

**Proximity of backwash ileitis in colon cancer linked to ulcerative colitis**

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**Introduction**

The rectum is always affected by ulcerative colitis (UC), a chronic inflammatory disease that frequently spreads proximally over time. Although backwash ileitis (BWI) is the term for the additional spread of the inflammatory process into the terminal ileum, ulcerative colitis usually does not affect other parts of the gastrointestinal tract. In patients with UC, the incidence of BWI varied from 6% to 20%. A distinct subset of UC patients, backwash ileitis is known to advance to the fulminant stage, be younger, be resistant to treatment, and necessitate early surgery.

According to recent findings, individuals with BWI may have a higher risk of colorectal cancer related with UC, often known as colon cancer. However, nothing is known about the underlying pathomechanisms of BWI, and the condition's clinical trajectory is still unknown. The current UC case developed colitic carcinoma and exhibited proximal extension of BWI on endoscopy.

**Case Report**

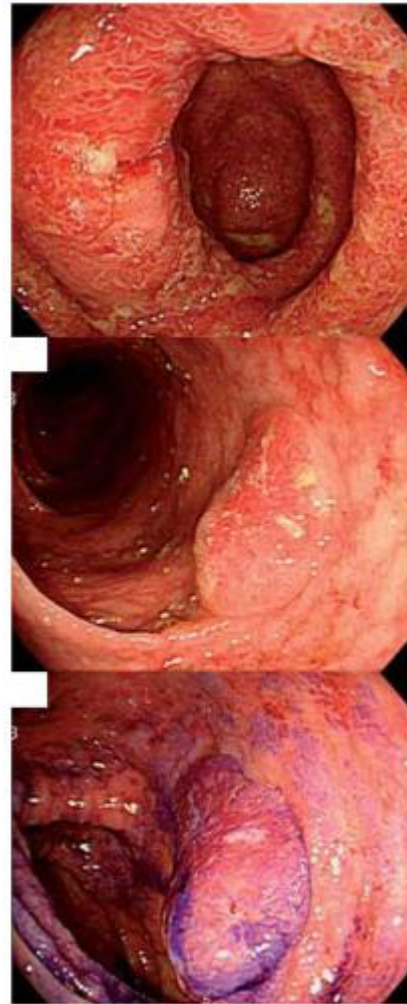
Four months prior to admission, a 67-year-old man with hematochezia had a colonoscopy at a nearby facility.

Friable, edematous, uneven mucosa without normal-looking intervening mucosa was observed throughout the whole colon, extending from the rectum, during a colonoscopy. At that point, the terminal ileum was not affected. Colorectal biopsy specimens without other pathognomonic features, such as granulomas, inclusion bodies, or vasculitic lesions, revealed diffuse mucosal infiltration of inflammatory cells, distorted atrophic crypts, goblet cell depletion, and crypt abscess on histology. The haustra coli disappeared after a barium enema, and the colon that was afflicted had an uneven surface and spiculation. An upper gastrointestinal endoscopy and barium examination of the small intestine produced normal results. The patient was prescribed oral prednisolone and mesalazine for his moderately active widespread UC diagnosis; nonetheless, with time, his symptoms worsened. The patient's anemia and ongoing bloody diarrhea led to their admission. His personal experiences and family background did not play a role. Other than a minor palpebral conjunctival pallor, there were no abnormalities found during the physical

examination. The results of the laboratory tests revealed the following: leukocyte count, 5300/mm<sup>3</sup> (normal, 3500–9100/mm<sup>3</sup>); platelets, 20.6×10<sup>4</sup>/mm<sup>3</sup> (normal, 13.0–36.9×10<sup>4</sup>/mm<sup>3</sup>); hemoglobin, 9.8 g/dL (normal, 11.3–15.2g/dL); C-reactive protein, 1.51 mg/mL (normal, <0.3 mg/mL); serum total protein, 6.8 g/dL (normal, 6.5–8.2 g/dL); immunoglobulin G, 1840 mg/dL (normal, 870–1700 mg/dL); IgA, 272 mg/dL (normal, 110–410 mg/dL); IgM, 94 mg/dL (normal, 46–260 mg/dL); antinuclear antibodies, <40 (normal, <40); and C3, 113 mg/dL (normal, 86–160 mg/dL). Urinalysis findings were normal, and repeated screenings failed to find any infectious pathogens in stool specimens. There was no evidence of Clostridium difficile toxin in the feces.

A tiny amount of intestinal gas was visible on the abdominal plain radiograph; there was no free air, no air-fluid level, and no hazardous colon dilatation. The cecum and ascending colon were the area's most severely damaged by the mucosal granularity, mucous exudates, diffuse erosions, and leaking throughout the colonic segments that were shown by a colonoscopy upon admission. It also involved the opening of the appendix. Interestingly, chromo endoscopy revealed a flat, raised lesion in the significantly inflamed transverse colon. The results of magnified chromo endoscopy indicated nonstructural mucosa devoid of pits, destruction of pits, irregularity in pit size and distribution, and dilatation or swelling of the pits. These findings were suggestive of invasive cancer.

Tumor biopsies examined histopathologically revealed a well-differentiated adenocarcinoma. In addition, erythema, a continuous pattern of erosions, mucosal friability, absence of the vascular pattern, and a dilated patulous Bauhin's valve were all seen within 30 cm of the terminal ileum from the cecum.



