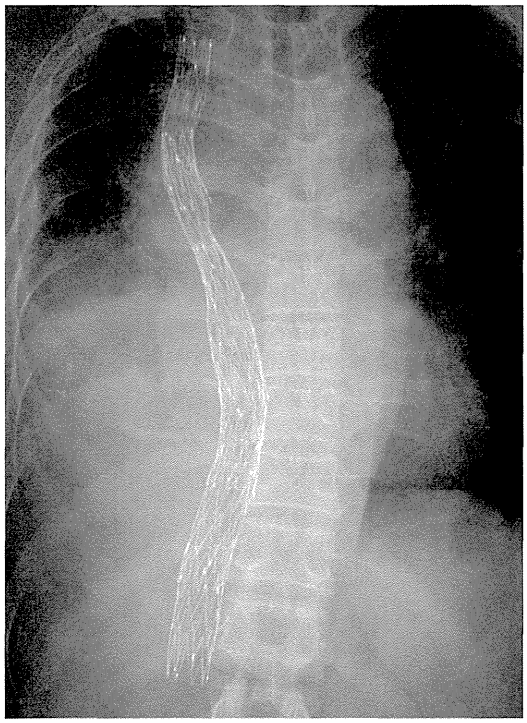

Fig. 3 Bridging stent. Five 20-mm-diameter, 80-mm-long self-expandable metallic stents, and one 20-mm-diameter, 60-mm-long self-expandable metallic stent deployed in tandem with enough overlap from the right brachiocephalic vein to the inferior vena cava using a guide wire that was inserted from the right common femoral vein to the right internal jugular vein with use of a pull-through technique

and an insufficient outline of the RA (not shown). A 4F catheter (Fansac II; Terumo-Clinical Supply, Gifu, Japan) was inserted into the IVC from the right IJV. The catheter was captured by a 12–20-mm snare (EnSnare; Merit Medical, South Jordan, UT, USA) in the IVC and was pulled out via a 12F sheath. A 0.035-inch guide wire (Amplatz Extra Stiff Wire Guide; William Cook, Bjaeverskov, Denmark) was inserted from the right common femoral vein to the right IJV; so-called pull-through was achieved [4].

After injection of 3,000 U of heparin, five 20-mm-diameter, 80-mm-long self-expandable metallic stents (Spiral Z stent; Medicos Hirata, Osaka, Japan) and one 20-mm-diameter, 60-mm-long self-expandable metallic stent were deployed in tandem from the right BCV to the IVC (Fig. 3) in the same manner as Nagata et al. [3]. We also checked the positional relationship between vertebra and venous structures on MRI, CO_2-venography and fluoroscopy. Postprocedural CO_2-venography confirmed excellent blood flow from the right BCV and IVC to the RA through the stents (Fig. 4). The pressure gradient between the SVC and the RA decreased from 42 to 5 cm of water. Symptoms of SVC syndrome improved quickly after stent placement. Anticoagulation therapy (10,000 IU/day of heparin) was continued for 1 day after the procedure and then discontinued. The patient was discharged 2 days after the procedure, and ultimately died of his primary disease 30 days later without recurrence of the SVC syndrome.

Discussion

Stent migration is a rare but potentially lethal complication of SVC stent placement [2]. The reported frequency of stent migration is 0.7–2.0 % [1, 5]. These data were collected in the setting of ICM use. When the use of ICM is contraindicated, visualization of the detailed anatomy may be difficult, and the risk of stent migration may increase even if CO_2-venography was used.

Placement of multiple stents in tandem through the SVC to the IVC could be safe and clinically useful, even in patients with ICM contraindications. Taylor et al. [2] introduced this method, named as bridging stent, to rescue stent migration from the IVC to the RA. Our report was the first to describe the successful placement of an SVC stent without the use of ICM venography. Sato et al. [6] deployed stents in the same manner for the treatment of IVC syndrome with RA involvement. SVC or IVC syndrome with RA involvement could also be successfully treated using a bridging stent.

Gadolinium contrast material may be useful to reveal stenosis. However, venography of a large vein such as the vena cava would need a large amount of contrast medium. Several bottles of gadolinium contrast material would be needed because content of commercialized gadolinium contrast material does not exceed 20 ml in Japan. Renal dysfunction may lead to the nephrogenic systemic fibrosis [7]. In our case, a bridging stent could be safely deployed with an effective combination of CO_2-venography, enhanced MRI, and positional relationship between vertebra and venous structures on fluoroscopy.

There could be some disadvantages of a bridging stent. A bridging stent could be expensive because of multiple stents. Risk of vascular injury might increase as numbers of stent delivery increase. Unknown adverse effects could exist.

Another method to reveal stenosis might be balloon dilation prior to stent placement [8]. To delineate the detailed anatomy, dilation using a sufficiently large balloon is required, but this might result in rupture of SVC [9]. Balloon rupture, which rarely occurs, might cause anaphylactic shock owing to ICM in the balloon in a patient with an ICM allergy when diluted ICM is used for inflating the balloon [10].

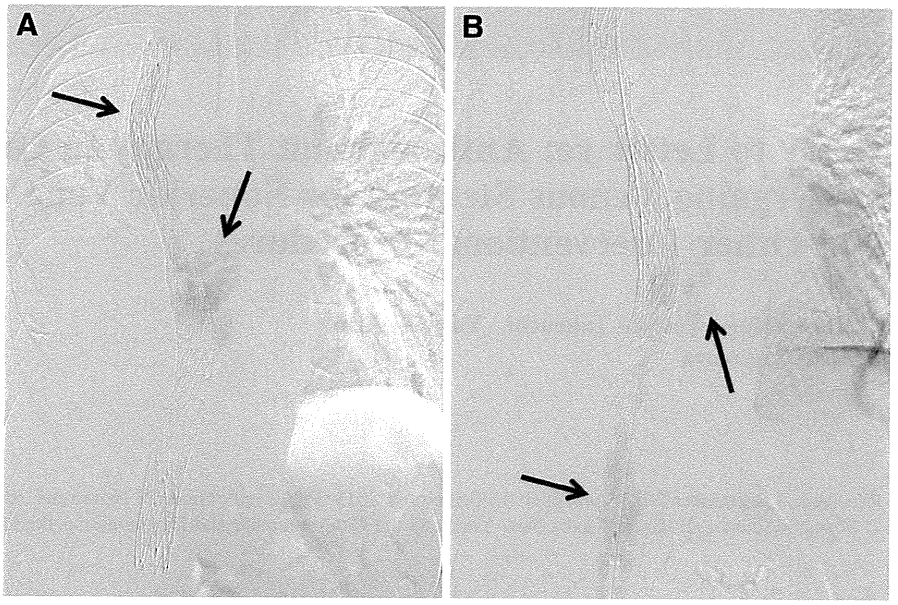

Fig. 4 Digital subtraction venography using carbon dioxide (CO_2-venography) after stent placement. CO_2-venography from the internal jugular vein (**a**) and the inferior vena cava (**b**) shows good flow into the right atrium through the mesh of stents. *Arrows* indicate CO_2

In conclusion, here we successfully treated a case of malignant SVC syndrome using an SVC stent in a patient with an ICM allergy. Secure stent placement was achieved using the bridging stent technique through the SVC to the IVC without the use of ICM venography.

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Conflict of interest We have nothing to disclose.

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CIRSE

LETTER TO THE EDITOR

Reply to Letter re: Anticoagulant Therapy in Oncologic Patients Undergoing Venous Stenting for Superior Vena Cava Syndrome and Other Interventional Procedures

Keitaro Sofue · Yoshito Takeuchi · Yasuaki Arai · Kazuro Sugimura

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To the Editor,

We thank Iaccarino et al. for their interest in our case report [1], which mentioned recanalization and restenting for acute occlusion of superior vena cava (SVC). Anticoagulation therapy with heparin was administered for 7 days, followed by warfarin sodium after discharge from the hospital.

