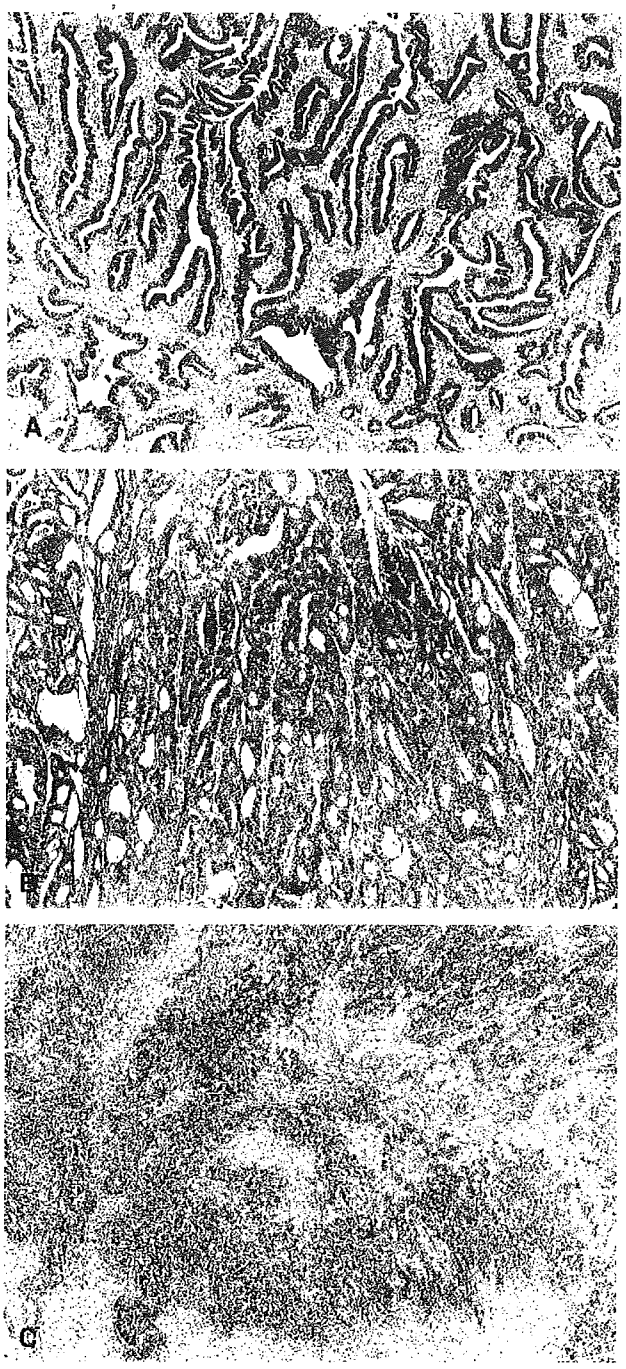

Figure 1. Histologic types: (A) well, (B) moderately, and (C) poorly differentiated adenocarcinoma (hematoxylin and eosin; original magnification, ×40).

45M1, respectively. Substitutions of similar dilutions of control mouse immunoglobulin G1 were used in negative controls. Positive membrane staining by CD10 and positive cytoplasmic staining by MUC2 and HGM were judged as positive reactions for each marker. Tumors were designated positive for a marker when >5 percent of tumor cells showed a positive reaction for that marker (Fig. 3).

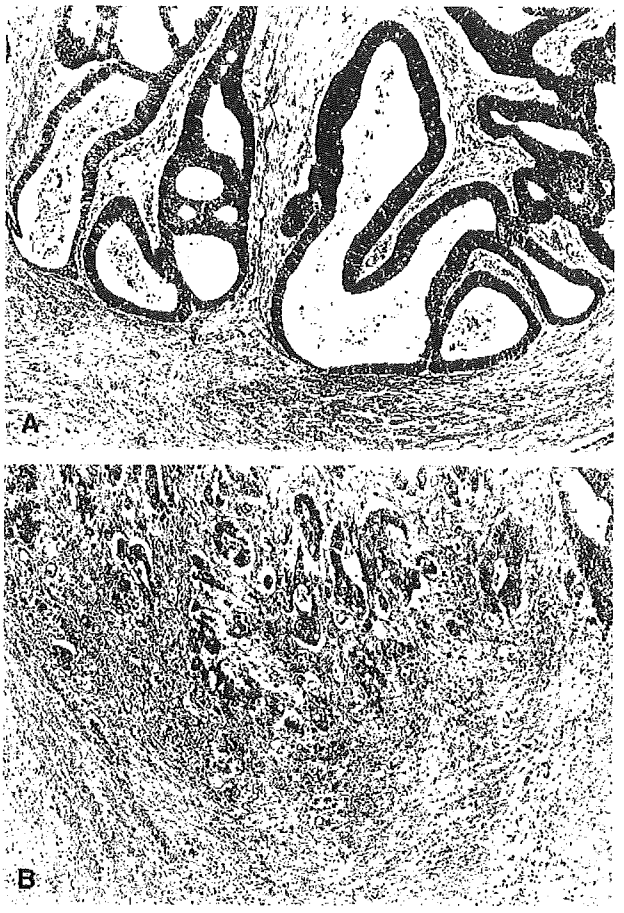

Figure 2. Dedifferentiation of invasive front: (A) negative, (B) positive (hematoxylin and eosin; original magnification, ×100).

Fisher exact test or the chi-squared test was used for univariate comparisons. A multivariate logistic regression was performed to identify significant contributors that were independently associated with liver metastases from among all the factors examined. Statistical analysis was performed with the SPSS Statistical Software Package (version 11.0J, SPSS-Japan Inc., Tokyo, Japan). All variables were dichotomized for analysis. The cutpoint for dichotomization was set according to the previous studies. Differences were considered significant at $P < 0.05$.

RESULTS

Patient characteristics are summarized in Table 1. The patients had been followed up for a range of 0.1 to 99 (median, 67) months. Two patients were followed up for 0.1 months. One of them died of pulmonary thrombosis three days after operation, and the other patient had synchronous metastasis, and

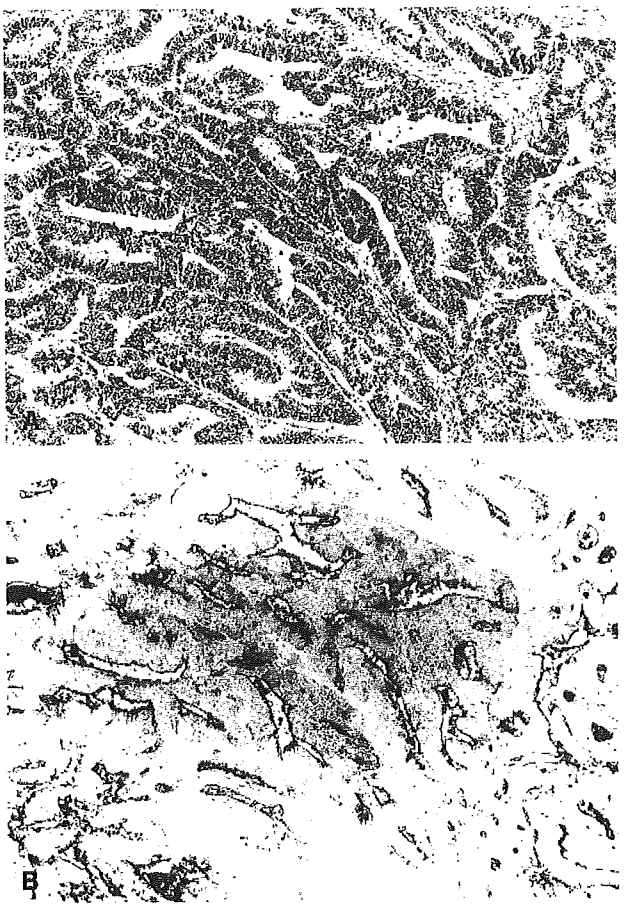

Figure 3. Colonic adenocarcinoma with CD10 expression. A. The tumor is a moderately differentiated adenocarcinoma (hematoxylin and eosin; original magnification, ×100). B. CD10 is expressed on the luminal surface along the apical membranes of the carcinomatous glands (CD10, original magnification, ×100).

died four days postoperatively of septic shock secondary to anastomotic leakage. All of the other survivors had been followed for at least five years. Resectable and unresectable hepatic metastases were detected in 122 patients who had been followed for at least five years: 71 (58 percent) synchronous metastases, and 51 (42 percent) metachronous. The overall rate of resectability was 45 percent (55/122): 24 of 71 (34 percent) synchronous metastases, and 31 of 51 (61 percent) metachronous lesions.

