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【 CASE REPORT 】

Blind Pouch Syndrome-associated Anastomotic Ulcer Diagnosed with Capsule and Double-balloon Endoscopy

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Abstract:
Blind pouch syndrome-associated anastomotic ulcer is rare, and its endoscopic features remain poorly described. A 79-year-old man was referred to our hospital for melena. Capsule endoscopy revealed multiple ulcers in the small intestine. Double-balloon endoscopy (DBE) and a gastrografin examination through DBE revealed a potential anastomotic ulcer, a blind pouch, and a side-to-side anastomosis in the middle of the small intestine. Laparoscopic partial resection of the small intestine with anastomosis was performed on the suspected blind pouch syndrome-associated anastomotic ulcer. To our knowledge, this is the first report describing the endoscopic features of a blind pouch syndrome-associated anastomotic ulcer.

Key words: blind pouch syndrome, anastomotic ulcer, capsule endoscopy, double-balloon endoscopy

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Introduction

Blind pouch syndrome is defined as a series of symptoms, such as anemia, intermittent abdominal pain, diarrhea, and weight loss, all of which are associated with the formation of a blind pouch secondary to a side-to-side intestinal anastomosis (1). Blind pouch syndrome-associated anastomotic ulcer is rare, and its endoscopic features remain poorly described.

We herein report a case of a blind pouch syndrome-associated anastomotic ulcer diagnosed with capsule endoscopy (CE) and double-balloon endoscopy (DBE).

Case Report

A 79-year-old man was referred to our hospital for melena. Of note, he had a history of arteriosclerosis obliterans (ASO) and angina pectoris (AP) and had taken low-dose aspirin (LDA) and cilostazol for 14 years. In addition, he was confirmed to have undergone surgery for ileus 15 years earlier, although the details of the procedure were unknown. He had suffered a stroke three months ago and started receiving dabigatran at an increased dose. He was found to have severe anemia (Hb, 4.2 g/dL) due to melena and received a blood transfusion, and hemostasis was achieved with three-day fasting.

Esophagogastroduodenoscopy (EGD) findings were normal, and colonoscopy (CS) revealed small polyps and diverticulosis, which led to diverticular bleeding being suspected. After discharge, however, he was re-hospitalized 18 days later for a blood transfusion based on evidence of recurrent melena, and hemostasis was achieved with four-day fasting, with a blood clot confirmed in the terminal ileum on CS. Again, CE revealed multiple ulcers in the small intestine (Fig. 1), which led to LDA-induced small intestine ulcers being suspected. At this point, the patient was discharged with LDA replaced by clopidogrel and rebamipide initiated; however, he was re-hospitalized for a blood transfusion for recurrent melena 52 days later, with hemostasis being achieved again with two-day fasting. The patient underwent trans-oral and trans-anal DBE as well as a gastrografin enema examination through DBE, which revealed an anastomotic ulcer (Fig. 2A and B), a blind pouch (Fig. 2C and D),

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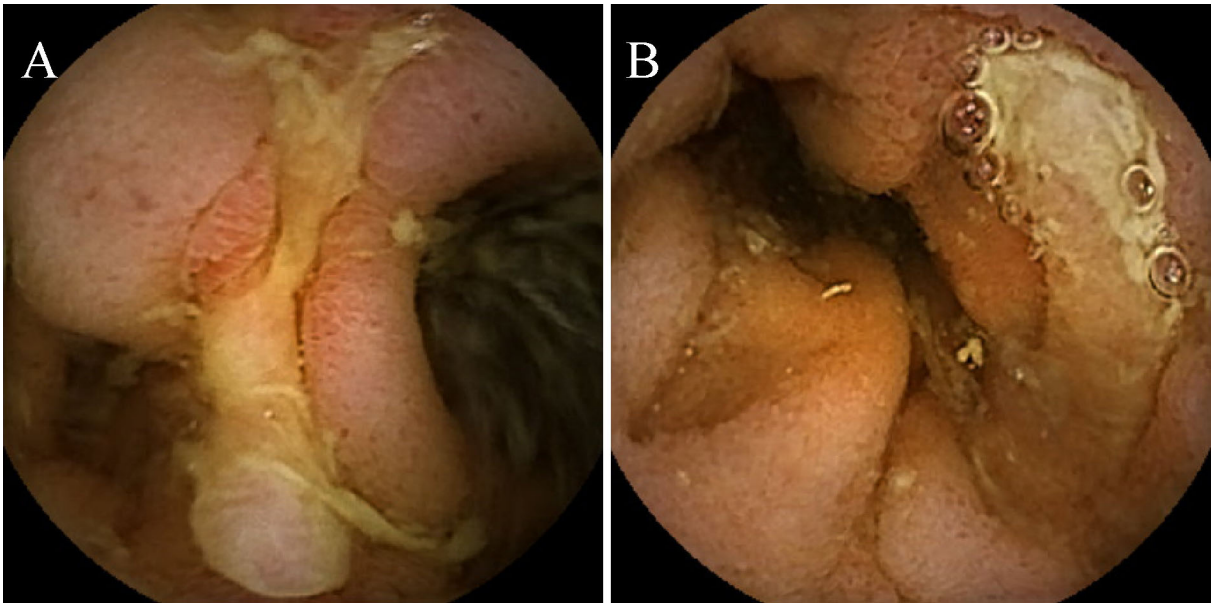