The association of vascular thrombosis in patients underlying malignancy has been known since Trousseau reported it in 1865 [2]. One published study with a large population demonstrated that oncologic patients have a sevenfold increased risk of developing venous thrombosis compared with those without cancer [3]. During the last few decades, this has become of increasing interest: the number of cancer patients with advanced-stage disease, high tumor volume, and lengthy hospitalization has recently increased as a result of recent therapeutic developments. Several factors, including altered immune response, production of abnormal proteins, and cancer cells, affect the prothrombotic or hypercoagulative state, and these conditions further increase after endovascular procedures.

The necessity of long-term anticoagulant therapy remains unclear, and hemorrhagic complications are an unresolvable dilemma [4, 5], although we also think that antithrombotic therapy was not useful to prevent venous thromboembolism, as described in the letter. However, long-term anticoagulation therapy with warfarin sodium was applied in our case because restenting for acute thrombotic occlusion of SVC was necessary on the fifth day after the initial stent placement.

The American Society of Clinical Oncology (ASCO) guidelines reported an evidence-based clinical practice on prophylaxis and treatment of venous thromboembolism in patients with cancer [6]. ASCO recommends the utilization of low-molecular-weight heparin (LMWH) because LMWH has several advantages over unfractionated heparin and warfarin sodium, including dose-dependent plasma levels, long action, and lower bleeding risk [7]. Undoubtedly LMWH is the best option for the prevention of venous thrombosis after the endovascular procedure. However, we did not use LMWH after the treatment of the SVC stent because in our country, LMWH was available only for prophylaxis of deep venous thrombosis after orthopedic surgery of a lower limb, after abdominal surgery, during hemodialysis, or for the treatment of disseminated intravascular coagulation. Moreover, LMWH costs more than unfractionated heparin and warfarin sodium, and as outpatients, patients have to visit a hospital to receive subcutaneous treatment once or twice a day.

Although the provision of LMWH after treatment of the SVC stent is desirable for its convenience and lower hemorrhagic risk, we could not use it because it was not approved for this use and because it is costly. Further studies might to be conducted to evaluate the utility of

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LMWH for the prevention of venous thromboembolism and its safety in oncologic patients undergoing interventional procedures.

Conflict of interest The authors declare that they have no conflict of interest.

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ORIGINAL ARTICLE – COLORECTAL CANCER

Selective Lateral Pelvic Lymph Node Dissection in Patients with Advanced Low Rectal Cancer Treated with Preoperative Chemoradiotherapy Based on Pretreatment Imaging

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ABSTRACT

Background. The significance of lateral pelvic lymph node (LPLN) metastasis in advanced low rectal cancer treated with preoperative chemoradiotherapy (CRT) remains unclear. The objective of this study was to evaluate the outcomes of selective LPLN dissection (LPLD) based on the pretreatment imaging in patients with advanced low rectal cancer treated with preoperative CRT.

Methods. We reviewed 127 consecutive patients with clinical stage II–III low rectal cancer below the peritoneal reflection who underwent preoperative CRT and curative resection. LPLD was performed in patients with suspected LPLN metastasis based on MDCT or MRI before CRT (LPLD group, $N = 38$), and only total mesorectal excision (TME) was performed in patients without suspected LPLN metastasis (TME group, $N = 89$). Clinical characteristics and the oncological outcome were compared between groups.

Results. The median tumor-to-anal verge distance was 40 mm in both groups. The median maximum long-axis LPLN diameter before CRT was 0 mm in the TME group and 10.5 mm in the LPLD group. Pathological LPLN metastasis was confirmed in 25 patients (66 %) in the LPLD group. Local recurrence at LPLN developed in 3 patients (3.4 %) in the TME group and in none (0 %) of the LPLD group. Multivariate analysis showed that only ypN was an independent prognostic factor for relapse-free survival (RFS), but LPLN metastasis was not associated with poor RFS.

Conclusions. The incidence of LPLN metastasis is high even after preoperative CRT, and LPLD might improve local control and survival of patients with LPLN metastasis in advanced low rectal cancer treated with preoperative CRT.

For patients with locally advanced (stage II–III) rectal cancer, preoperative chemoradiotherapy (CRT) and total mesorectal excision (TME) is the widely accepted standard treatment. Randomized, clinical trials have indeed shown that fluorouracil-based preoperative CRT reduced local recurrence compared with preoperative radiation without chemotherapy or postoperative CRT.[1–3] In Japan, the Japanese Society for Cancer of the Colon and Rectum (JSCCR) guidelines 2010 for the treatment of colorectal cancer recommend lateral pelvic lymph node (LPLN) dissection (LPLD) for advanced low rectal cancer that extends below the peritoneal reflection because of possible LPLN metastases.[4] The incidence of LPLN metastasis has been demonstrated to be approximately 15 % in patients with advanced low rectal cancer who underwent LPLD.[5–8] However, previous studies from Japan regarding LPLN metastasis adopted TME and LPLD without preoperative

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CRT. In western countries, surgeons do not perform LPLD regularly because preoperative CRT without extended surgery can provide acceptable local control and LPLN metastasis is generally considered a metastatic disease that is not amenable to surgical cure.[9–11] Thus, there are very limited studies about the incidence of LPLN metastasis and oncologic outcome of patients with LPLN metastasis in patients who underwent standard preoperative CRT.

The objective of the present study was to assess the oncologic outcome for an institutional cohort of 127 consecutive patients with advanced low rectal cancer below the peritoneal reflection who received preoperative CRT and selective LPLD based on the pretreatment imaging.

MATERIALS AND METHODS

Study Population

Between July 2004 and December 2010, 141 patients with biopsy-proven, locally advanced (cT3-4 or cN+) low rectal cancer were treated at our institution with long-course preoperative CRT followed by surgery. Ten patients who had distant metastases at surgery, two patients who underwent induction systemic chemotherapy before CRT, and two patients who underwent noncurative resection were excluded from the present study. As a result, 127 patients were included in the present study.

Clinical Staging and Treatment Strategy

Pretreatment clinical stage was assessed based on MDCT or MRI. Low rectal cancer is defined as a tumor of which the inferior border is located below the peritoneal reflection, which is equivalent to 8 cm from the anal verge. The level of the peritoneal reflection was assessed by barium enema (the middle Houston's valve as corresponding to the peritoneal reflection) and/or sagittal MRI before CRT. Patients were treated with 5-fluorouracil-based CRT with a total dose of 45 Gy (115 patients) or 50.4 Gy (12 patients). The lateral pelvic area was usually included in the radiation target volume. Surgery was performed 4–8 weeks following completion of preoperative CRT. If preoperative MDCT or MRI before CRT showed unilateral swollen LPLNs, unilateral LPLD were performed regardless of the imaging after CRT (LPLD group, $N = 38$). If preoperative imaging showed bilateral swollen LPLNs, bilateral LPLD was performed. If no swollen LPLNs were detected, only standard TME was performed (TME group, $N = 89$). The swollen LPLNs were generally considered to measure 7 mm or larger, but the final indication for LPLD was determined at the multidisciplinary team meetings. The basic indication for adjuvant chemotherapy at our institution was pathologically node-positive disease, but adjuvant chemotherapy was also delivered to some pathologically node-negative patients at the discretion of the surgeon and oncologist.