Correlations between clinicopathologic and immunohistochemical factors in the 122 cases of colorectal carcinoma with liver metastasis and in the 383 cases of colorectal carcinoma without liver metastasis are shown in Table 1. Univariate analysis showed that tumor size of >5 cm, histologic type of non–well-differentiated adenocarcinoma, dedifferentiation of invasive front, invasion deeper than the subserosa, lymphatic invasion, venous invasion, lymph-node metastasis, and CD10 expression were significantly correlated with liver metastasis. Multivariate analysis showed that invasion deeper than the subserosa, venous invasion, lymph-node metastasis, and CD10 expression were significantly correlated with liver metastasis (Table 2).

CD10 expression was not significantly correlated with synchronous *vs.* metachronous lesions. We did not analyze for any correlations between CD10 expression and the resectability of liver metastases or other histologic markers.

DISCUSSION

Surgical resection is currently the only treatment that can provide long-term survival and a possible cure for patients with liver metastases from colorectal carcinoma.[1–4] We have to distinguish between the limited impact of aggressive surgery on the entire sample of patients suffering from metastatic spread and the possible advantage that may be offered to a subgroup with completely resectable disease. Several studies have reported that only 24 to 31 percent of patients with liver metastases from colorectal carcinoma can undergo resection.[3,5,33] In our study, the overall rate of resectability of liver metastases was 45 percent: 34 percent of the synchronous metastases, and 61 percent of the metachronous ones.

These differences between our rate of resectability and those of previous reports can be explained by the following two reasons. First, in our hospital, techniques, such as preoperative portal embolization[34] and intraoperative ultrasonography,[35] which have been associated with improvements in perioperative patient management, have enabled safe hepatic resections with no deaths and have extended the possibility of liver surgery to patients with advanced metastatic tumors. Second, recent developments in technologies, such as CT and magnetic resonance imaging, have enabled physicians to detect metastases in their early stages.

However, for patients with unresectable metastases, there is essentially no therapy that can provide long-term survival. If it were possible to identify characteristic features in the primary lesion that were strongly related to liver metastasis, the early detection of liver metastasis could become feasible by use of such markers, and it would be possible to select patients who needed close monitoring. We found that

Table 1.
Comparison of Clinicopathologic and Immunohistochemical Factors of Colorectal Carcinomas With Liver Metastasis and Colorectal Carcinomas Without Liver Metastasis

Factor	Liver Metastasis		
	Positive (n = 122)	Negative (n = 383)	*P* Value
Age ≤60/≥61 (yr)	63/59	167/216	NS
Male/female ratio	75/47	231/152	NS
Location (colon/rectum)	84/38	228/155	NS
Gross type (polypoid/ ulcerative)	7/115	37/346	NS
Size (<5 cm/≥5 cm)	61/61	234/149	0.035
Histologic type (well/non-well)[a]	38/84	176/207	0.004
Dedifferentiation (+)[b]	41 (34)	89 (23)	0.024
Depth (T2/ T3,T4)[c]	4/118	86/297	<0.001
Lymphatic invasion (+)	108 (89)	255 (67)	<0.001
Venous invasion (+)	104 (85)	198 (52)	<0.001
Lymph node metastasis (+)	100 (82)	183 (48)	<0.001
CD10-positive[d]	63 (52)	132 (34)	0.001
MUC2-positive[d]	62 (51)	210 (55)	NS
HGM-positive[d]	11 (9)	40 (10)	NS

NS = not significant.
Data are numbers with percentages in parentheses unless otherwise indicated.
[a]Histologic type was defined by the histologic features constituting >50% of the tumor area (well = 214 patients with well-differentiated adenocarcinoma; non-well = non–well-differentiated adenocarcinoma, which include 256 patients with moderately differentiated adenocarcinoma, 19 poorly differentiated adenocarcinoma, 12 mucinous adenocarcinoma, 2 endocrine cell carcinoma, and 2 squamous-cell carcinoma).
[b]The tumor was designated as positive for dedifferentiation when, in the section containing the deepest site of cancer invasion, >10 percent of the area of the invasive front consisted of dedifferentiation units.
[c]TNM classification.
[d]Tumors were designated as positive for a marker when > 5 percent of tumor cells showed a positive reaction for that marker.

Table 2.
Logistic Regression Analysis of Liver Metastasis[a]

Factors	OR	(95% CI)	*P* Value
Depth[b]	3.588	(1.178–10.928)	0.025
Venous invasion	3.413	(1.829–6.371)	<0.001
Lymph node metastasis	3.137	(1.769–5.564)	<0.001
CD10 expression	2.009	(1.234–3.272)	0.005

OR = odds ratio; CI = confidence interval.
[a]Logistic regression analysis was performed for all factors, including age, gender, tumor location, gross type, size, histologic type, dedifferentiation of invasive front, depth of invasion, lymphatic invasion, venous invasion, lymph-node metastasis, and expression of CD10, MUC2, and human gastric mucin.
[b]Invasion deeper than subserosa.

CD10 expression in the primary colorectal carcinoma was a new independent predictor of liver metastasis. CD10 expression was found in 52 percent of the patients with liver metastasis. Although the odds ratio of the CD10 staining (odds ratio was approximately 2) in the multivariate analysis was lower than that of other more well-known markers, such as depth of invasion, vascular invasion, and lymph node metastasis (odds ratios were approximately 3), CD10 expression was an independent predictor for liver metastasis. We, therefore, believe that each marker is important for predicting liver metastasis. CD10 expression could, however, be a useful newly identified predictor of liver metastasis and could be a useful marker to select patients who require close monitoring.

Many authors have indicated a close relationship between venous invasion and liver metastasis.[10–15,19] Some studies have shown a significant correlation between lymph node metastasis with liver recurrence and patient prognosis.[13,16,17,19] The independent effects of venous invasion and lymph node metastasis on liver metastasis also were confirmed in our multivariate analysis.

The overall incidences of positive CD10, MUC2, and HGM expression in colorectal carcinoma were 39, 54, and 10 percent, respectively. The incidences of CD10, MUC2, and HGM expression in colorectal carcinoma with liver metastases were 52, 51, and 9 percent, respectively. Previously, the only study to observe the incidence of phenotypic expression of these markers in colorectal carcinoma was by Yao *et al.*[18] They reported that 22, 49, and 14 percent of all speci-

mens were stained positively with CD10, MUC2, and HGM, respectively. Our larger series of consecutive patients confirmed the results of these researchers.

Five percent was used as a cutoff after the recommendations of previous reports.[18,19] After analyzing the variable positivities of tumor cells according to the percentage of immunopositive staining as a potential cutoff index (*e.g.*, 5, 10, 50 percent), we chose 5 percent as the cutoff level, because its *P* value was the highest. When comparing the statistical significance of the variable cutoff levels with each other, and we selected 5, 10, and 50 percent as the cutoff level; their respective *P* values were 0.001, 0.002, and 0.013.

Until now, little had been reported of the correlation between CD10 expression and colorectal carcinoma, and CD10 expression in the primary colorectal carcinoma had not been evaluated by multivariate analysis as a predictor of liver metastasis. Yao *et al.*[19] had reported that the incidence of CD10 expression was significantly higher in colorectal carcinoma with liver metastasis (58 percent) than in a control group (22 percent) by the chi-squared test. Although their study lacked multivariate-type analysis, we confirmed by our multivariate analysis that there was a significant association between CD10 expression in colorectal carcinoma and liver metastases. The reason for this association is unknown. CD10 seems to have some roles in vascular invasion, because the incidence of CD10 expression was significantly higher in colorectal carcinoma with liver metastasis. Although the data were not shown in our study, CD10 expression tends to be seen more frequently at the luminal surfaces of the cribriform glands in moderately differentiated adenocarcinoma than that in well-differentiated adenocarcinoma. Therefore, we can speculate that CD10 expression has some role in tumor differentiation and is correlated with liver metastasis, because non–well-differentiated tumors showed a higher incidence of liver metastasis than did well-differentiated tumors.

In previous studies, it has been reported that MUC2 is abundantly expressed in normal colonic goblet cells and that expression of MUC2 mucin is decreased in colonic adenoma and carcinoma cells compared with normal mucosa.[27–29] Some studies have indicated that suppression of the MUC2 gene in colorectal carcinoma cells *in vitro* is strongly associated with the metastatic process,[20] and MUC2 is involved in the suppression of colorectal cancer in mice.[21] Li *et al.*[22] reported that reduced MUC2 expression might be related to malignant transformation of colorectal adenoma and carcinoma, because MUC2 mucin expression was significantly lower in both adenomas with moderate or severe dysplasia and carcinomas than in adenomas with mild dysplasia. Yao *et al.*[19] found that MUC2 positivity (in both primary and metastatic tumors) did not differ between colorectal carcinomas with liver metastasis and those without liver metastasis. In our study, MUC2 positivity was not associated with liver metastasis. These discrepancies between mRNA and gene expression analysis and immunohistochemical analysis might be related to differences in the sensitivity of each type of test in detecting MUC2 expression. Another reason may be that MUC2 expression is associated mainly with the development of colorectal carcinomas and not with liver metastases. Most previous reports examined the correlation between MUC2 expression and development of colorectal carcinomas. Our study examined the correlation between MUC2 expression and liver metastases. Ours is the first study to examine HGM expression in colorectal carcinomas.