Figure 1. **Capsule endoscopy. Multiple ulcers were recognized in the small intestine (A, B).**

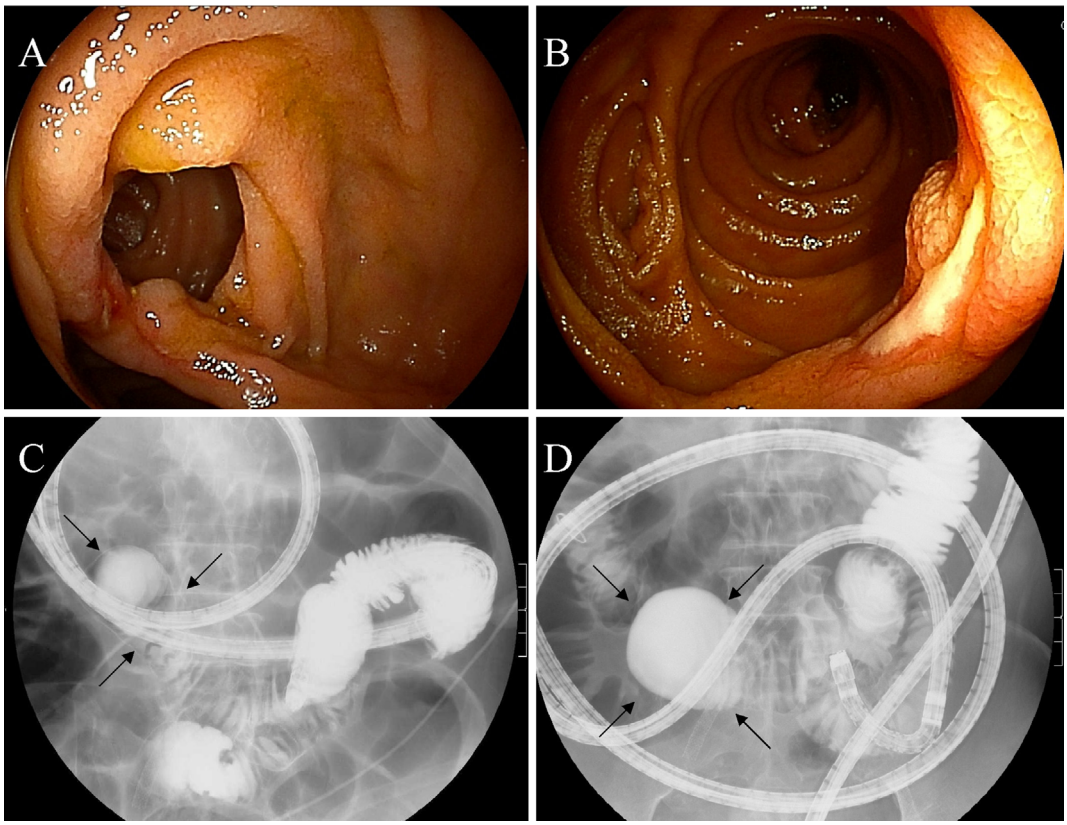

Figure 2. **Double-balloon endoscopy (DBE) and a gastrografin enema examination. Trans-oral and trans-anal DBE revealed anastomotic ulcer (A, B). A gastrografin enema examination through DBE revealed a blind pouch and a side-to-side anastomosis in the middle of the small intestine (C, D).**

and side-to-side anastomosis in the middle of the small intestine. The multiple ulcers detected with CE were retrospectively confirmed to be an anastomotic ulcer. Computed tomography (CT) revealed GI tract dilatation (axial; Fig. 3A) as well as a blind pouch with a side-to-side anastomosis (coronal; Fig. 3B).