Lateral Pelvic Lymph Node Dissection

The procedure of LPLD (open or laparoscopic) is standardized in our institution.[7,12] The lateral pelvic area is classified into four regions (internal iliac, obturator, external iliac, and common iliac) according to the JSCCR classification (Second English Edition).[13] However, we do not usually dissect the common iliac lymph nodes unless metastasis to this region is suspected, because the frequency of metastasis to this area is rare.[6] The extent of LPLD is shown in Fig. 1A. The lateral, medial, cranial, caudal, and dorsal anatomical borders of LPLD are the external iliac artery, pelvic plexus, bifurcation of the common iliac artery, levator ani muscle, and sciatic nerve, respectively. Whether to resect the internal iliac artery or its branching artery, such as the umbilical, superior or inferior vesical, or obturator artery en bloc with LPLN, is at the surgeon's discretion, but we usually resect the adjacent artery if invasion by the metastatic LPLN is suspected. The pathologic stage (ypT and ypN) was determined according to the seventh edition of the *American Joint Committee on Cancer (AJCC) Cancer Staging Manual.*[14] LPLNs other than internal iliac lymph nodes are not clearly defined as regional lymph nodes in AJCC, but we classified all LPLNs as regional lymph nodes in the present study.[5]

Size of LPLNs on MDCT

All patients underwent MDCT with 16 detectors of the abdomen and pelvis (LightSpeed; GE Medical Systems, Milwaukee, WI) without any preparation. Contrast medium (Iopamiron; Bayer, Osaka, Japan) was administered intravenously and the slice interval was adjusted to 5 mm. Sequences were downloaded onto a workstation (HOPE/EGMAIN-EX, Fujitsu, Tokyo, Japan). All images were retrospectively reviewed by one colorectal surgeon (T.A.; R1) experienced in LPLD and the anatomy of lateral pelvis and two experienced gastrointestinal radiologists (T.U.; R2, A.K.; R3), who were blinded to the clinical information of the patients. We measured the maximum long-axis and corresponding short-axis diameter of the LPLN after the bifurcation of the common iliac artery with electronic calipers on digitized images before and after CRT (Fig. 1B). To evaluate the interobserver reproducibility, agreement among the three observers was assessed using Spearman's correlation coefficient, calculated for each pair observed (three pair comparisons).

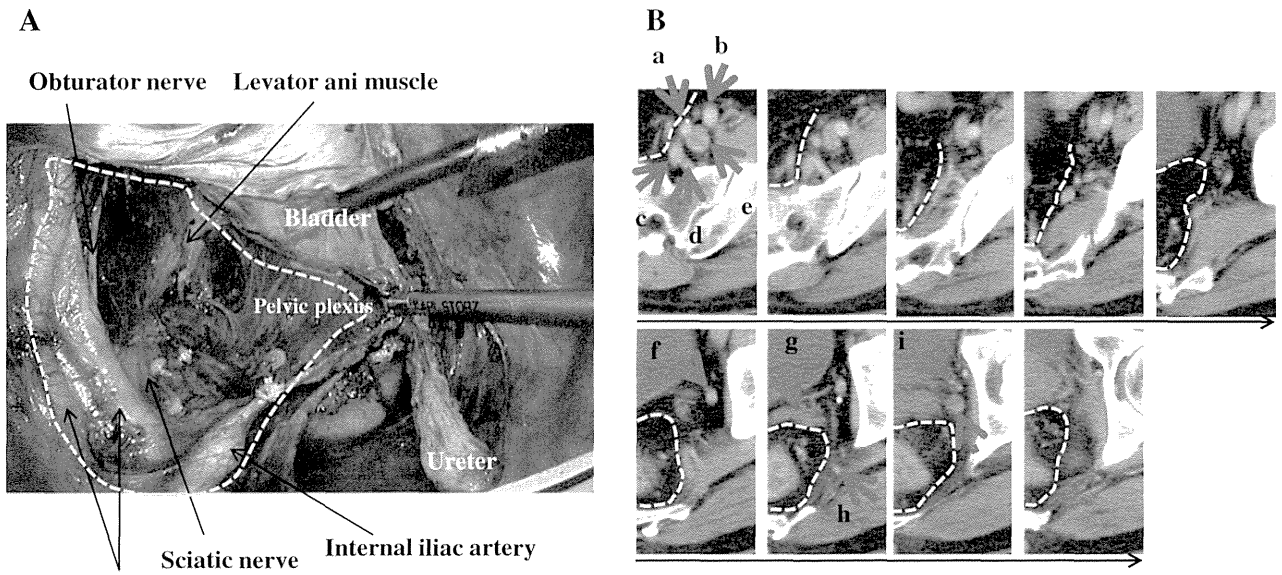

FIG. 1 **A** The surgical view after lateral pelvic lymph node dissection (LPLD). The extent of LPLD is shown by the *dashed yellow line*. The branching vessels from the internal iliac vessels are divided. **B** MDCT images of the left lateral pelvic area before preoperative chemoradiotherapy (CRT). The slice interval described here is 10 mm, although the actual slice interval was adjusted to 5 mm. (*a*) internal iliac artery, (*b*) external iliac artery, (*c*) internal iliac vein, (*d*) superior gluteal vein, (*e*) external iliac vein, (*f*) obturator vein, (*g*) seminal vesicle, (*h*) internal pudendal artery, (*i*) inferior vesical vessels (*dashed arrow*) swollen internal iliac lymph node. The *dashed yellow line* indicates the total mesorectal excision (TME) line

Statistical Analysis

Quantitative data were expressed by the median (range). Statistical analysis was performed using JMP software V 8.0.2 (SAS Institute, Cary, NC). Comparisons between the TME group and the LPLD group were made using the χ^2 test, Fisher exact test, or Mann–Whitney test. Survival analysis was performed using the Kaplan–Meier method with the log-rank test. Local recurrence was defined as any anastomotic, pelvic, or perineal tumor recurrence, which was radiologically, histologically, or clinically diagnosed. Calculation of local recurrence rates included patients who developed distant recurrence simultaneously or before local recurrence. Variables with a P value < 0.2 in a univariate analysis were further evaluated in a multivariate analysis using the Cox proportional hazard model to assess the predictors for survival. The results of Cox model analysis are reported using hazard ratios (HRs) and 95 % confidence intervals.

RESULTS

Patient characteristics are summarized in Table 1. LPLD was performed in 38 patients (29.9 %, LPLD group). The median distance of the tumor from the anal verge was 40 mm in both groups. Pathological LPLN metastasis was confirmed in 25 (65.8 %) patients in the LPLD group, in whom 13 patients (52 %) had no mesorectal lymph node metastasis. The percentage of ypN+ patients was significantly higher in the LPLD group than in the TME group (68.5 vs. 31.5 %), although the number of involved nodes was not significantly different. The rate of postoperative complication was not significantly different between groups.

The maximum long- and short-axis diameter of LPLN before and after CRT read by R1 was summarized in Fig. 2. The median long-axis diameter of LPLN before CRT was 10.5 mm (range 0–27) in the LPLD group and 0 mm (range 0–11) in the TME group. After CRT, the median long-axis diameter of LPLN in the LPLD group was decreased to 8 mm (range 0–24), but there were no patients in whom the LPLN completely disappeared. Spearman's correlation coefficients for measurements of the diameter of LPLN between the three observers were summarized in Supplementary Table 1. We found the strong interobserver correlation for measurements (Spearman's correlation coefficients = 0.964–0.993, $P < 0.0001$ for all correlations), and the long-axis diameter before CRT was the most highly correlated (mean Spearman's correlation coefficients = 0.992). Similarly, the median short-axis diameter of LPLN before CRT (8 mm, range 0–21) was decreased to 6 mm (range 0–21) after CRT in the LPLD group.