CONCLUSION

CD10 expression in the primary colorectal carcinoma was significantly associated with liver metastasis.

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Flat and Depressed Lesions of the Colorectum

SHIN-EI KUDO and HIROSHI KASHIDA
Digestive Disease Center, Showa University Northern Yokohama Hospital, Chigasaki-chuo, Tsuzuki-ku, Yokohama, Japan

Although flat and depressed-type lesions are found by regular endoscopic view, magnification and pit pattern observation are vital parts of a precise diagnosis of the lesion. The depressed-type lesions have a prominent tendency to show malignant character, and the recognition and timely treatment of this lesion is inevitable in improving the morbidity and mortality from colorectal cancer. A magnifying colonoscope has the capability of a regular colonoscope with an additional feature: magnification. With chromoscopic techniques, the surface pattern of the mucosal pits can be observed. The pit-pattern classification correlates well with actual histologic findings and provides important additional information before endoscopic treatment of the lesion.

The depressed-type colon cancer first was described in 1977.[1,2] The occurrence of its lesion is accepted widely and now is reported throughout the world.[3] The advent of commercially available magnifying videocolonoscopes with high-power resolution in 1993 accelerated the study of the microstructure of colonic lesions. The combination of chromoscopy and magnifying colonoscopy is useful for detecting small localized lesions for the differential diagnosis and for determining not only the lateral margins but also the depth of a lesion.[4] We use colonoscopes with magnification routinely for colonoscopic examinations. Therefore, we can perform detailed examination with magnification immediately at the time a lesion is found in the colon. The openings of the colonic crypts are referred to as *pits*, and the specific arrangement of the openings of the glands in various kinds of lesions is called the *pit pattern*.[4,5] The aim of this study was to clarify the characteristics and importance of flat and depressed lesions of the colorectum.

Materials and Methods

Our database consists of prospectively collected data of consecutive patients who underwent colonoscopic examinations for any indication over a period of 20 years: from 1985 to 2004. The colonoscopic examinations were performed with magnifying colonoscopes (CF 240ZI, 260AZI, or 200ZI; Olympus, Tokyo, Japan). When a lesion was suspected or identified, chromoscopy was performed with .2% indigo carmine dye and, in addition, .05% crystal violet dye as indicated. All resected lesions were documented with pit-pattern findings, diameter, and final pathologic findings. The number of endoscopically or surgically treated neoplastic lesions was 21,262. The lesions were divided into 3 groups: protruded, flat-elevated, and depressed. The pit patterns, or microstructures of the surface of the lesions, were classified as type I, II, IIIs (small), IIIL (large), IV, VI (irregular), or VN (nonstructural), as previously reported (Figure 1).[6] Indigo carmine dye accumulates in the glandular orifices, which usually clarifies the pit pattern. However, in some lesions, especially those with type IIIs and type V pit patterns, the openings are too narrow, too distorted, or too sparse for enough dye to be retained. In such cases, crystal violet dye is used, which stains the absorbent epithelium of the colon and makes the pit pattern conspicuous.

Results

The rates of invasive cancer in early colorectal neoplasms that appear as protruded and flat elevated lesions between 6 and 10 mm in diameter were 1.3% and .18%, respectively (Table 1). The invasive rate with depressed lesions for the same size group was 43.2%. On magnifying colonoscopy, most (95.7% and 95.7%) protruded and flat neoplasms showed type IIIL or IV pit pattern. Eighty-six percent of the depressed lesions were characterized by type IIIs, VI, or VN pit pattern. The pit patterns correlated well with the final histologic diagnosis (Table 2). Type IIIs, IIIL, and IV pit patterns were typical of adenomas, and were seen only rarely in invasive cancers (0%, 3.3%, 2.3%, respectively). On the other hand, 88.9% of type VN lesions were invasive.

Discussion

The gross appearances of early colorectal neoplasms are divided into 3 categories: protruded, flat elevated, and depressed.[7] Recognition of depression is very important because depressed lesions often are associated with invasive cancer when they are very small. Detecting a tiny area with a slight color change is important; some lesions look slightly reddish and some look pale or discolored. Bleeding spots, interruption of the capillary network pattern, or

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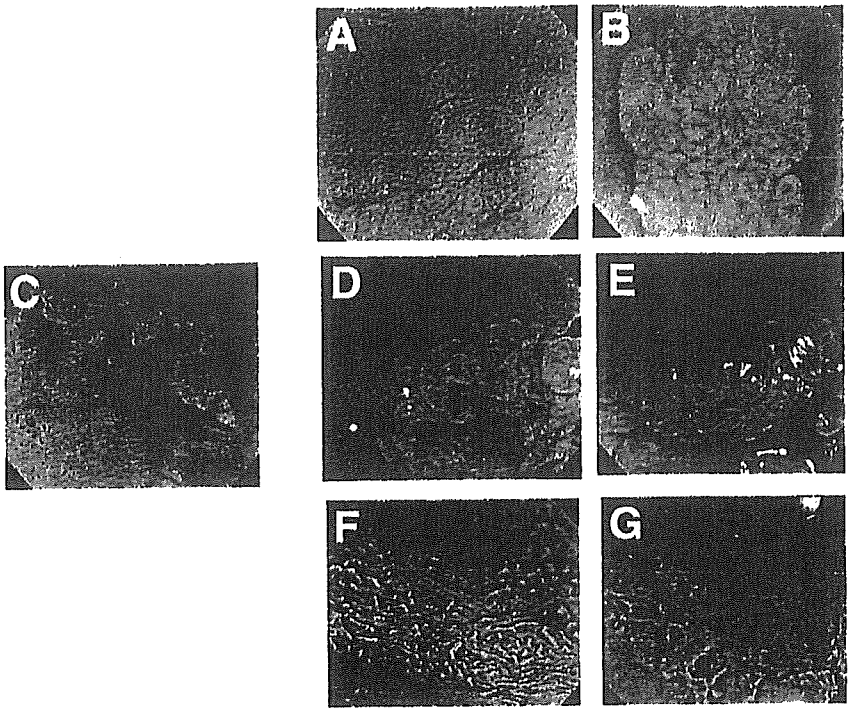

Figure 1. Pit pattern classification. (*A*) Type I: normal roundish pits; (*B*) type II: large star-shaped pits; (*C*) type IIIs: small roundish pits; (*D*) type IIIL: long tubular pits; (*E*) type IV: branched pits; (*F*) type VI: irregular pits; (*G*) type VN: nonstructural pit pattern. Types I and II are non-neoplastic pit patterns. Types IIIs, IIIL, and IV are adenomatous pit patterns. Types VI and VN are cancerous pit patterns.

slight deformation of the colonic wall may suggest the existence of a depressed lesion. The depressed-type colorectal cancers either can be absolutely depressed or accompanied by a slightly elevated margin. Some lesions with depression are elevated as a result of submucosal invasion and proliferation of the tumor cells.[8] Such lesions must not be mistaken for ordinary elevated neoplasms because they are quite different from each other in biological behavior. Chromoscopy is useful for confirming small colorectal lesions, for determining their lateral extent, and for clarifying their gross configuration; this is useful especially in determining the presence or absence of depression within the lesions.

There is some confusion about the depressed and flat lesions.[9] Lesions that are called *flat* adenomas are not absolutely flat, but often are elevated slightly. It is true that some adenomas appear to have a depression and resemble depressed-type early cancers; the depression in a depressed lesion is rather extensive and clearly demarcated. In contrast, the depression in flat elevated adenomas actually is an ill-defined pseudodepression with only a thorny or groove-like appearance. Depressed lesions are not part of flat adenomas but should be regarded as a different entity, because the latter almost invariably are benign. Invasive rates in flat elevated adenomas are slightly lower than but not remarkably different from those in protruded polyps. Flat lesions usually are benign or only focally malignant and grow very slowly, not becoming invasive until they are rather large. In contrast, depressed lesions apparently grow rather rapidly, advancing at an early stage. The depressed-type lesions are reported not to have K-*ras* point mutation, although their genetic alterations are not clear.[10] It is certain that they arise without adenoma-carcinoma sequence.[8]

Table 1. Rate of Submucosal (T1) Cancer in Early Colorectal Neoplasms

	Size (*mm*)					
	0–5	6–10	11–15	16–20	21+	Total
Depressed	20/249	64/148	49/70	19/22	14/16	166/505
	8.0%	43.2%	70.0%	86.4%	87.5%	32.9%
Flat-elevated	2/6573	2/1120	13/533	19/182	61/276	97/8684
	.03%	.18%	2.4%	10.4%	22.1%	1.1%
Protruded	0/5909	57/4464	85/1095	64/387	65/218	271/12,073
	0%	1.3%	7.8%	16.5%	29.8%	2.2%
Total	22/12,731	123/5732	147/1698	102/591	140/510	534/21,262
	.17%	2.1%	8.7%	17.3%	27.5%	2.5%

NOTE. Data from April 1985 to August 2004.