Laparoscopic partial resection of the small intestine with anastomosis was performed on the suspected blind pouch syndrome-associated anastomotic ulcer eight days after DBE. The resected specimen revealed a blind pouch with

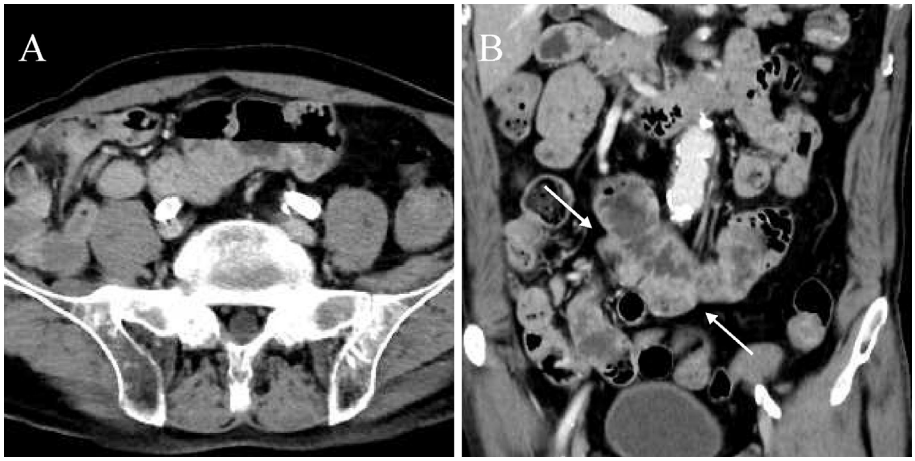

Figure 3. **A CT examination. (A) A dilated tract was recognized in the middle of the small intestine. (B) Coronal reformatted multidetector CT (MDCT) revealed a blind pouch and a side-to-side anastomosis.**

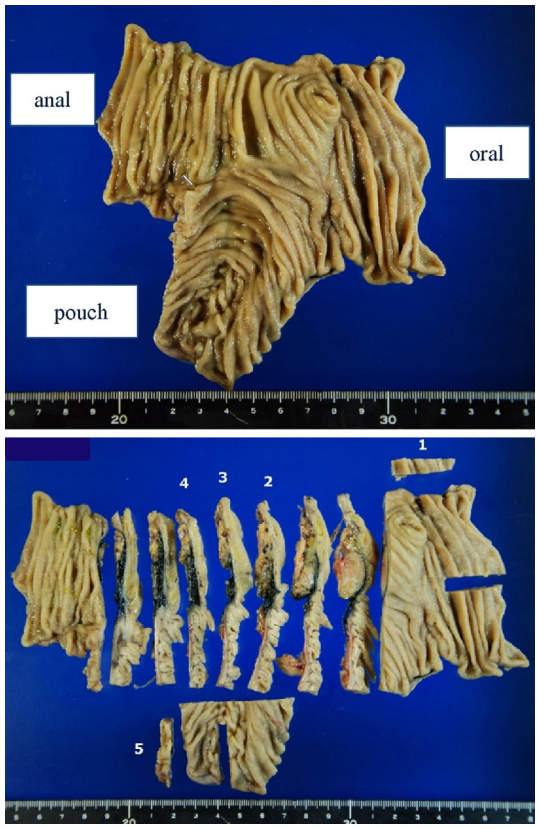

Figure 4. **Resected specimen. A blind pouch with side-to-side anastomosis was recognized, but no anastomotic ulcer was recognizable.**

side-to-side anastomosis but no anastomotic ulcer (Fig. 4). A histological examination showed anastomotic erosion but no ulcer (Fig. 5). LDA and dabigatran were restarted five and seven days after surgery, respectively. The patient's course was uneventful for six months after surgery.