TABLE 1 Patient and tumor characteristics

Variables	TME group (n = 89)	LPLD group (n = 38)	P
Sex			0.8314
Male	62 (69.7%)	28 (73.7%)	
Female	27 (30.3%)	10 (26.3%)	
Age, year (range)	60 (34–81)	61 (35–75)	0.726
Distance of tumor from AV, mm (range)	40 (10–80)	40 (10–80)	0.8623
Clinical stage			
II	39 (43.8%)	0 (0%)	<0.0001
III	50 (56.2%)	38 (100%)	
Histological type			1.000
Well/moderate	79 (88.8%)	34 (89.5%)	
Mucinous/poor/signet	10 (11.2%)	4 (10.5%)	
Operative procedure			0.2165
Low anterior resection	29 (32.6%)	16 (42.1%)	
Intersphincteric resection	29 (32.6%)	5 (13.2%)	
Hartmann's procedure	3 (3.4%)	2 (5.3%)	
Abdominoperineal resection	27 (30.3%)	15 (39.5%)	
Total pelvic excenteration	1 (1.1%)	0 (0%)	
Temporary stoma	58/58 (100%)	20/21 (95%)	0.2658
LPLD			<0.0001
Unilateral	0 (0%)	31 (81.6%)	
Bilateral	0 (0%)	7 (18.4%)	
Laparoscopy	70 (78.7%)	22 (57.9%)	0.0288
No. of lymph nodes removed	14 (6–26)	23.5 (12–37)	<0.0001
ypT			0.374
ypT0	9 (10.1%)	2 (5.3%)	
ypT1	3 (3.4%)	3 (7.9%)	
ypT2	29 (32.6%)	8 (21.1%)	
ypT3	46 (51.7%)	23 (60.5%)	
ypT4	2 (2.3%)	2 (5.3%)	
ypN			0.0005
ypN0	61 (68.5%)	12 (31.6%)	
ypN1	17 (19.1%)	18 (47.4%)	
ypN2	11 (12.4%)	8 (21.1%)	
Pathological LPLN metastasis	0	25 (65.8%)	<0.0001
Without mesorectal LN metastasis		13 (52.0%)	
With mesorectal LN metastasis		12 (48.0%)	
No. of involved lymph nodes	3 (1–9)	2 (1–9)	0.2657
Circumferential resection margin			0.1578
Positive	2 (2.3%)	3 (7.9%)	
Negative	87 (97.7%)	35 (92.1%)	
Lymphovascular invasion	54 (60.7%)	31 (81.6%)	0.0243
Postoperative complication	26 (29.2%)	14 (36.8%)	0.411
Anastomotic leakage	2 (2.3%)	1 (2.6%)	
Perineal wound infection	12 (13.5%)	8 (21.1%)	
Abdominal wound infection	3 (3.4%)	2 (5.3%)	
Bowel obstruction	6 (6.7%)	1 (2.6%)	
Urinary retention	0 (0%)	2 (5.3%)	
Stoma-related complication	2 (2.3%)	1 (2.6%)	
Enteritis	2 (2.3%)	0 (0%)	
Brain infarction	1 (1.1%)	0 (0%)	
Adjuvant chemotherapy	31 (34.8%)	23 (60.5%)	0.0105
Capecitabine	17 (54.8%)	9 (39.1%)	
5-FU/LV	0 (0%)	1 (4.3%)	
5-FU/LV/oxaliplatin	6 (19.4%)	10 (43.5%)	
UFT/leucovorin	8 (25.8%)	3 (13%)	

AV anal verge, *LPLD* lateral pelvic lymph node dissection, *LPLN* lateral pelvic lymph node, *LN* lymph node, *LV* folinic acid, *UFT* tegafur-uracil

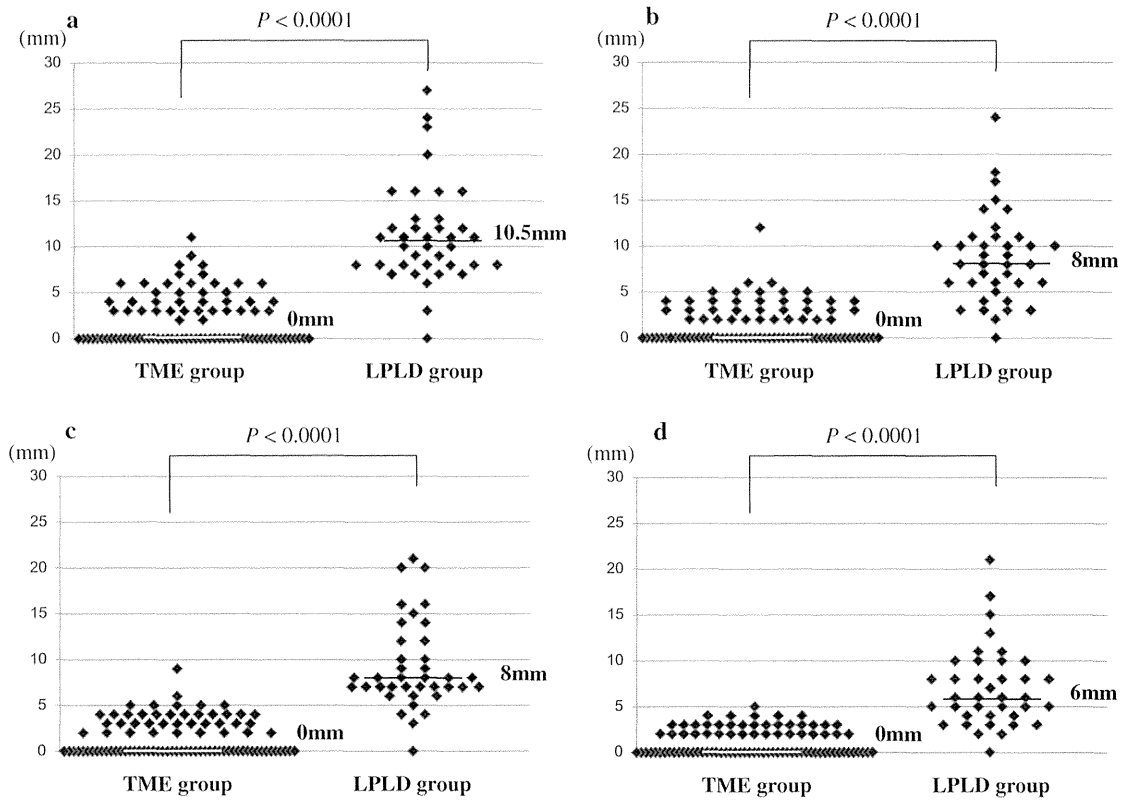

FIG. 2 Maximum diameter of the lateral pelvic lymph node by MDCT. Long-axis diameter before (**a**) and after (**b**) chemoradiotherapy (CRT), and short-axis diameter before (**c**) and after (**d**) CRT. Each *plot* indicates each patient. The *horizontal line* indicates median size

The median follow-up duration was 47.5 months (range 3.5–105.4) for the entire cohort. Of the 127 patients, local recurrence was developed in 7 patients in the TME group and one patient in the LPLD group (Supplementary Table 2). For the entire cohort, the 3-year local recurrence rate was 5.8 %. The 3-year local recurrence rates were 7.1 % in the TME group and 2.7 % in the LPLD group ($P = 0.2668$; Fig. 3b). The local recurrence at LPLN developed in 3 patients (3.4 %) in the TME group, and none developed local recurrence at LPLN in the LPLD group.

For the entire cohort, the 3-year, relapse-free survival (RFS) was 77.4 %. The 3-year RFS was 74.6 % in the TME group and 83.8 % in the LPLD group ($P = 0.1315$; Fig. 3a). In the univariate analysis, operative procedures, ypT, ypN, and lymphovascular invasion were significant predictors of RFS (Table 2). Pathological LPLN metastasis was not a significant prognostic factor for RFS and local recurrence (Fig. 3c, d). In the multivariate analysis, only ypN remained an independent prognostic factor for RFS (Table 2).

DISCUSSION

Our present study surprisingly showed that the incidence of LPLN metastasis (20 % of the total patients) in patients who underwent preoperative CRT was similar to that of the previous reports in which patients underwent surgery alone.[5–8] We also showed that the LPLN metastasis itself was not a poor prognostic indicator after preoperative CRT and LPLD, suggesting that LPLN metastasis is a regional disease that is amenable to curative resection. To date, the present study is the largest study to analyze the incidence and oncologic outcome of LPLN metastasis in patients with advanced low rectal cancer treated with preoperative CRT and selective LPLD.

