

Original Research Article

Risk Factors for Stoma Outlet Obstruction after Proctocolectomy for Ulcerative Colitis

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Abstract

Objectives: Stoma outlet obstruction (SOO) occurs with an incidence of approximately 40% after proctocolectomy for Ulcerative colitis (UC) with diverting ileostomy.

This study aimed to identify the risk factors for SOO after proctocolectomy with diverting ileostomy for patients with UC.

Methods: We reviewed the data of 68 patients with UC who underwent proctocolectomy and diverting ileostomy between April 2006 and September 2021. These cases were analyzed on the basis of clinicopathological and anatomical factors. SOO was defined as small bowel obstruction displaying symptoms of intestinal obstruction, such as abdominal distention, abdominal pain, insertion of a tube through the stoma.

Results: The study included 38 (56%) men and 30 (44%) women with a median age of 42 years (range, 21-80). SOO categorized as at least Clavien-Dindo grade II occurred in 11 (16%) patients. Six patients required earlier stoma closure than scheduled. Compared with patients without SOO, patients with SOO had a significantly higher total steroid dose from the onset of UC to surgery ($p = 0.02$), a small amount of intraabdominal fat ($p = 0.04$), and a higher rate of laparoscopic surgery ($p < 0.01$).

Conclusions: A high preoperative steroid dose, a small amount of intraabdominal fat and laparoscopic surgery were identified as risk factors for SOO. Early detection and treatment for SOO are important for patients at risk.

Keywords

stoma outlet obstruction, ulcerative colitis, risk factor, diverting ileostomy, postoperative complication

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Introduction

Ulcerative colitis (UC) is a chronic inflammatory disease of the intestinal tract characterized by repeated relapse and remission. The number of patients with UC in Japan has been increasing annually[1]. Although advances in medicine are increasing the number of patients who can avoid surgery, they are also increasing the number of patients undergoing

emergency surgery due to second- and third-line treatment failures[2].

The standard surgical technique for UC is proctocolectomy with ileal pouch-anal anastomosis (IPAA) or ileal pouch-anal canal anastomosis (IACA)[3]. A diverting ileostomy is created to avoid the risk of anastomotic leakage. However, this sometimes leads to stoma-associated complications. Stoma outlet obstruction (SOO) is often ob-

served after surgery.

SOO occurs with an incidence of approximately 40% after IPAA with diverting ileostomy, making it a critical complication to monitor[4]. SOO is often indicated by ileus and the absence of fecal discharge. Patients develop bloating, nausea, and vomiting. In most cases, vacuum tubing is effective against obstruction of the oral side of the entry hole[5,6]. However, if it cannot be treated conservatively, the stoma should be closed. However, if anastomotic leakage or ileal pouch-related complications occur, stoma closure cannot be performed. Therefore, it is important to prevent SOO.

Our study aimed to identify the risk factors for SOO after proctocolectomy with diverting ileostomy for patients with UC.

Methods

Patients

We retrospectively reviewed the data of patients with UC who underwent proctocolectomy and diverting ileostomy between April 2006 and September 2021.

Definition of SOO and high-output stoma

SOO was defined as small bowel obstruction with symptoms of intestinal obstruction, such as abdominal pain, abdominal distention, or vomiting, and computed tomography (CT) revealing caliber changes near the ileostomy or recovery following the insertion of a tube through the stoma.

High-output stoma (HOS) was defined as stoma drainage of 2000 ml/day.

Signed informed consent form was obtained from all patients before enrollment to this study.

Surgical procedure

IACA were performed for refractory or severe UC. IPAA were performed in cases with severe inflammation around the anal canal. All patients had their skin marked at the appropriate stoma site by wound ostomy care nurses preoperatively. The rectus abdominal muscle was split, and the peritoneum was broadened to at least two fingerbreadths. The segment of ileum to be lifted was selected 30-50 cm perorally from the anastomosis.