Table 2. Pit Pattern and Histology of the Lesion

Pit pattern	Adenoma		Cancer	Total
	Low grade	High grade	(T1 stage)	
IIIL	1984	464	0	2448
	81.0%	19.0%		
IV	266	261	18	545
	48.8%	47.9%		
IIIs	21	21	1	43
	48.8%	48.8%		
VI	32	140	59	231
		60.6%	25.5%	
VN	0	8	64	72
			88.9%	
Total	2303	894	142	3339

NOTE. Data from April 2001 to August 2004.

Dye spraying can be used during routine examination with an ordinary colonoscope, but it is especially useful when combined with magnifying colonoscopy. The magnified view can be obtained instantaneously simply by slightly rotating the magnification knob of the scope or stepping on the foot controller. Zoom colonoscopes have all the basic functions of conventional colonoscopes; therefore, they can be used during routine examinations with an ordinary view. The combination of chromoscopy and magnifying colonoscopy is useful for the differential diagnosis of a colorectal lesion and for predicting the depth of a cancer because it enables one to observe the detailed structure of the lesion. There is definite correlation between the gross appearance and the pit pattern of a colorectal lesion.[4,11] Depressed lesions present with type IIIs or V pit pattern; the latter implies that the lesion is cancerous. Almost all flat and protruded neoplasms have type IIIL or IV pits.

There also is correlation between the pit pattern and the histology of the lesion. The pit patterns are useful for distinguishing neoplastic lesions from nonneoplastic changes. In neoplastic lesions, pit-pattern analysis is useful for distinguishing between low-grade adenomas and invasive cancers. The majority of the lesions that present only type IIIs, IIIL, or IV pits are low-grade adenomas. Type V pit pattern is typical of cancers and can be subdivided into 2 subtypes. In deeply invasive or advanced cancers the surface of the lesion is rough and often ulcerated; therefore it almost is devoid of pits and looks nonstructural. Such a pit pattern is named type VN (nonstructural [N]). In severely dysplastic adenomas and minimally invasive carcinomas, the pit pattern is not completely nonstructural, but fairly irregular. Such an irregular pit pattern is named type VI (irregular [I]).

Treatment selection should be based on an accurate prediction of cancer invasion of the colorectal lesions.[12] We suggest the use of the highly accurate pit-pattern diagnosis for the determination of treatment. The lesions with type I or II pit pattern almost always are nonneoplastic; therefore, they need not be treated except for submucosal tumors such as carcinoids. Those with type IIIs, IIIL, or IV pit pattern usually are benign adenomas that can be treated endoscopically. The lesions with type VI encompass a variety of lesions from benign adenoma to invasive carcinoma. Therefore, lesions with type VI pit pattern first are treated endoscopically, and additional surgical colectomy and lymph node dissection is considered after the histologic analysis of the excised specimen. The lesions with type VN pit pattern usually are invasive cancers that should be referred for surgery.

Limitations

It cannot be denied that there are some limitations to the pit-pattern diagnosis because pit patterns are the changes of the surface of lesions and do not permit direct analysis of the deeper part. However, the changes in the deeper layers also are reflected on the surface to some extent; therefore, pit patterns generally are more useful in practice.[13]

Conclusions

Flat, and especially depressed, lesions are important in the colorectum. Chromoscopy is important for an accurate diagnosis of these lesions. The pit-pattern analysis helps predict the histology of the lesions and therefore is useful in determining the treatment selection.

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Address requests for reprints to: Shin-ei Kudo, MD, PhD, Digestive Disease Center, Showa University Northern Yokohama Hospital, 35-1 Chigasaki-chuo, Tsuzuki-ku, Yokohama 224-8503, Japan. e-mail: kudos@med.showa-u.ac.jp or kashi-md@xf6.so-net.ne.jp; fax: (81) 45-949-7535.

Internal Hernia Through the Mesenteric Opening After Laparoscopy-Assisted Transverse Colectomy

Koichi Nagata, MD, Jun-ichi Tanaka, MD, PhD, Shungo Endo, MD, PhD, Kishiko Tatsukawa, MD, Eiji Hidaka, MD, PhD, and Shin-ei Kudo, MD, PhD

Abstract: We report a case of a rare complication in laparoscopic colectomy. A 55-year-old woman underwent a laparoscopy-assisted transverse colectomy for transverse colon cancer. On the 5th postoperative day, she developed bowel obstruction. Decompression by a long intestinal tube failed to resolve the bowel obstruction. She underwent operative intervention. Abdominal exploration showed jejunal loop caused by a strangulation forming on an internal hernia through the mesenteric opening at the anastomotic colonic stumps, which had not been sutured during the previous operation. Our experience might indicate the need for closure of small mesenteric opening after laparoscopic colectomy.

Key Words: internal hernia, intestinal obstruction, laparoscopic surgery, colon cancer, complication

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A 55-year-old woman was admitted to our hospital for surgical treatment of transverse colon cancer. Colonoscopic study demonstrated a 4.5 × 5.0-cm protruded lesion in the transverse colon. Biopsies were performed, and the results showed well-differentiated adenocarcinoma. The patient underwent a laparoscopy-assisted transverse colectomy for transverse colon cancer. Intracorporeal ligation of tumor-feeding vessels (middle colic vessels) at their origins was performed initially. After mobilization of hepatic and splenic flexures, the bowel loop was delivered under a wound protector through a 6-cm midline incision of the upper abdomen. The division of the marginal vessels and functional end-to-end anastomosis was performed extracorporeally with linear staplers (PROXIMATE Linear Cutter 75; Ethicon Endo-Surgery, Cincinnati, OH). The mesenteric opening region resulting from bowel resection was not closed for preference. After the closure of the minilaparotomy, the anastomosis and the small mesenteric opening region were checked laparoscopically and found to be normal without internal hernia. On postoperative day (POD) 5, she had nausea, vomiting, and abdominal distention. Plain abdominal x-ray film revealed air-fluid levels in the left upper quadrant (Fig. 1). This finding was thought to be compatible with an adhesive small bowel obstruction initially. Surgery was not chosen because her clinical symptoms and plain x-ray film improved after decompression by a long intestinal tube. Body temperature, pulse, and white blood cell count were within normal limits. There were no signs of clinical indication of strangulation suggesting a need for urgent surgery. She was treated conservatively with bowel rest and total parenteral nutrition. Despite this treatment, high-volume output from a long intestinal tube persisted (1600–3000 mL/day), and, on POD 15, an upper gastrointestinal radiologic contrast study demonstrated obstruction of the proximal jejunum (Fig. 2). She eventually underwent surgical intervention. At laparotomy, there was jejunal loop caused by a strangulation forming on an internal hernia through the mesenteric opening at the anastomotic colonic stumps, which had not been sutured during the previous surgical procedure. A part of jejunum, 80 cm in length corresponding to 5 to 85 cm of the jejunum from Treitz's ligament was strangulated (Fig. 3). After lysis of the adhesions and relieving of the strangulation, the jejunal loop was returned to the abdomen. The operation was concluded by sealing the mesenteric opening and closing the wall of the abdomen in layers. The postoperative course was uneventful. At 11 months' follow-up, there was no clinical or radiographic evidence of recurrence of the cancer or the internal hernia.

DISCUSSION

Laparoscopic adhesiolysis for small bowel obstruction was first reported in 1991.[1] Laparoscopic adhesiolysis has been shown to decrease the incidence, extent, and severity of intraabdominal adhesions when compared with open approach, thus potentially decreasing the recurrence rate for adhesive small bowel obstruction.[2,3] We expected this patient with a complete obstruction caused by adhesions preoperatively (Fig. 2) to not be a good candidate for laparoscopic adhesiolysis.[3]

Laparoscopy-assisted colectomy (LAC) becomes the procedure of choice for colonic disease at many facilities worldwide.[4–9] There are many recognized advantages of LAC, including decreased pain, improved cosmesis, decreased postoperative ileus, shortened hospital stay, and more rapid return to normal activities.

The frequently reported complications after LAC are incisional infection, anastomotic fistula, abdominal abscess, intestinal obstruction, incisional hernia, perineal hernia, and others.[5–13] Intestinal obstruction is one of the common complications after LAC. The incidence is reported to be

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From the Digestive Disease Center, Showa University Northern Yokohama Hospital, Yokohama, Japan.
Reprints: Koichi Nagata, MD, Digestive Disease Center, Showa University Northern Yokohama Hospital, 35-1 Chigasaki-chuo, Tsuzuki-ku, Yokohama 224-8503, Japan (e-mail: Nagata7@aol.com).