The present study has shown clearly that LPLN metastasis cannot be eradicated completely by preoperative CRT alone, because the rate of pathological LPLN metastasis was 66 % when we perform LPLD based on the size of LPLN before CRT. LPLN might be the sentinel node in some patients with advanced low rectal cancer, because 52 % of the patients with LPLN metastasis had no mesorectal lymph node metastasis. Furthermore, our data suggested that LPLD was effective to control local recurrence at LPLN, because there was no local recurrence at LPLN in patients with LPLN metastasis who underwent LPLD. In contrast, it also is clinically important whether patients without swollen LPLN on pretreatment imaging who underwent TME alone would not experience local

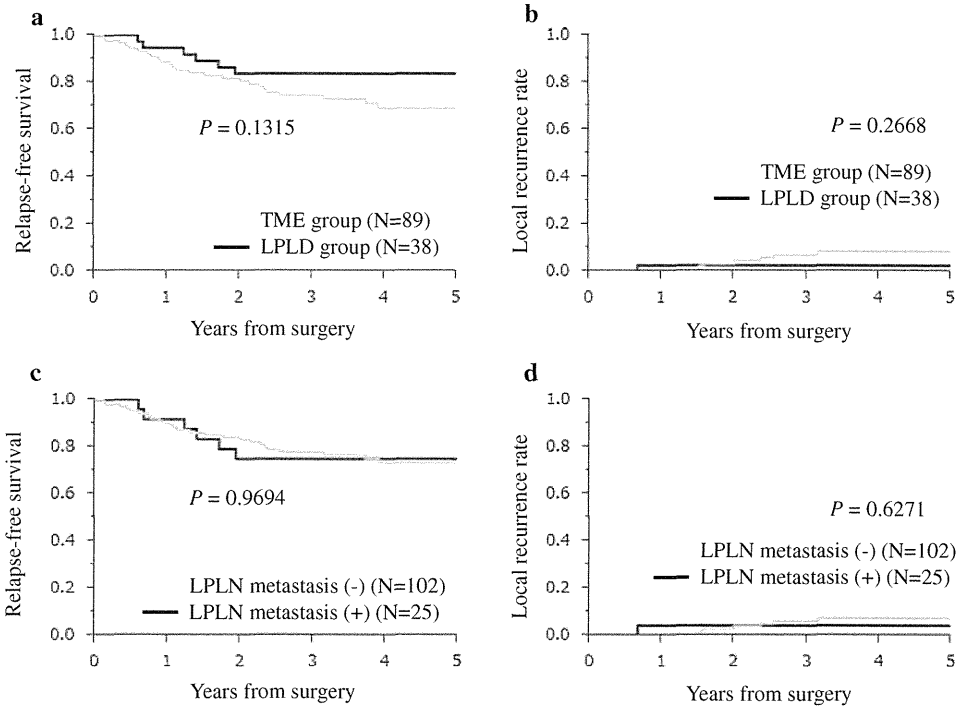

FIG. 3 Relapse-free survival (**a**, **c**) and local recurrence rate (**b**, **d**) of patients with and without lateral pelvic lymph node (LPLN) dissection (**a**, **b**) or LPLN metastasis (**c**, **d**)

recurrence at LPLN. The present study found that local recurrence at LPLN developed in 3 of 87 patients (3.4 %) who underwent TME alone, suggesting that pretreatment imaging might underdiagnose LPLN metastasis in some patients. However, relatively low incidence of LPLN recurrence in the TME group suggests that omitting LPLD based on the findings of pretreatment imaging is feasible if treated with preoperative CRT.

With regard to the oncologic outcome, many previous reports adopting surgery alone have shown that the survival of patients with LPLN metastasis was poorer than of those with only mesorectal lymph node metastasis.[5,7,8,15,16] The 5-year survival rate of patients with LPLN metastasis who were treated with LPLD without preoperative CRT was reported to be approximately 40–50 % and the 5-year local recurrence rate was 22–44%.[7,8,15,16] Interestingly, the present study found that survival and local recurrence rate of patients with LPLN metastasis were much better than those of the previous reports.[7,8,15,16] Our data suggest that the combination of preoperative CRT and LPLD can improve significantly the local control and even survival of patients with LPLN metastasis compared with surgery alone and that LPLN metastasis is a regional disease that can be equally cured as mesorectal lymph node metastasis. Significantly higher percentage of adjuvant chemotherapy in patients with LPLN metastasis (76 %) compared with patients without LPLN metastasis (34 %) also might contribute to good survival of patients with LPLN metastasis.

Our result might prompt the question whether LPLD is truly necessary to control local recurrence at LPLN for patients with pathological LPLN metastasis who underwent preoperative CRT. Kusters et al. analyzed 379 patients with low rectal cancer who underwent preoperative short-course radiation (25 Gy in five daily fractions) and TME alone in the Dutch TME trial and showed that only 3 patients (0.8%) developed local recurrence in the lateral pelvic subsite.[10] Although this study included 129 stage I patients who were at very low risk of developing LPLN metastasis, their results suggest that preoperative radiation might effectively control the LPLN metastasis and some patients with LPLN metastasis might be cured by preoperative CRT without LPLD. Furthermore, Watanabe et al. demonstrated that when preoperative radiotherapy was performed, TME without LPLD could provide similar oncological outcome compared with extended surgery without preoperative radiotherapy.[17] However, there are no data regarding the LPLN size on preoperative imaging in Kusters' and Watanabe's study. If there were no patients with swollen LPLN by preoperative imaging, TME without LPLD would be sufficient. Thus, we cannot conclude whether the percentage of the candidates of LPLD and actual LPLN metastasis is truly similar between our study and their study. In contrast, Kim et al. analyzed 366 patients with stage T3 or T4 tumors who underwent preoperative CRT and TME alone.[18] They showed that lateral pelvic recurrence was a major cause of local recurrence, and ypN+ and

TABLE 2 Univariate and multivariate analysis for relapse-free survival

Variables	Univariate analysis			Multivariate analysis		
	HR	95% CI	*P* value	HR	95% CI	*P* value
Sex			0.7539			
Male	1					
Female	1.128	0.511–2.323				
Age (year)			0.2611			
≤60	1					
>60	1.5	0.743–3.161				
Location of tumor from AV (mm)			0.9827			
40–80	1					
<40	1.008	0.499–2.125				
Clinical stage			0.2058			
II	1					
III	1.677	0.765–4.204				
Histological type			0.1384			0.2278
Well/moderate	1			1		
Mucinous/poor/signet	2.069	0.769–4.713		1.816	0.661–4.263	
Operative procedure			0.0105			0.1887
Sphincter preservation/Hartmann	1			1		
APR/TPE	2.496	1.243–5.063		1.638	0.783–3.462	
ypT			0.0007			0.1567
ypT0-2	1			1		
ypT3-4	3.925	1.725–10.55		1.944	0.787–5.607	
ypN			0.0002			0.0066
ypN0	1			1		
ypN+	3.937	1.901–8.745		2.858	1.331–6.586	
Pathological LPLN metastasis			0.9694			
Absent	1					
Present	1.018	0.378–2.31				
Lymphovascular invasion			0.001			0.2023
Absent	1			1		
Present	4.436	1.733–15.02		2.056	0.697–7.61	
Adjuvant chemotherapy			0.416			
No	1					
Yes	1.339	0.657–2.703				

AV anal verge, *APR* abdominoperineal resection, *TPE* total pelvic excenteration, *LPLN* lateral pelvic lymph node

the maximum lateral lymph node size before or after CRT were risk factors for lateral pelvic recurrence. Their results suggest that TME alone following preoperative CRT is not sufficient to control LPLN metastasis. The authors reported that lateral pelvic recurrence developed in 12.5 % (6/48) and 68.8 % (11/16) of patients with short-axis LPLN diameters of 5–9.9 and ≥10 mm, respectively.[18] In the present study, lateral pelvic recurrence developed in 3.4 % (1/29) and 0 % (0/12) of patients with short-axis LPLN diameters before CRT of 5–9.9 and ≥10 mm, respectively, even though the median follow-up period was longer in the present study (47.5 vs. 40.1 months). Although a simple comparison is difficult because of possibly different patient backgrounds between the studies, the better local control compared with previous reports strongly suggests that LPLD should be considered in patients with suspected LPLN metastasis even if patients were treated with preoperative CRT.

There are potential limitations of this study. The patient number is relatively small and our study included two

patients (1.6 %) who were followed for less than 2 years. Longer follow-up is necessary to evaluate the actual rate and pattern of local recurrence, because late local recurrence is not uncommon in patients who underwent preoperative CRT. Second, we did not evaluate other criteria, such as a spiculated border and the presence of mixed signal intensity by pretreatment MRI,[19] which might be useful to reduce the number of candidates of LPLD. Further studies are necessary to identify the most reliable imaging criteria of LPLN metastasis in patients treated with preoperative CRT.