Variables

We compared between the patients who developed SOO (SOO group) or those who did not (non-SOO group). We noted patients' characteristics (age, gender, body mass index (BMI), the surgical indication, and the total steroid dose from the onset of UC to surgery, surgical indications, and extent of colitis), perioperative data (surgical procedures, type of anastomosis, and complications), and anatomical

factors such as the visceral thickness, depth of the abdominal cavity, vertical fat area (VFA), and distance between the root of their superior mesenteric artery and the bottom of the external anal sphincter (rSMA-bEAS), were previously reported as risk factors for SOO[7]. We retrospectively calculated these variables using the axial and sagittal views of preoperative CT images. The use of clinical data was approved by the Human Ethics Review Board of Dokkyo Medical University (Protocol #R-27-9J).

Statistical analysis

All statistical analyses were performed using EZR (Saitama (The R Foundation for Statistical Computing, Vienna, Austria)[8]. Significance was evaluated using Student's t-test, analysis of variance, the Mann-Whitney U test, and the chi-squared test as appropriate. Binary logistic regression was used to assess univariate and multivariate associations between factors. The optimal cutoff was selected according to receiver operating curve analysis. Differences were defined significant at $p < 0.05$.

Results

Between April 2006 and September 2021, 74 patients underwent surgery for UC. Of these, 68 patients were included in this study after excluding the patients who did not undergo diverting ileostomy. The main clinical characteristics are presented in Table 1. The study included 38 (55.9%) men and 30 (44.1%) women with a median age of 42 years (range, 21-80). The median disease duration was 5 years (range, 1-26).

SOO categorized as at least Clavien-Dindo grade II occurred in 11 (16.2%) patients. The median timing of SOO was postoperative day 2 (range, 1-7). All patients underwent insertion of a decompression tube, and their obstructive symptoms conservatively resolved within 3 days. Six patients required earlier stoma closure than scheduled. Compared with patients without SOO, patients with SOO had a significantly higher total steroid dose from the onset of UC to surgery ($p = 0.01$) and a higher rate of laparoscopic surgery ($p = 0.001$). SOO was significantly associated with HOS ($p = 0.04$, Table 2).

No significant difference was observed in terms of age, gender, BMI, blood loss, or operative time between the groups.

VFA $< 74 \text{ cm}^2$ and rSMA-bEAS (adjusted according to the patient's height) $> 174 \text{ mm/m}$ were significantly associated with a higher incidence of SOO ($p = 0.04$, $p = 0.03$) (Table 3). No significant difference was observed regarding the thickness of the abdominal wall and depth of the abdominal cavity between the two groups.

In logistic regression analysis for relevant variables, laparoscopic surgery, preoperative steroid dose $> 10,000 \text{ mg}$,

Table 1. Patient Characteristics.

Factor			
Age (y)		46.0	(±18.6)
Gender	Male	38	56%
	Female	30	44%
Disease duration (y)		9.3	(±8.2)
Extent of colitis	Pancolitis	59	87%
	Left-sided colitis	9	13%
Surgical indication	Cancer/Dysplasia	19	28%
	Severe/Fulminant	26	38%
	Refractory	23	34%
Body mass index (kg/m ²)		19.9	±3.5
Albumin (g/dl)		2.9	±1.0
Total lymphocyte count		1410	±695
Onodera prognostic index		35.9	±11.3
Preoperative medication	Steroid (>10000 mg)	28	41%
	Anti-TNFα	24	35%
	Immunosuppressant	38	56%
	Amino salicylate	57	84%
Extent of colitis	Pancolitis	59	87%
	Left-sided colitis	9	13%
Surgical indication	Cancer/Dysplasia	19	28%
	Severe/Fulminant	26	38%
	Refractory	23	34%

Table 2. Patient Characteristics and Treatment Outcomes in the Compared Groups.