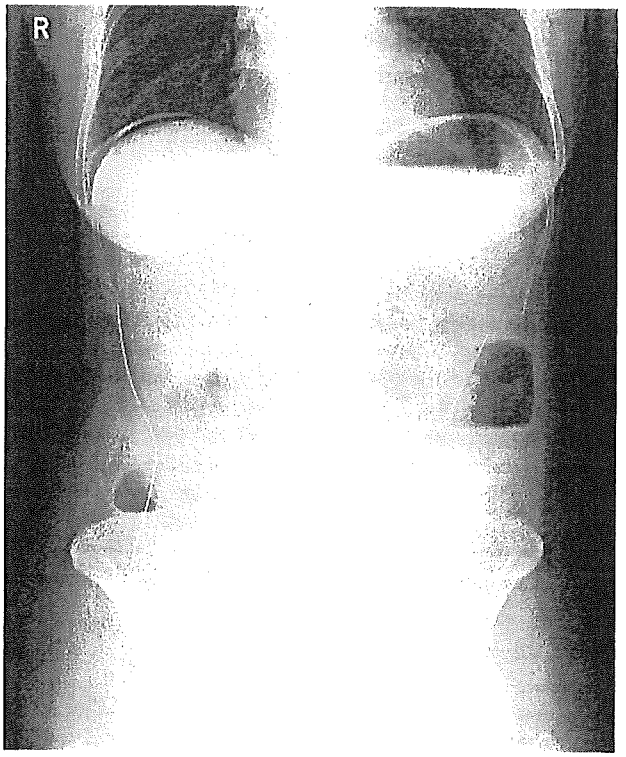

FIGURE 1. Plain abdominal x-ray. Air-fluid levels in the left upper quadrant.

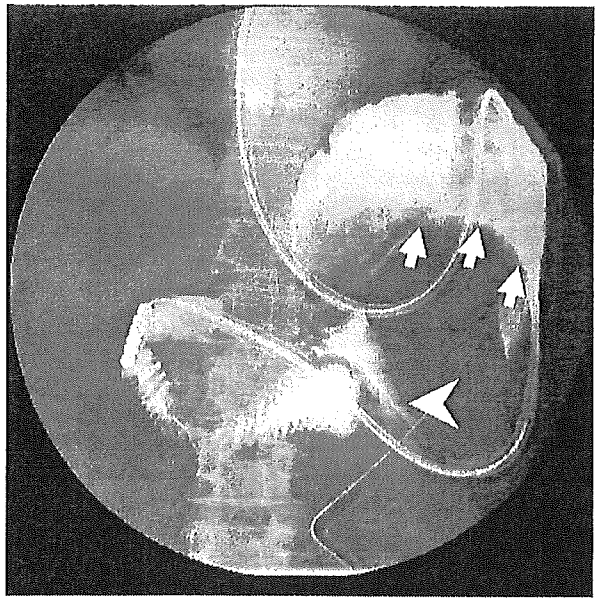

FIGURE 2. Upper gastrointestinal radiologic contrast study. The arrowhead is pointing obstruction of the proximal jejunum. The stomach is excluded upper side (arrows).

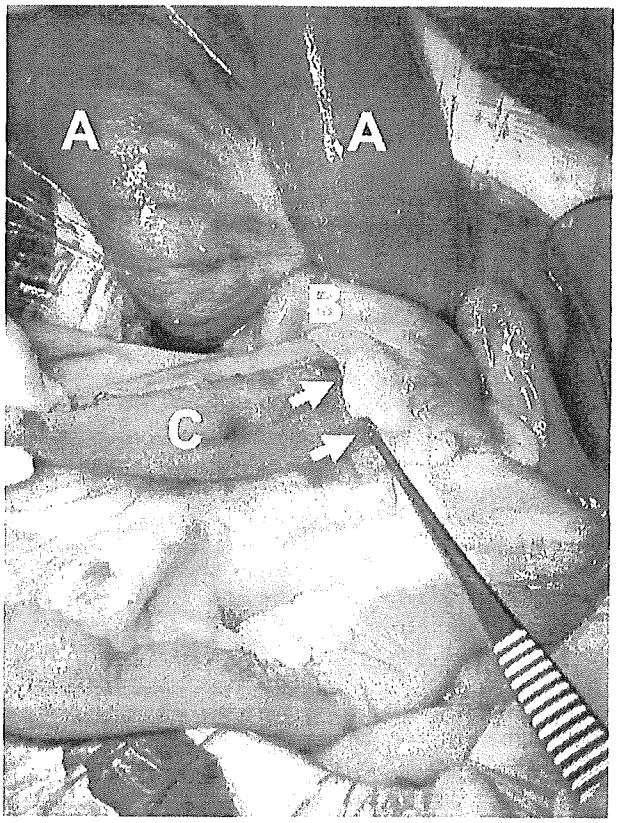

FIGURE 3. Intraoperative photograph. The jejunum (A) has herniated through the mesenteric opening (arrows) in the anastomotic colonic stumps (B). C, Distal side of the jejunum.

0.7%–2.7%.[5-14] The most frequent cause of intestinal obstruction is Richter's hernia at the trocar site. Internal hernia, herniation of the internal organs through defects in the intraabdominal cavity, is not a common cause of intestinal obstruction. Intestinal obstruction secondary to internal hernia after laparoscopic surgery is a rare event. Such conditions have been reported after laparoscopic Roux-en-Y gastric bypass,[15,16] laparoscopic donor nephrectomy,[17] and laparoscopic Nissen fundoplication.[18] However, few data exist regarding the complications of postoperative internal hernia after LAC.

A total of 269 patients underwent LAC without closing the mesenteric opening between April 2001 and October 2004 at our institution. This is the first case (one of the 269 patients; 0.37%) of internal hernia through the mesenteric opening after LAC. The data on the incidence of small bowel obstruction from internal hernia after LAC are scarce. In 1996, Kok et al[11] reported a case of small bowel hernia through a mesenteric defect after laparoscopy-assisted sigmoidectomy. In 1998, Elio et al[19] reported the case of ileal volvulus on internal hernia after laparoscopy-assisted left hemicolectomy. In 1999, Kawamura et al[20] reported the case of transmesenteric hernia after laparoscopy-assisted sigmoidectomy. To the best of our knowledge, only the above-mentioned cases were reported in the literature.

The omission of closing the mesenteric opening can expose the patient to the risk of small bowel obstruction from internal hernia. Although this complication is a rare event, it involves significant morbidity in an otherwise healthy patient who has undergone LAC.

Duepree et al[14] reported that the incidence of both ventral hernia and postoperative small bowel obstruction after laparoscopic resection is lower than that of after open surgery. There have been reported only 3 patients with small bowel obstruction from internal hernia after LAC, and the incidence is very low.[11,19,20] It would seem safe to assume that there is no need to close the mesenteric opening in all cases of laparoscopic colectomy. But if the mesenteric opening is small, then it is likely to cause internal hernia, and our experience might indicate the need to close the mesenteric opening by suturing or clipping after bowel resection. However, more cases are required to explain the indication for closure of mesenteric opening to prevent this complication.

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Rectovaginal fistulas after rectal cancer surgery: Incidence and operative repair by gluteal-fold flap repair

Chihiro Kosugi, MD,[a,c] Norio Saito, MD,[a] Yoshitaka Kimata, MD,[b] Masato Ono, MD,[a] Masanori Sugito, MD,[a] Masaaki Ito, MD,[a] Kazunori Sato, MD,[a] Keiji Koda, MD,[c] *and* Masaru Miyazaki, MD,[c] *Chiba, Japan*

Background. *We investigated the correlation between operative procedures for rectal carcinoma and postoperative rectovaginal fistulas (RVF), and treatment for RVF.*
Methods. *The medical records of 161 female patients with rectal carcinoma were examined retrospectively with respect to the cause, incidence, and methods of treatment for RVF occurring after rectal cancer operations, and to the outcomes of gluteal-fold flap repairs for RVF.*
Results. *Of the 161 patients, 16 developed RVF clinically. The incidence of RVF was significantly higher in patients who were anastomosed by the double stapling technique (DST) and had concomitant resection of the vaginal wall. No statistical difference was found between the established diverting ostomy group and the no-stoma group. Six patients recovered by the establishment of a diverting ostomy only. The gluteal-fold flap technique was performed for 5 patients. No RVF recurrences were noted in these 5 patients.*
Conclusions. *The incidence of RVF was higher in the patients who were anastomosed by DST or had concomitant resection of the vaginal wall. Although some RVFs heal with only fecal diversion, for patients in whom RVF is caused by involvement of the vaginal wall in the circular staple or intersphincteric resection, good results are obtained with the gluteal-fold flap repair technique. (Surgery 2005;137:329-36.)*

From the Divisions of Colorectal (Pelvic) Surgery,[a] and Plastic and Reconstructive Surgery,[b] National Cancer Center Hospital East, Kashiwa; and the Department of General Surgery,[c] Graduate School of Medicine, Chiba University

FOR PATIENTS WITH LOW RECTAL CARCINOMA, a coloanal anastomosis or low colorectal anastomosis using double stapling has become popular.[1-4] This anastomotic method may sometimes result in rectovaginal fistula (RVF) as a clinical technical complication although most surgeons are alert to this complication.[5-6] Furthermore, anastomotic leakage often causes RVF when an intrapelvic abscess penetrates the posterior vaginal wall.