CONCLUSIONS

The incidence of LPLN metastasis in patients with advanced low rectal cancer is high even after preoperative CRT when we perform selective LPLD based on the pretreatment imaging. LPLN metastasis can be treated with a multidisciplinary approach consisting of preoperative CRT and selective LPLD.

CONFLICTS OF INTEREST The authors indicate no potential conflicts of interest.

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Original article

Sinistral portal hypertension after pancreaticoduodenectomy with splenic vein ligation

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Background: Splenic vein ligation may result in sinistral (left-sided) portal hypertension and gastrointestinal haemorrhage. The aim of this study was to analyse the pathogenesis of sinistral portal hypertension following splenic vein ligation in pancreaticoduodenectomy.

Methods: Patients who underwent pancreaticoduodenectomy for pancreatic cancer between January 2005 and December 2012 were included in this retrospective study. The venous flow pattern from the spleen and splenic hypertrophy were examined after surgery.

Results: Of 103 patients who underwent pancreaticoduodenectomy with portal vein resection, 43 had splenic vein ligation. There were two predominant venous flow patterns from the spleen. In the varicose route (27 patients), flow from the spleen passed to colonic varices and/or other varicose veins. In the non-varicose route, flow from the spleen passed through a splenocolonic collateral (14 patients) or a spontaneous splenorenal shunt (2 patients). The varicose route was associated with significantly greater splenic hypertrophy than the non-varicose route (median splenic hypertrophy ratio 1·52 *versus* 0·94; $P < 0·001$). All patients with the varicose route had colonic varices, and none had a right colic marginal vein at the hepatic flexure.

Conclusion: Pancreaticoduodenectomy with splenic vein ligation may lead to sinistral portal hypertension. To avoid the development of varices, it is important to preserve the right colic marginal vein. Reconstruction of the splenic vein should be considered if the right colic marginal vein is divided.

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Introduction

In patients with cancer of the pancreatic head, tumour invasion of the portal vein (PV) or superior mesenteric vein (SMV) is common because of the close anatomical relationship of the pancreatic head and uncinate process to the portal system. Extensive portal venous resection with pancreaticoduodenectomy (PD) is well accepted for pancreatic head cancer because of the improvement in margin-negative resection and survival rates[1–3], without increasing postoperative morbidity and mortality in experienced centres[4–6].

When a tumour infiltrates the PV–SMV confluence, the splenic vein (SV) is sometimes ligated during PD with vascular resection to achieve a margin-negative resection. However, SV ligation may result in sinistral (left-sided) portal hypertension and gastrointestinal bleeding[1,7–18]. Some authors[9,15,18] have recommended reimplantation of the SV (SV–SMV anastomosis), whereas others[14,16] have reported that an SV–inferior mesenteric vein (IMV) anastomosis or preservation of a natural SV–IMV confluence may provide sufficient venous drainage of the spleen and gastric remnant. Misuta and colleagues[15] examined the direction of splenic venous flow in 29 patients who underwent PD with SV ligation, and decided to reconstruct an SV–IMV or SV–SMV anastomosis. Use of a temporary mesocaval shunt with a distal splenorenal shunt may facilitate segmental resection of the PV–SMV confluence during PD complicated by cavernous transformation of the PV[17]. In contrast, other authors[19–22] have reported that it is safe to divide the SV. In a small study[22], the pattern of collateral venous development after ligation of the SV was examined in five patients, and occlusion of the SV about

2 cm from its confluence with the SMV did not result in splenomegaly or symptoms.

Few reports have investigated why sinistral portal hypertension and gastrointestinal bleeding occur in PD with SV ligation. The aim of this study was to analyse the pathogenesis of sinistral portal hypertension following SV ligation in PD.

Methods

All patients who underwent PD for cancer of the pancreatic head or uncinate process between January 2005 and December 2012 at the authors' institution were included in this retrospective study. Before PD, abdominal contrast-enhanced multidetector-row CT and thoracic CT were performed to exclude distant metastatic disease. Patients with obstructive jaundice underwent preoperative endoscopic biliary drainage. Patient demographics, perioperative and postoperative details, and clinicopathological factors were collected retrospectively from patient charts.

After PD, each patient underwent a monthly physical examination and routine laboratory tests, including evaluation of tumour markers (carcinoembryonic antigen and carbohydrate antigen 19-9), and routine chest and abdominal thin-sliced (1–2 mm) contrast-enhanced CT every 6 months during the first year after surgery. If abnormalities were detected at the monthly follow-up, the patient underwent CT or other examinations before the routine CT scan at 6 months. As a result, CT was performed 4–8 months after the operation and the pattern of collateral development was evaluated using these CT images. Veins emanating from the spleen were traced carefully on serial transaxial or reconstructed three-dimensional images. Hepatic steatosis was also evaluated on CT images[23]. All images were reviewed retrospectively by two experienced gastrointestinal radiologists. Patients consented to the use of clinical data for research purposes when the operation was performed.

Splenic volumetry

Volumetric measurements of the spleen were carried out as reported previously[24]. Briefly, serial transverse intravenous contrast-enhanced CT images of the abdomen were obtained at intervals of 5·0 mm. On each slice of the preoperative and postoperative images, the splenic surface was traced with the cursor and spleen volume was calculated as volume per slice (surface area × thickness) × sum of the slices. The increase in splenic volume was expressed as the splenic hypertrophy ratio: volume measured 4–8 months after surgery/volume before surgery.

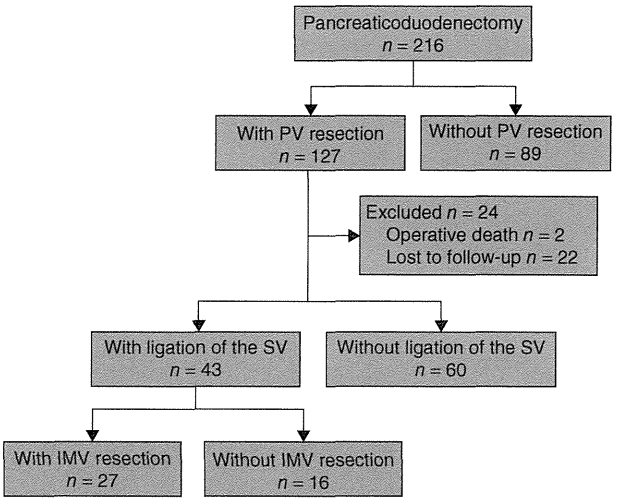

Fig. 1 Flow chart detailing patients who underwent pancreaticoduodenectomy. PV, portal vein; SV, splenic vein; IMV, inferior mesenteric vein

Surgical techniques

Adequate exploration and intraoperative ultrasonography were undertaken routinely during laparotomy to detect unsuspected peritoneal dissemination or liver metastases, and to assess resectability. Resection was performed in the absence of metastases and dissemination, and when no gross retroperitoneal tumour infiltration or complex artery infiltration was evident. As preoperative assessment regarding PV wall invasion is sometimes unreliable, departmental policy was to resect the PV in suspicious cases using a tumour no-touch isolation technique. In patients with borderline or locally advanced tumour[25], resection was not regarded as a contraindication if it was thought that cancer-free margins could be achieved even when the PV was occluded by cancer.