		SOO (n=11)	Non-SOO (n=57)	p-value
Age (year)		43.8 (±16.8)	46.4 (±18.6)	0.30
Gender	Male	6 (54.5%)	32 (56.1%)	0.90
	Female	5 (45.5%)	25 (43.9%)	
Disease duration (y)		10 (1-32)	7 (1-30)	0.20
Preoperative medication	Steroid (>10000 mg)	8 (72.7%)	20 (35.1%)	0.02
	Anti-TNFα	5 (45.4%)	19 (33.3%)	0.44
	Immunosuppressant	6 (54.5%)	32 (56.1%)	0.92
	Amino salicylate	10 (90.9%)	47 (82.5%)	0.49
Extent of colitis	Pancolitis	9 (81.8%)	50 (87.7%)	0.60
	Left-sided colitis	2 (18.2%)	7 (12.3%)	
Surgical indication	Cancer/Dysplasia	4 (36.4%)	15 (26.3%)	0.77
	Severe/Fulminant	4 (36.4%)	22 (38.6%)	
	Refractory	3 (27.3%)	20 (35.1%)	
Surgical approach	Laparoscopy	10 (90.9%)	21 (36.8%)	<0.01
	Open	1 (9.1%)	36 (63.2%)	
Anastomosis	IPAA*	7 (63.6%)	33 (57.8%)	0.83
	IACA*	4 (36.3%)	24 (42.1%)	
Strategy	2 stage	7 (63.6%)	22 (38.6%)	0.20
	3 stage	4 (36.3%)	35 (71.4%)	
Complication	SSI*	1 (9.1%)	5 (9.0%)	0.72
	Ileus	1 (9.1%)	4 (7.0%)	
	HOS*	4 (36.3%)	6 (10.5%)	

* SSI: surgical site infection, HOS: high-output stoma, IPAA: ileal-pouch anal anastomosis, IACA: ileal-pouch anal canal anastomosis

Table 3. Univariate Analysis of Anatomical Risk Factors Associated with Stoma Outlet Obstruction.

	SOO (n=11)	Non-SOO (n=57)	p-value
Thickness of abdominal wall (mm)	23.5 (±7.7)	23.0 (±10.3)	0.44
Depth of the abdominal cavity (mm)	43.2 (±15.8)	46.6 (±18.8)	0.70
Adjusted rSMA-bEAS (mm/m)	165.6 (±23.9)	173.9 (±14.8)	0.03
Vertical fat area (VFA) (cm ²)	35.2 (±22.2)	59.9 (±49.5)	0.04

※Adjusted rSMA-bEAS: (rSMA-bEAS) / Height

Table 4. Multivariate Analysis of Risk Factors Associated with Stoma Outlet Obstruction.

	Odds ratio	95%CI		P-Value
		Lower	Upper	
Laparoscopy	7.33	1.51	35.33	0.02
HOS	1.64	0.12	20.41	0.26
Steroid	5.97	1.13	31.92	0.02
Vertical fat area (>74.0 cm ²)	0.55	0.16	1.99	0.92
Adjusted rSMA-bEAS (>174.0 mm/m)	0.04	0.002	0.41	0.04

and adjusted rSMA-bEAS > 174.0 mm/m² were identified as independent risk factors for SOO (Table 4).

Discussion

To date, three factors have been reported as etiologies of SOO: surgical technique, anatomical factors, and physiological factors. Surgical causes include laparoscopic surgery[9] and ileostomy procedures[10], whereas the anatomical factors associated with SOO include the abdominal wall thickness, mesenteric length, and VFA. SOO can also be caused by HOS and oral intestinal edema.