Crohn's disease, cryptoglandular disease, and obstetrical injury are known causes of RVF. Various methods of repair of RVF arising in these diseases and as a complication of rectal cancer surgery have been used, including the use of a mucosal advancement flap,[7-9] a transsphincteric approach that combines a rectal mucosal advancement flap and an anal sphincteroplasty,[10] fibrin sealant instillation,[11] an ileal pouch mucosal advancement flap or circumferential pouch advancement, and a protectomy with colonic pull-through and delayed coloanal anastmosis.[5,8] Of these methods, an endoanal and endovaginal advancement flap is the most frequently used technique. However, RVF repairs sometimes break down when patients undergo RVF repairs more than once.[12] In 1996, the lotus petal flaps procedure was reportedly used to reconstruct a vulvovaginal defect.[13] We adapted this flap method—the gluteal fold flap—for treatment of RVF in postoperative patients with rectal cancer who have undergone intersphincteric resection or for patients in whom RVF resulted from involvement of the posterior wall of the vagina in a circular stapled anastomosis. Few studies have examined the clinical features, operative methods,

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Reprint requests: Chihiro Kosugi, MD, Department of Colorectal (Pelvic) Surgery, National Cancer Center Hospital East, 6-5-1 Kashiwanoha, Kashiwa, Chiba, Japan. E-mail: ckosugi0126@yahoo.co.jp.

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anastomotic technique, and establishment of a diverting ostomy for postoperative RVF. We retrospectively investigated the relationship between operative procedures and RVF, and whether a gluteal-fold flap repair is an effective and simple solution for the treatment of RVF.

PATIENTS AND METHODS

The medical records of 204 patients undergoing treatment for primary rectal cancer during July 1992 to November 2002 at our institute were reviewed. Forty-three patients who underwent abdominoperineal resection, Hartmann's operation, or total pelvic exenteration were excluded. The remaining 161 patients who underwent sphincter-preserving surgery were retrospectively examined. Of these 161 patients, high anterior resection (HAR) was performed for 34 patients, low anterior resection (LAR) for 81 patients, very low anterior resection (vLAR) for 39 patients, and intersphincteric resection (ISR) for 7 patients.

Of the 161 patients, 8 patients had diabetes and 12 had vascular disease (eg, angina pectoris and arteriosclerosis). No patients had inflammatory bowel disease. Only 4 of the 161 patients who underwent an ISR received preoperative radiotherapy. The total dose of radiation delivered through a linear accelerator was 45 Gy in 25 fractions over 5 weeks, followed by an operation 2 weeks later. The vaginal wall was opened intraoperatively in 26 patients (16.1%), either because of concomitant hysterectomy with closure of the vaginal stump (n = 5) or as part of the resection of the posterior vaginal wall due to cancerous invasion (n = 21).

A straight coloanal or colorectal anastomosis was accomplished by using the stapling technique or hand sewing in all 161 patients. All patients underwent anastomosis by the single stapling technique (SST) (n = 84; 52.2%), double stapling technique (DST) with a circular stapler (n = 65; 40.4%), or coloanal anastomosis with hand sewing (n = 12; 7.4%). At the time of the anastomosis with the circular stapler, the completeness of all stapler donuts was confirmed by the surgeons. If a stapler donut was incomplete, we tested the anastomotic site for patency with air and sewed up the leakage point of the anastomosis to provide reinforcement with absorbable interrupted sutures. A diverting loop ostomy was established in 79 patients (49.1%) at the initial operation. One or two drains (Pleats Drainage Tube MD-45110, external diameter 10 mm; Sumitomo Bakelite, Tokyo, Japan) were introduced to the posterior and/or anterior side of the colorectal anastomosis. None of the patients had omental interposition of preventive measures for RVF.

Diagnostic evaluation of the patients with RVF included gastrografin enema and/or digital examination after postoperative vaginal flatus, fecal leakage, and/or vaginal discharge were clinically presented.

The cause and incidence of RVF in every operative procedure were examined. Statistical analysis was performed with the chi-square test for independence for comparison between operative methods and the incidence of RVF. A probability value of $P < .05$ was designated as statistically significant for all analysis.

The results of some surgical treatments for RVF and outcomes of the patients who underwent gluteal-fold flap repair were analyzed. Written informed consent was obtained from all patients before the gluteal-fold flap repair. An ethics committee approved the operative treatment for RVF in the rectal cancer patients.

Gluteal-fold flap repair technique for RVF. The patient undergoes a mechanical bowel preparation preoperatively; then, under general anesthesia, the patient is placed in the lithotomy position and the RVF identified. A solution of 1:500,000 adrenalin in saline is injected under the posterior vaginal mucosa, and a transvaginal incision is then made around the RVF. The edges of the incision are undermined circumferentially just over the external rectal muscular layer. The fistula is probed and debrided. The rectal opening of the fistula is then closed with the use of 2 layers of absorbable sutures in an interrupted horizontal mattress fashion (Fig 1).

To adequately cover the closed rectal fistula, we designed the gluteal-fold, which is a triangular or diamond-shaped flap consisting of skin, subcutaneous fat, and superficial fascia (Fig 2). A skin incision is made at the posterolateral margin of the vaginal introitus with some perforators of the internal pudendal artery being used as a pedicle for the flap. We did not mark the points of the perforators of the internal pudendal artery with a Doppler probe. We constructed some perforators of the internal pudendal artery in the fatty tissue to act as a pedicle or an axis of the flap; then, we used thinned subcutaneous fatty tissue to adjust the vaginal mucosal thickness. The flap is turned 180 degrees and then advanced into the vagina (Fig 3), where it is sutured to the vaginal mucosal defect above the fistula and closed with the use of absorbable interrupted sutures. The gluteal skin defect is closed directly (Fig 4). Drains are introduced

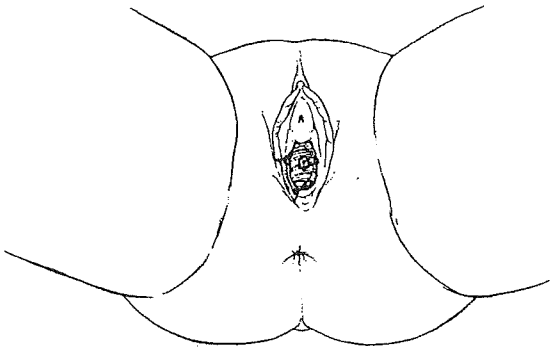

Fig 1. A transvaginal incision, with the edge being undermined circumferentially just over the external rectal muscular layer, is made around the RVF; the rectal opening of the fistula is then closed with the use of 2 layers of absorbable suture in an interrupted horizontal mattress fashion.

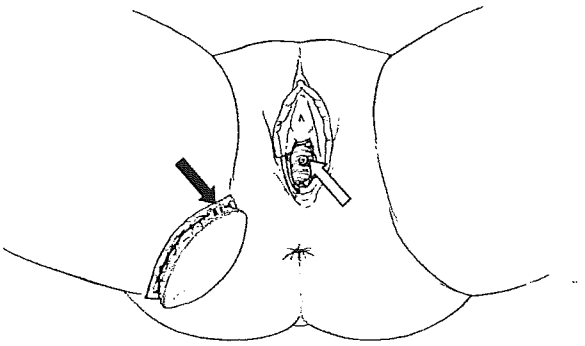

Fig 2. A diamond flap consisting of skin, subcutaneous fat, and the superficial fascia is designed in the glutealfold. Some perforators of the internal pudendal artery in the fatty tissue are constructed to act as a pedicle or an axis of the flap (*black arrow*). The *white arrow* indicates the position of the RVF.

under the subcutaneous tissue of the skin flap. A diverting loop ostomy is established at the ileum or transverse colon if this procedure has not already been performed.

Prophylactic antibiotic coverage is used for 3 days. The drain is removed 5 to 7 days after surgery, and the patients are permitted to walk and sit 7 days after surgery. The diverting loop ostomy is closed 3 to 6 months later to ensure the RVF has healed without recurrence, as determined by barium enema examination.