All patients underwent a standard PD with antrectomy and Child reconstruction. Extended lymph node dissection, encompassing the lymph nodes of the hepatoduodenal ligament along the common hepatic artery, coeliac trunk and right side of the superior mesenteric artery, was performed. The anastomosis between the SMV and PV was fashioned using a 5/0 non-absorbable running suture impregnated with a growth factor. In the event of PV–SMV confluence resection, the left gastric vein (coronary vein), right gastric vein, gastrocolic trunk of Henle and middle colic vein were divided with the SV. The SV was ligated approximately 1–3 cm to the left of its confluence with the SMV. When the IMV drained into the SMV or the splenomesenteric angle, the IMV was divided

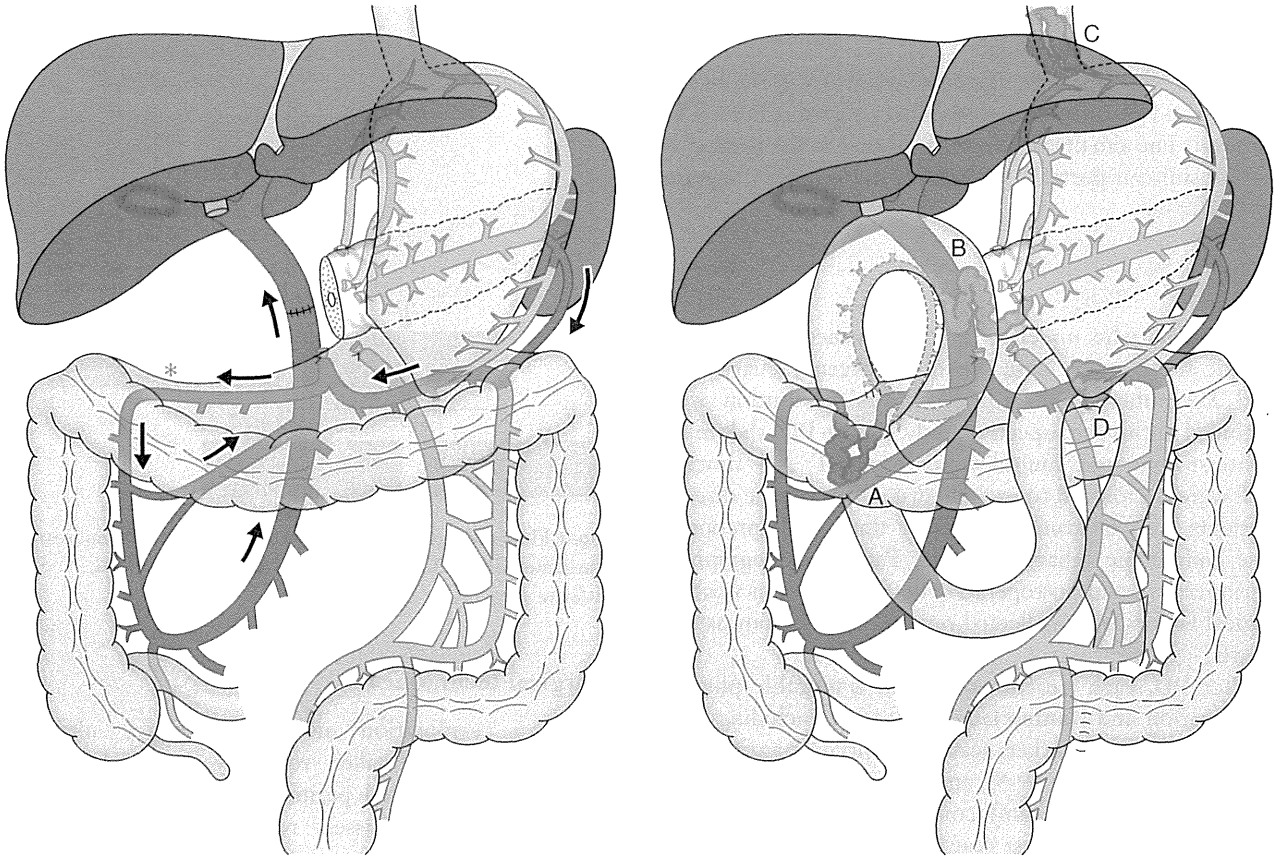

Fig. 2 *Venous flow from the spleen after portal vein–superior mesenteric vein confluence resection.* **a** The splenocolonic collateral was the main venous flow of the non-varicose route. The flow from the spleen passed through the arc of Barkow (first arrow from the spleen) to the right colic marginal vein (*), and then into the portal vein. Curved arrows indicate direction of blood flow. **b** Varices of the varicose route: A, colonic varices; B, pancreatojejunostomy varices; C, oesophageal varices; D, gastrojejunostomy varices

with the PV–SMV resection and reconstruction was not performed.

All patients were diagnosed pathologically with invasive pancreatic carcinoma. Curative resection (R0) was defined by a specimen with clear resection margins and no gross tumour mass remaining at the operation site or in other organs. As the orientation of the retroperitoneal margin can be difficult to discern after surgery, this margin was inked in collaboration with the pathologist. Postoperative complications were graded according to the Dindo–Clavien classification[26].

Statistical analysis

Continuous data are presented as median (range). χ^2 analysis was used to compare categorical data between groups, and the Mann–Whitney U test for analysis of continuous variables. Overall survival probabilities following PD were estimated by the Kaplan–Meier method and compared by means of the log rank test. All statistical analyses were performed using SPSS® version 21.0 (IBM, Armonk, New York, USA) and $P \leq 0{\cdot}050$ was considered statistically significant.

Results

A total of 216 patients underwent PD for cancer of the pancreatic head or uncinate process in the study interval. Of these, 127 patients had PD with venous resection. Twenty-four patients were excluded from the study, owing to operative death (2) and inadequate follow-up (22). Of the remaining 103 patients, 43 underwent PV–SMV confluence resection with ligation of the SV, and 27