Previous studies identified subcutaneous fat and the thickness of the rectus abdominal muscle are risk factor for SOO[11,12]. These studies reported three important factors related to the development of SOO after the construction of a diverting ileostomy during laparoscopic colorectal surgery: a thick rectus abdominal muscle, a high abdominal wall thickness/abdominal rectus muscle thickness ratio, and an ileostomy inclined to the medial side[13]. Okita et al showed that low BMI (<21 kg/m²) and younger age (<16 years) at the time of surgery might be predictive factor for SOO after construction of a diverting ileostomy in patients with UC[14]. When the condition of the ileostomy was evaluated using postoperative abdominal CT, it was found that the angle of the stoma outlet lumen was tilted inward in SOO patients. In this study, a low amount of visceral fat was a risk factor. The relative angle of the abdominal wall and stoma caused by a low amount of vertical fat might lead to SOO.

Our study revealed that SOO was independently associ-

ated with adjusted rSMA-bEAS > 174.0 mm/m by multivariate analysis. It was suggested that SMA is the defining factor of mesenteric tension, mesenteric length both makes anastomosis more difficult and increases the risk of developing SOO.

After IPAA with redirection ileostomy, fixation of the mesentery by excessive tension and free movement of the small intestine causes twisting of the ileum, making it difficult to reposition[7].

In addition, our study indicated low-VFA as a risk factor for SOO. It was suggested less vertical fat increases the degree of freedom of the intestinal tract in the abdominal cavity and increases the possibility of twisting the intestinal tract. However, there have not been reported relationship between VFA and SOO. Further investigation is required to understand the effect of a difference in VFA on the incidence of a SOO.

Laparoscopic surgery has been previously reported as a risk factor for SOO, which has been hypothesized to result from the aforementioned technical difficulties during ileostomy[15-17].

This is caused by the fact that the small intestine can more easily twist because of the increased movable space after total proctocolectomy and decreased postoperative adhesion after laparoscopic surgery. A previous report noted that a high dose of steroids may reduce the risk of adhesions[18]. The current study showed that a high dose of steroids was an independent risk factor for SOO. Our study corroborated these results.

As a technical issue, when constructing an ileostomy, it is important to ensure that the stoma does not bend or exhibit

stenosis at the site where the abdominal wall is penetrated to prevent SOO. The oral side is often constructed at the 6 o'clock position to prevent the inflow of stool into the anal side and to facilitate self-care. However, because the oral limb tends to bend at the site where it transitions from the stoma tunnel to the abdominal cavity, some reports recommend that the oral limb was oriented to the 12 o'clock position[19,20].

Twisting of the mesentery is greater in loop ileostomy than in end ileostomy. Therefore, it is important to ensure the correct positioning of the ileum when elevating the ileum[21].

Our study had a few limitations. These results are based on a single-center, retrospective study with a small sample size. Prospective studies with large numbers of patients are needed to assess the true risk factors for SOO and determine appropriate preventive measures.

In conclusion, it is necessary to devise strategies to prevent twisting of the stoma (e.g., position and direction of stoma construction) for patients with risk factors (laparoscopic surgery, short mesentery, low VFA). In addition, adjusting the steroid dosage before surgery and lifting the intestine perpendicular to the rectus abdominis muscle could also be effective. Because of the high incidence of SOO, early detection and treatment are critical for patients at risk.

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Conflicts of Interest

There are no conflicts of interest.

Author Contributions

Keisuke Ihara: Conceptualization; Funding acquisition; Writing - review & editing.

Yusuke Nishi, Junki Fujita, Masashi Takayanagi and Yasunori Maeda: Formal analysis; Methodology; Visualization; Writing - review & editing.

Norisuke Shibuya, Hiroyuki Hachiya and Mitsuru Ishizuka: Conceptualization; Methodology; Writing - review & editing.

Takatoshi Nakamura: Visualization; Writing - original draft.

Kazuyuki Kojima, Keiichi Tominaga and Atsushi Irisawa: Supervision; Writing - review & editing.

Approval by Institutional Review Board (IRB)

We use of clinical data was approved by the Human Ethics Review Board of Dokkyo Medical University (Protocol #R-27-9J).

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