RESULTS

Table I shows the correlation of the incidence of RVF with the operative method. Of the 161 patients, RVF occurred clinically in 16 (9.9%). The onset of postoperative RVF ranged from 3 to

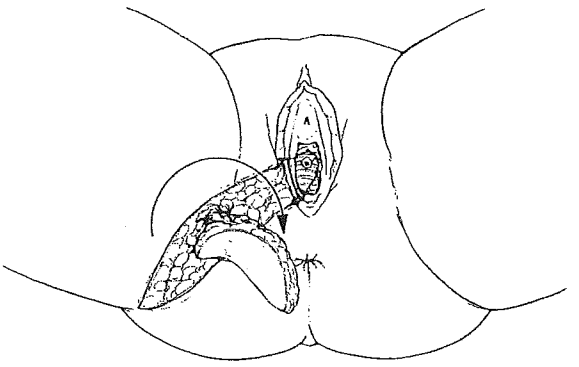

Fig 3. Flap is turned 180 degrees and then advanced into the vagina.

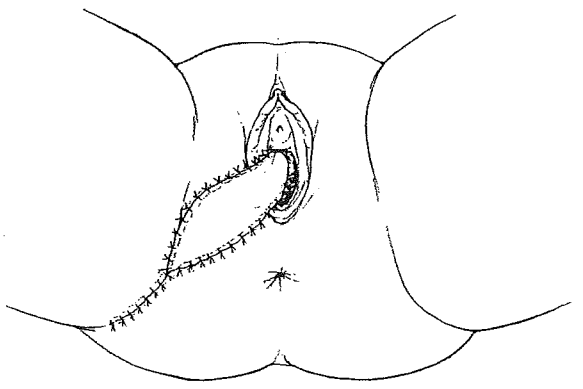

Fig 4. Flap is sutured to the vaginal mucosal defect above the fistula and closed with the use of absorbable interrupted sutures.

21 days after rectal cancer operations, (mean ± SD; 11.5 ± 6.2 days). Ideally, the symptoms of 16 patients with RVF were vaginal flatus and fecal leakage (n = 7), and vaginal discharge (n = 9). RVF was found in 6 of the patients who underwent LAR (7.4%), 7 of the patients who underwent vLAR (17.9%), and 3 of the patients who underwent ISR (42.8%). No statistical differences in RVF incidence were observed between LAR and vLAR ($P = .128$). However, a statistically significant difference was observed between anterior resection and ISR ($P = .0101$).

Tables II and III show the correlation between the operative procedures and the incidence of RVF. For anastomotic technique, a statistically significant difference was observed between SST and DST ($P = .00763$). In the 26 patients who underwent a concomitant hysterectomy and/or resection of the vaginal wall, 10 patients (38.5%) developed RVF. In 8 of these 10 patients, a diverting ostomy was established when the initial curative resections for rectal cancer were performed. A

Table I. The incidence of RVF according to operative procedure

	RVF(+)	*RVF(−)*
Surgical procedures		
HAR	0 (0%)	34
LAR	6 (7.4%)	75
vLAR	7 (17.9%)	32
ISR	3 (42.9%)	4
Total no. of patients	16 (9.9%)	145

RVF, Rectovaginal fistula; *HAR*, high anterior resection; *LAR*, low anterior resection; *vLAR* very low anterior resection; *ISR*, internal sphincteric resection.

Table II. The incidence of RVF for operative procedures excluding HAR

	RVF(+)	*RVF(−)*
Anastomosis technique		
SST	3 (4.8%)	59
DST	9 (15.2%)	44
Hand sewing	4 (33.3%)	8
Diverting ostomy		
(+)	9 (11.4%)	70
(−)	7 (14.9%)	41
Combined resection of uterus or posterior vaginal wall at operation for rectal cancer		
(+)	10 (38.5%)	16
(−)	6 (5.9%)	95

RVF, Rectovaginal fistula; *SST*, single stapling technique; *DST*, double stapling technique.

statistically significant difference was observed between patients with a concomitant hysterectomy and/or partial vaginectomy, and those without concomitant resection ($P < .001$). Although 5 patients who underwent a hysterectomy with closure of the vaginal stump did not develop RVF, 10 of the 21 (47.6%) patients who underwent a partial vaginectomy developed RVF. There was a statistical difference between hysterectomy and partial vaginectomy ($P = .0491$). RVF was found in 9 of the 79 (11.4%) patients in whom a diverting ostomy had already been established at the initial operation. The remaining 7 patients of the 16 patients with RVF received the diverting ostomy when RVF was confirmed. There were no statistical differences between the established diverting ostomy group and the group that did not undergo a diverting ostomy at the initial operation ($P = .545$).

The factors associated with higher rates of RVF were not seen in any preoperative co-morbid diseases (eg, diabetes and vascular disease).

Two patients did not undergo closure of the diverting ostomy due to death from metastatic tumor progression in the follow-up period. Six patients with RVF arising from anastomotic leakage recovered completely by undergoing only a diverting ostomy within 6 months postoperatively.

Table III. Details of vaginal surgery at initial operation

	RVF(+)	*RVF(−)*
Hysterectomy with closure of vaginal stump	0 (0%)	5
Partial vaginectomy	10 (47.6%)	11

RVF, Rectovaginal fistula.

Operative treatment for RVF was performed in 8 patients because a diverting loop ostomy alone did not heal the RVF. Two patients had RVF arising from partial resection of the vaginal wall in the circular stapler (1 after LAR, 1 after vLAR), and 6 had RVF arising from anastomotic leakage and abscess drainage to the vagina after a concomitant vaginal wall resection (1 after LAR, 3 after vLAR, and 2 after ISR).

The RVF healed in 1 patient who underwent re-vLAR with establishment of a diverting ileostomy. Although 1 patient underwent a transanal fistelectomy and direct closure, the RVF relapsed after closure of the diverting colostomy. A second patient underwent a diverting ileostomy; she recovered completely 2 years later. Two patients underwent repair of an endovaginal advancement flap. One patient in whom RVF arose from partial resection of the vaginal wall by double stapled anastomosis recovered after repair of the fistula with an endovaginal advancement flap 3 months postoperatively. However, in the other patient in whom the RVF arose from anastomotic leakage, the RVF relapsed despite the endovaginal advancement flap.

A gluteal-fold flap repair was performed in 5 patients who developed RVF after rectal cancer operation (Table IV): 1 from inclusion of the vaginal wall in a double stapled anastomosis, 2 from anastomotic leakage after DST in an anterior resection, and 2 from anastomotic leakage after a coloanal anastomosis with hand sewing in an ISR. The RVF was located in the higher position of the vagina in 2 patients (patients 2, 3), the middle position in 1 (patient 1), and the lower position in 2 (patients 4, 5). These 5 patients were followed for 137 to 880 days. One underwent repair of an endovaginal advancement flap; however, the RVF did not heal (patient 1).

One patient who underwent repair of an endovaginal advancement flap did not have a defunctioning ostomy constructed (patient 1), while in

Table IV. Clinical background of patients with RVF who underwent gluteal-fold flap repair

Patient	Age	TMN stage	Operation/anastomosis technique	Diverting ostomy at first operation	Onset of RVF (POD)	Position of RVF	Cause of RVF
1	65	T3N0M0	vLAR/DST	+	4	Mid	Leakage
2	56	T3N1M0	vLAR/DST	–	21	High	Leakage
3	63	T3N2M0	LAR/DST	–	20	High	Involvement*
4	47	T3N0M0	ISR/hand sewing	+	15	Low	Leakage
5	53	T3N2M0	ISR/hand sewing	+	7	Low	Leakage

RVF, Rectovaginal fistula; *POD*, postoperative day; *vLAR*, very low anterior resection; *LAR*, low anterior resection; *DST*, double stapling technique; *ISR*, intersphincter resection.
*Involvement: RVF arose from inclusion of the vaginal wall in a double stapled anastomosis.

Table V. Outcomes of gluteal-fold flap repairs

Patient	Interval from operation (d)	Duration of operation for internal pudendal artery flap (min)	Size of flaps (cm)	Interval from flap operation to ostomy closure (d)	Recurrence of RVF
1	880	60	13.0 × 4.0	—	—
2	350	100	15.0 × 4.5	229	—
3	154	105	12.0 × 3.5	147	—
4	381	120	10.5 × 3.0	95	—
5	137	30	8.0 × 3.0	110	—

RVF, Rectovaginal fistula.

the remaining 4 patients (patients 2-5) a defunctioning loop ostomy (2 ileostomies, 2 transverse colostomies) was constructed when a gluteal-fold flap repair was performed.