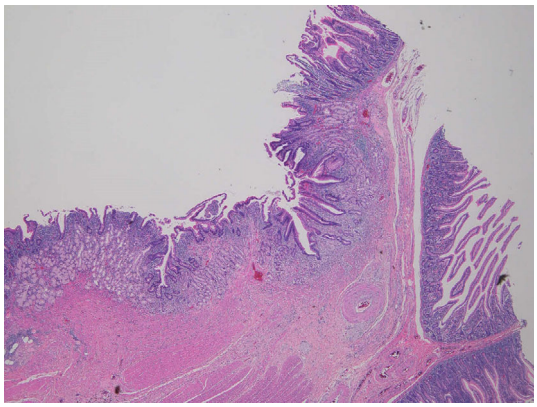

Figure 5. **A histopathologic examination of the resected specimen. Anastomotic erosion was shown to be present in section 3.**

Discussion

Our case has two important clinical implications. First, side-to-side anastomosis of the small intestine may result in severe melena by the formation over time of a blind pouch syndrome-associated anastomotic ulcer following surgery. However, the endoscopic features of blind pouch syndrome-associated anastomotic ulcer remain unknown with no reports available in the literature.

Blind pouch syndrome is defined as a series of symptoms, including anemia, intermittent abdominal pain, diarrhea, and weight loss, associated with the formation of a blind pouch secondary to side-to-side intestinal anastomosis (1). According to Cannon and Murphy (2), side-to-side intestinal anastomosis may interfere with normal peristalsis as a result of interruption of the circular muscle fibers. The enclosed food may accumulate in the blind end, since the two opposed loops do not act synchronously. Therefore, the gradually delated intestine may form a blind pouch. The overgrowth of

bacteria in a blind pouch induces mucosal inflammation, edema, ulceration, and even perforation of the intestine (3). To date, upper gastrointestinal series (1, 3, 4) and CT (5) have been reported as useful preoperative diagnostic methods. Recently, coronal reformatted multidetector CT (MDCT) has been shown to aid in detecting a blind pouch by revealing a dilated tract or surgical suture (6, 7). In the present case, CE and DBE clearly revealed an anastomotic ulcer, and a gastrografin enema examination through DBE and MDCT revealed a blind pouch with side-to-side anastomosis. Thus, an accurate diagnosis of the present case was achieved only by using multiple modalities.

The profile of melena has previously been reported in patients with blind pouch syndrome-associated anastomotic ulcer (1, 8-11), and the time from surgery to melena was reported to vary among reports: 3 times in 2-19 years and 15 years (1), 30 years (8), 3 times in 50-52 years and 13 years (9), 39 years (10), and 10 years (11). In the present case, severe melena occurred 15 years after surgery. To our knowledge, this is the first report describing blind pouch syndrome-associated anastomotic ulcer diagnosed with CE and DBE.

The second important clinical issue that emerged in our case was that hemostasis of melena due to blind pouch syndrome-associated anastomotic ulcer was achieved with a few days of fasting, and the anastomotic ulcer had healed by eight days after surgery. According to Miyoshi et al. (9), not only the overgrowth of bacteria but also stasis of the intestinal contents and mechanical irritation associated with peristalsis may be responsible for blind syndrome-associated anastomotic ulcer. Our case appears to support this idea, as the ulcer had healed after a few days of fasting. In the present case, melena occurred three months after increasing the dabigatran dose. In addition, given that blind pouch syndrome-associated ulcer was reported to be formed at the anastomosis or mucosa of the blind pouch (11), the present case was determined to be one of anastomotic ulcer. According to a recent review, dabigatran is reported to be associated with a risk of major gastrointestinal bleeding (12), especially in the presence of pre-existing lesions, such as angiodysplasias and erosion (13). Potential explanations include the following: its tartaric acid coating has a direct effect on the intestinal lumen (14); and the metabolism of dabigatran etexilate by esterases leads to progressively higher concentrations of the active drug during its passage through the gastrointestinal tract (15). Thus, dabigatran may have been the cause of bleeding from blind pouch syndrome-associated anastomotic ulcer in our case.

In conclusion, side-to-side anastomosis of the small intestine may cause severe melena by the formation of blind pouch syndrome-associated anastomotic ulcer over a long period of time after surgery. CE and DBE appear to be promising modalities for diagnosing a blind pouch syndrome-associated anastomotic ulcer.

The authors state that they have no Conflict of Interest (COI).

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