithm to assess the relationship among docetaxel, carboplatin, and colitis. The total score was 5, indicating a probable association [13]. However, the Naranjo scale has well-recognized limitations in the context of combination chemotherapy. It places substantial weight on clinical improvement after drug discontinuation, which may be confounded by the simultaneous withdrawal of multiple agents and the use of supportive care. Therefore, the score should be interpreted as supportive rather than definitive evidence. We acknowledge that the clinical improvement observed after switching

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to THP could have resulted from reduced myelosuppression, spontaneous resolution of colitis, or supportive care rather than the removal of a specific causative agent. Although systematic evaluation of potential causative agents is a standard approach in adverse drug reaction assessment and is not inherently novel, its application to the TCbHP regimen has not previously been described. The present case provides a practical example of identifying docetaxel and/or carboplatin as the most likely causative agents, thereby allowing continuation of anti-HER2 therapy through a switch to THP. The therapeutic challenges associated with chemotherapy-induced colitis may arise from several factors, including prolonged drug persistence in the body, impaired DNA synthesis leading to delayed mucosal regeneration and repair, increased susceptibility to secondary infection due to slow mucosal healing, and vascular endothelial injury resulting in coagulopathy, platelet aggregation, and fibrin thrombosis [7]. Current management strategies for confirmed colitis include symptomatic treatment and surgical intervention. Mesalazine may be used to promote ulcer healing and modulate the gut microbiota; colectomy may be required in cases of progressive colitis complicated by necrosis [14].

Adjustments to chemotherapy regimens should be individualized according to the severity and recurrence of colitis. For first-onset colitis, reducing the dose to at least 80% of the original regimen may be considered. If toxicity persists despite 2 dose reductions or after a treatment delay of less than 2 weeks, discontinuation of the regimen and substitution with alternative agents should be considered (eg, replacing docetaxel/carboplatin with weekly nab-paclitaxel, as in the present case) [15]. Compared with the largest cohort study of taxane-associated colitis by Chen et al [16], our case has several distinctive features. First, the patient received a 4-drug TCbHP regimen, whereas most previously reported cases involved taxane monotherapy or doublet regimens. Second, unlike the series reported by Carrion et al [17], in which a patient required surgery for colonic perforation, our patient was successfully managed with conservative treatment and regimen modification, resulting in complete resolution of colitis. Third, no cases of colitis attributable to trastuzumab or pertuzumab have been reported, suggesting that docetaxel and/or carboplatin were the most likely causative agents. Finally, after switching to THP, the patient completed 6 cycles of chemotherapy and 17 cycles of targeted therapy without recurrence of colitis, demonstrating that anti-HER2 therapy can be safely continued after removal of the suspected causative agents.

Conclusions

Colitis associated with breast cancer chemotherapy is rare; it presents diagnostic and therapeutic challenges. Severe complications, including colonic necrosis, may develop if the condition is not recognized and managed promptly. In the present case, timely intervention and regimen modification before the development of intestinal necrosis appeared to prevent serious sequelae, including intestinal resection. However, given the limitations of a single case report and the use of combination chemotherapy, a definitive causal relationship cannot be established.

Acknowledgments

We acknowledge the staff involved in the treatment of this patient.

Patient Consent

The patient provided written informed consent for publication of this report.

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Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

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