The outcomes of the gluteal-fold flap repairs are shown in Table V. The average operation time for flap repair and/or ostomy construction was 83 minutes (range, 30-120 minutes). The size of the flaps ranged from 12.0 × 3.5 cm to 15.0 × 4.5 cm for the patients with RVF situated at the higher end or middle of the vagina (patients 1-3), and from 8.0 × 3.0 cm to 10.5 × 3.0 cm for the patients with RVF situated lower in the vagina (patients 4, 5). No complications, such as necrosis of the flaps, bleeding, infection, or pain, were noted. Diverting ostomies were closed 95 to 229 days after operative treatment. None has failed to heal, and no RVFs have relapsed after more than 1 year postoperatively (Fig 5).

Two patients were able to have sexual intercourse postoperatively. No patients experienced incontinence, rectal stricture, feeling of wrongness, or pain of the external genitals when seated.

DISCUSSION

RVF may occur as a complication in female patients undergoing operative treatment for lower rectal carcinoma. The incidence of RVFs after anterior resections has been reported to be 0.9 to 2.9 percent.[3,5,14] This complication leads to vaginal flatus, fecal leakage, and/or vaginal discharge, and is uncomfortable. In the present study, anastomosis by DST in patients undergoing LAR or vLAR, ISR, and concomitant hysterectomy and/or resection of the vaginal wall were significantly different causes of RVFs.

Although we inserted 1 or 2 drains into the posterior and/or anterior side of the colorectal anastomosis, abscess drainage into the vagina resulted in RVF in 10 patients with surgical damage of the vaginal wall opened intraoperatively. Therefore, although 8 of the 10 patients with RVF had the diverting ostomy established at the initial operation, it was impossible to prevent RVF.

A function-preserving operation for pelvic malignant disease has been attempted at our institute. Total pelvic excenteration (TPE) has been advocated as the best treatment for locally advanced rectal cancer involving the urogenital organs. As a result, patients sometimes require a double stoma for urinary and fecal diversion. However, an extended colorectal resection with preservation of partial bladder and/or anal sphincter is sometimes possible when cancer-free margins can be obtained. Thus, we designed a function-preserving operation to avoid TPE or posterior pelvic excenteration, such as ISR for low rectal cancer,[15] neourinary bladder reconstruction using the small intestine for patients with rectal cancer invading the urinary tract, and partial resection of the

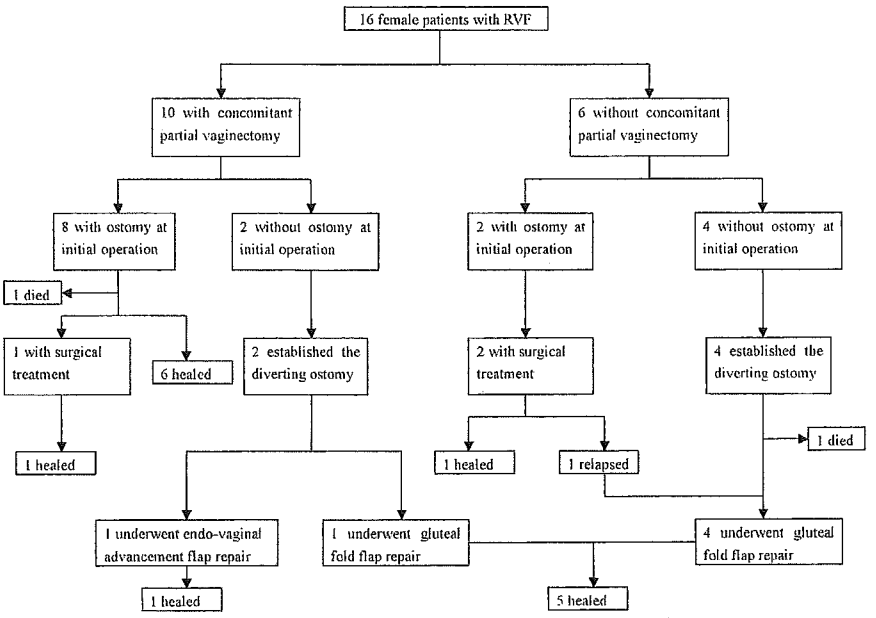

Fig 5. Outcomes of operative treatments for rectovaginal fistula (RVF).

vaginal wall for patients with rectal cancer invading the vaginal wall. This preserving surgery concept was considered to cause a high rate (9.9%) of RVF. Thus, the occurrence of postoperative RVF should be taken into account if the vaginal wall is resected during surgery for lower rectal cancer. For prevention of postoperative RVF in patients with partial resection of the vagina, surgeons need to practice some preventive measures (eg, omental interposition[16,17] or rectus abdominis muscle[30]).

Significant differences in the occurrence of RVF were observed between the DST and SST anastomotic procedures in the present study. In 1979, Nance[18] described an end-to-end ileocolostomy using a circular stapler to cut through a liner stapler line. This procedure has expanded and developed into the DST of colorectal anastomoses. Postoperative complications related to the DST are anastomotic leakage, stricture, and RVF.[3] DST involving the posterior wall of the vagina in a circular stapled anastomosis and anastomotic leakage cause RVF in rectal cancer. Nakagoe et al[19] reported a technique for preventing RVF after LAR using the DST. However, in the present study, 2 patients developed RVF due to inclusion of the vaginal wall in a double stapled anastomosis. Furthermore, 6 patients with postoperative RVF caused by anastomotic leakage were healed within 6 months by undergoing diverting ostomy alone; however, the patients who developed RVF due to involvement of the posterior wall of the vagina in the circular stapled anastomosis never healed after undergoing diverting ostomy alone even after a long period of time. We suggest that these patients in whom RVF developed as a result of inclusion of the vaginal wall in the stapling technique require an operation to repair the RVF.

The surgical management for RVF patients is difficult and often unsatisfactory. Rex et al[5] reported a high success rate (10 of 14 patients who were treated conservatively) for spontaneous closure of the fistula with low-residual diets and antibiotics or bowel rest, whereas simple diverting colostomy resulted in a low rate of successful spontaneous healing (6 of 17 patients). Similarly, in the present study, spontaneous closure of the fistula with simple diverting ostomy occurred infrequently (6 of 14 patients, excluding 2 patients who underwent gluteal-fold flap repair). Indeed, in the present 3 patients who underwent gluteal-fold flap repair, simple diverting ostomy failed to cause spontaneous closure after more than 137 days. Patients whose RVF did not heal 3 months after establishment of a diverting ostomy alone underwent operative treatment for RVF.

Repair with local procedures such as an endorectal advancement flap produces good or excellent results for the simple fistula.[20-22] However, local repair of complex fistulas fail to heal more commonly.[9,23-25] For postoperative patients with lower rectal cancer, we believe that an endorectal advancement flap is not an efficient method

technically because it is difficult to advance colonic mucosa of the oral side beyond the anastomosis.

Various surgical procedures for treatment of RVF have been reported. These procedures can be performed by abdominal, rectal, vaginal, perineal, transsphincteric, or transsacral approach.[7,14,26-30] Many different reconstructive techniques with various flaps also have been reported; however, these surgical procedures can result in incontinence, failure to heal, and the need for a laparotomy.

In 1996, Yii et al[13] reported on lotus petal flaps, which are raised on perforators around the perineum to resemble the petals of the lotus and can be used to reconstruct a variety of vulvovaginal defects. They described the gluteal-fold flaps as good flap donors because the perineum is an area of rich blood supply with multiple arterial anastomoses. Indeed, although an incision was made on the entire flaps in our patients, the flaps showed good mobility and no subsequent necrosis. In addition, Haray et al[31] reported a procedure involving a diamond-shaped, cutaneous flap advancement for patients with recurrent RVF.

Our procedure is a modification of Yii's and Haray's methods, as a diamond-shaped cutaneous flap advancement cannot be raised to a higher position RVF. Indeed, the RVF location of 2 of our patients (patients 1, 2; Table IV) were in a higher position in the vagina, and the size of the gluteal-fold flaps were longer than those of the lower position.

The gluteal-fold flap has been considered a fasciocutaneous flap. Hashimoto et al[32] reported that direct cutaneous vessels supplied blood flow and that it was easy to adjust the flap volume by resecting the fatty tissue except around the pedicles. This thinning of fatty tissue in the subcutaneous area and a rare growth of hair is suitable for flaps inserted into the vagina. This procedure is good for female patients because the gluteal fold renders the surgical wounds inconspicuous. Indeed, in the present study, no major complications accompanied gluteal-fold flap repair postoperatively.

CONCLUSION

The incidence of RVF was high in patients who underwent ISR, anastomosis by DST and hand sewing in anterior resection, and concomitant hysterectomy and/or resection of the vaginal wall regardless of the establishment of a diverting ostomy. Although some RVFs heal with only fecal diversion, patients whose RVF was caused by involvement of the vaginal wall in a circular stapling or ISR procedure obtain good results with the gluteal-fold flap repair technique. We suggest that a gluteal-fold flap repair is sufficiently wide and long to ensure adequate vascular supply and good mobility, and is a reliable surgical treatment for patients with RVF.

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