Figure

Biopsies taken from the ileum in question revealed persistent, aggressive, non-specific inflammation in the lamina propria. Both the macroscopic and microscopic results supported BWI. Abdominal and pelvic contrast computed CT revealed extensive mural thickening of the affected intestine, but no intra-abdominal tumors or swelling of the lymph nodes in the area. The patient received oral prednisolone, commencing at 65mg per day, and mesalazine, 2250 mg per day. Levofloxacin, 300 mg per day, was administered for the first 10 days of the patient's treatment. In addition, the patient received leukocytapheresis therapy, which was carried out once a week for five weeks using a commercially available leukocyte removal column. These rigorous treatments,

meanwhile, were unable to manage the illness. These rigorous treatments, meanwhile, were unable to control the illness. Two months later, during a follow-up colonoscopy, it was discovered that BWI had progressed further, with significant erosions reaching 50 cm from the ileocecal valve on the oral side and more severe mucosal inflammation.

Proctocolectomy, ileostomy, and an urgent ileectomy with a margin of 10 cm from the afflicted ileum were performed on the patient. The surgical specimen's histopathology showed a well-differentiated adenocarcinoma with a mucinous component invading into the transverse colon's submucosa, pan colitis throughout the entire colon, and a marked BWI in continuity. It also showed homogenous mucosal infiltration of neutrophils and lymphoplasmacytes, resulting in villous blunting and surface fibrin purulent debris concurrently. Nongaseous epithelioid granulomas and mucous gland metaplasia, which are characteristics that are thought to promote a diagnosis of CD rather than BWI, were not present. His recovery from the surgery went smoothly, and five years after the procedure, at his last appointment at the outpatient clinic, he was still doing well.

### **Discussion**

BWI, or incompetence of the inflamed ilein retrograde flow of colonic contents into the distal ileum, is a common complication of persistent, severe colonic illness in ulcerative colitis (UC). Patients with BWI who have ulcerative colitis only ever experience chronic concurrent pancolitis. Pancolitis was found in 94% of individuals with ileal inflammatory alterations in a recent systematic investigation of the distal ileum in UC patients. This incidence was much greater than the rate of pancolitis (39%) in patients without ileal pathology. Once more, it has been noted that there is a correlation

between cecal UC and BWI activity level. In fact, BWI was discovered to be associated with active cecal colitis in this instance, where the cecum was the colon and rectum's most impacted area.

The majority of writers concur that BWI and UC share a morphologic pattern of mucosal inflammation and damage. The distal ileum of the current patient had erythema, a lack of vascular pattern, friability, and erosions without any normal-appearing mucosa in between, which resembled the intestinal lesion of UC. From a histopathological perspective, the ileal mucosa showed uniform neutrophilic and plasmalymphocytic infiltration along with villous blunting.

Aside from the impacted ileum, there were no other anomalies in the small intestine or upper gastrointestinal tract.

Furthermore, no pathognomonic signs were shown by either the microbiological or histopathologic investigations in this instance, making it easy to distinguish between UC-associated BWI and other potential inflammatory bowel disorders, such as Crohn's disease.

Uncertainty surrounds BWI's clinical trajectory. It has not been possible to determine the precise extent of BWI since the histopathologic investigation of BWI has only been performed on proctocolectomy specimens, and only a small portion of the terminal ileum is usually available [2]. The ileal involvement by UC ranged from 3 to 45 cm in autopsy investigations. To the best of our knowledge, however, this is the first example of illness extension that has been proven endoscopically that has been published in English literature. In the distal ileum, within 30 cm of the ileocecal valve, repeat colonoscopy performed upon admission revealed morphological characteristics compatible with BWI, despite the terminal ileum being intact on endoscopy four months prior to hospitalization.

Furthermore, in spite of vigorous UC therapy, it was discovered that the ileal lesion had spread about 20 centimeters closer to the original affected region in less than two months. Patients with UC are known to experience proximal extension of disease in the colorectum; it is crucial for clinicians to understand that BWI distributions can also stretch over time.

While discussing the causes of BWI is outside the purview of this paper, prior research indicates that bacterial overgrowth, drug interactions, and infections may be the source of ileal inflammation in UC patients. The current patient's ileal involvement increased with time in spite of vigorous treatment with prednisolone, mesalazine, and leukocytapheresis, which is consistent with the treatment-resistant nature of BWI. Patients with ulcerative colitis receiving steroid treatment are more vulnerable to infections and may experience medication toxicity, both of which can happen in the ileum. Although repeated testing in this instance did not find any infectious agents in the stool or biopsy specimens, nonculturable microbiota may be the cause of long-term immunological and inflammatory processes in the ileum. In this sense, information about the bacterial community unique to BWI pathogenesis may be obtained by nuclear-acid-based techniques for detection and enumeration.

### **Conclusion**

According to Heuschen and colleagues, there is a substantial correlation between BWI and colorectal cancer in UC. Compared to patients with pancolitis or left-sided colitis without BWI, cancer patients with pancolitis and BWI had noticeably higher numerous tumor growths. Previous investigations have proven the role of extended duration and breadth of UC as risk factors for colorectal cancer. Therefore, it is widely acknowledged that the persistent inflammation-dysplasia-carcinoma sequence in UC leads to the development of

so-called colitic cancer. The correlation between BWI and colon cancer, however, cannot be explained by this approach because BWI patients with UC are more likely to be younger and require earlier surgical intervention. Moreover, colonoscopy surveillance studies are needed to look into the prognostic usefulness of BWI for colorectal cancer and dysplasia in UC patients.

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