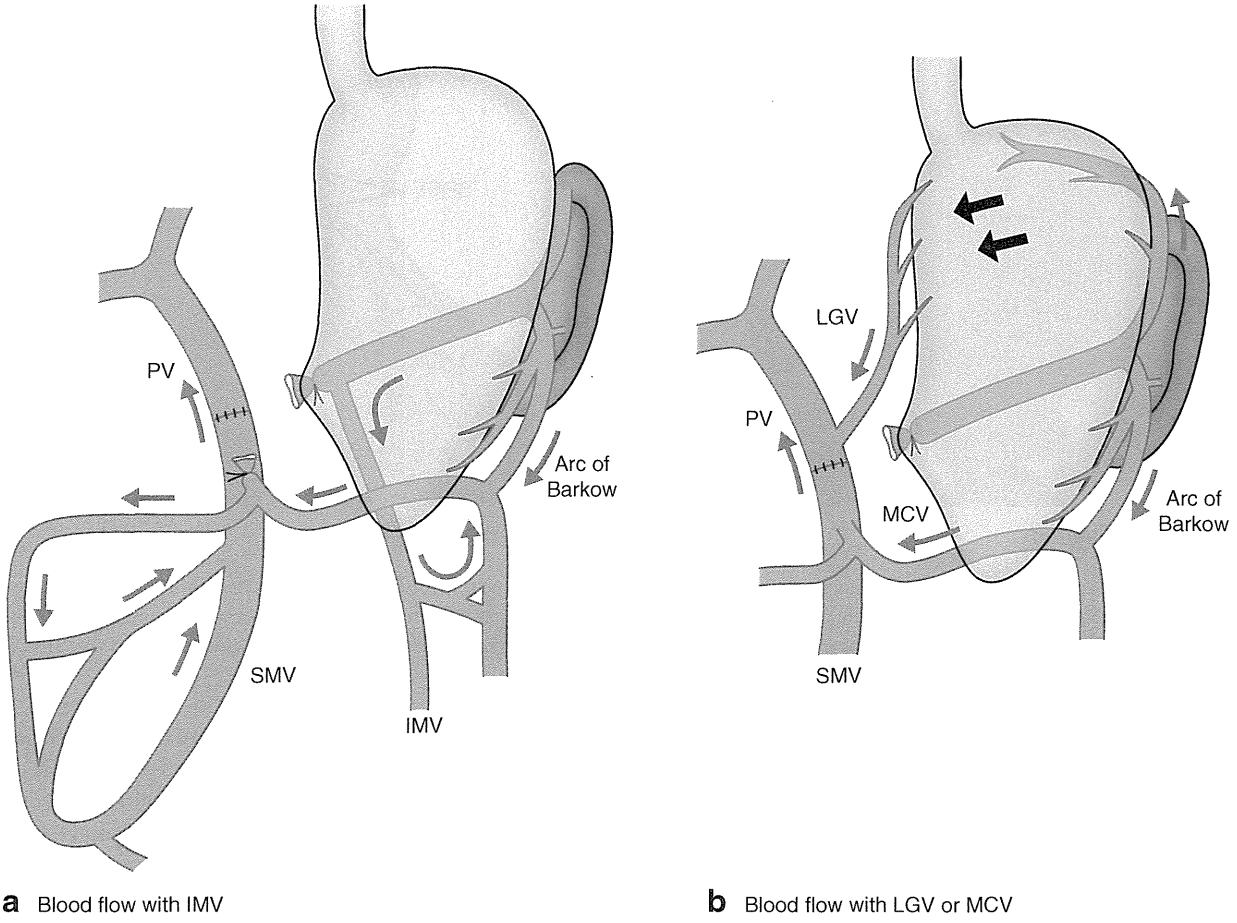

Fig. 3 Venous flow pattern from the spleen after portal vein (PV)–superior mesenteric vein (SMV) confluence resection. **a** When the inferior mesenteric vein (IMV) was not divided, the flow from the spleen passed through the IMV or arc of Barkow to the colonic marginal vein, and was delivered into the portal vein (PV). Red arrows indicate the direction of blood flow. **b** If the middle colic vein (MCV) was preserved, the flow from the spleen passed through the arc of Barkow and drained into the SMV via the MCV. If the left gastric vein (LGV) was not divided, blood flow from the spleen passed through the short gastric veins and drained into the PV via the LGV. The large black arrows indicate the direction of blood flow in the stomach wall

of these patients had division of the IMV with SV ligation (*Fig. 1*).

Postoperative hospital stay for patients who underwent PD without PV resection was no different from that for patients who had PD with PV resection (both median 27 days; $P = 0{\cdot}568$). Median survival was significantly longer after PD without PV resection than with PV resection (29 *versus* 18 months; $P = 0{\cdot}003$).

Perioperative clinical characteristics of the 43 patients in the SV ligation group were compared those of 60 patients who underwent PV resection without SV ligation. There were no significant differences between the SV ligation and no-ligation groups in demographic and clinical factors, including age (median 66 (range 37–86) *versus* 67 (37–83) years; $P = 0{\cdot}373$), sex distribution (58 *versus* 43 per cent women; $P = 0{\cdot}164$), median duration of surgery (571 (410–1118) *versus* 529 (379–1108) min; $P = 0{\cdot}095$), hepatic steatosis rate after surgery (47 *versus* 37 per cent; $P = 0{\cdot}416$), R0 margin rate (53 *versus* 62 per cent; $P = 0{\cdot}425$) and length of hospital stay (28 (21–75) *versus* 27 (range 18–111) days; $P = 0{\cdot}215$). Perioperative morbidity rates (Dindo–Clavien grade III or more) were almost the same for patients with SV and without SV ligation (12 *versus* 10 per cent; $P = 1{\cdot}000$). Median overall operative blood loss for patients undergoing SV ligation was 870 (range 50–6700) ml, significantly higher than the median of 620 (120–2360) ml among patients

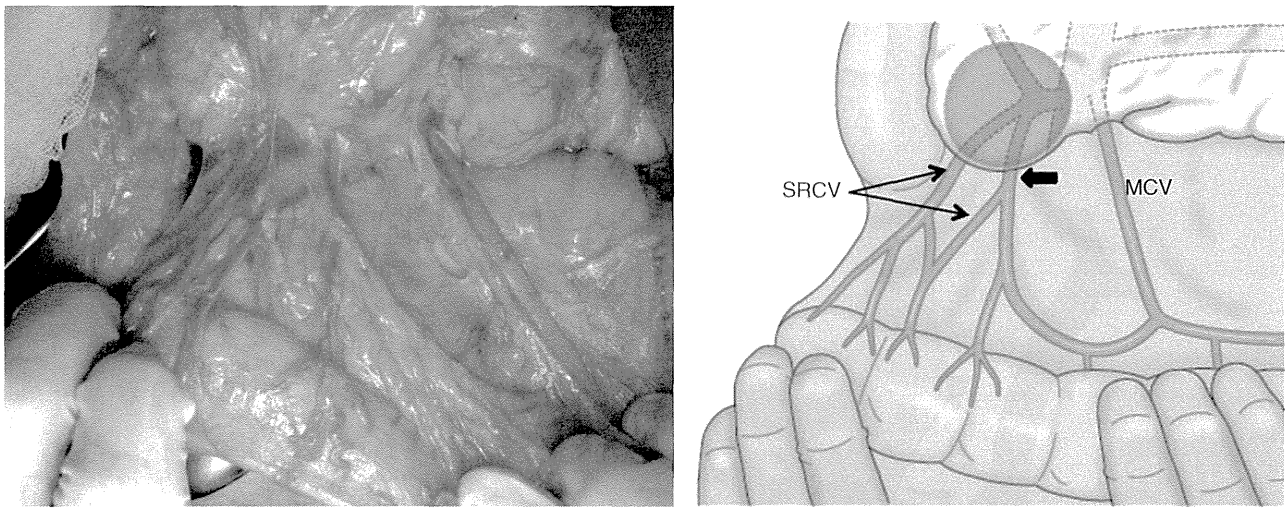

Fig. 4 Confluence of the transverse colic marginal vein and superior right colic vein (SRCV). **a** Intraoperative photograph showing the anatomical relationship between the middle colic vein (MCV) and SRCV. **b** Schematic diagram of the photographic image. The red circle indicates pancreatic tumour. The large black arrow shows the confluence of the transverse colic marginal vein and the SRCV

not undergoing SV ligation ($P = 0{\cdot}043$). Median survival times were no different (21 and 18 months respectively; $P = 906$).

Venous flow patterns from the spleen

The collateral venous flow was evaluated by CT at 4–8 months in the 43 patients who had PD with PV–SMV confluence resection (SV resection). After PD with SV ligation, veins emanating from the spleen were enlarged in almost all patients.

In 14 patients, the veins draining from the spleen could be traced until they merged into the SMV via the colic marginal vein. These 14 patients were classified as having a splenocolonic collateral (*Fig. 2a*; *Fig. S1*, supporting information). In these patients, the flow from the SV passed through the IMV or arc of Barkow[27] to the colonic arcade and then into the PV (*Fig. 3a*). Two other patients had a spontaneous venous shunt from the spleen to the left renal vein; these patients were classified as having a splenorenal shunt. The flow pattern in patients who had either a splenocolonic collateral or a splenorenal shunt was termed the non-varicose route.

In the remaining 27 patients, veins from the spleen ended in the colonic wall at the hepatic flexure and made varicose veins in the colonic wall; this flow pattern was termed the varicose route. Four types of varicose venous pattern were identified (*Fig. 2b*). All 27 patients had colonic varices, which were located in the right flexure of the colon. These colonic varices were most likely caused by ligation of the SV and the confluence of the superior right colic vein and transverse colic marginal vein (*Fig. 4*). The blood flow from the spleen drained into the arc of Barkow and marginal vein along the transverse colon, then finally towards the SMV via the right colic marginal vein. The flow of the marginal vein along the transverse colon to the right colon was cut off by division of the confluence of the superior right colic vein (*Fig. 4*). As a result, colonic varices formed to produce a left-to-right colonic venous pathway (*Figs 2b* and *5a–c*). Three other types of varicose vein and two types of collateral pathway also developed in these patients, as part of the venous flow from the spleen. The varicose veins were pancreatojejunostomy varices (15 patients) (*Fig. 5d,e*), oesophageal varices (14 patients) *Fig. 6a,b*) and gastrojejunostomy varices (8 patients) (*Fig. 6c–e*). The collateral pathways included an aberrant left gastric vein (4 patients) and a gastrorenal shunt (1). Gastric varices did not develop in any of these 27 patients in the present study.

Two patients with the varicose route experienced gastrointestinal haemorrhage from the varicose veins and another developed oesophageal varices, all of which required treatment. The first two patients had repeated gastrointestinal bleeds starting approximately 1 year after operation. As the exact site of bleeding could not be determined from CT images, upper gastrointestinal endoscopy or colonoscopy, the patients underwent angiography.

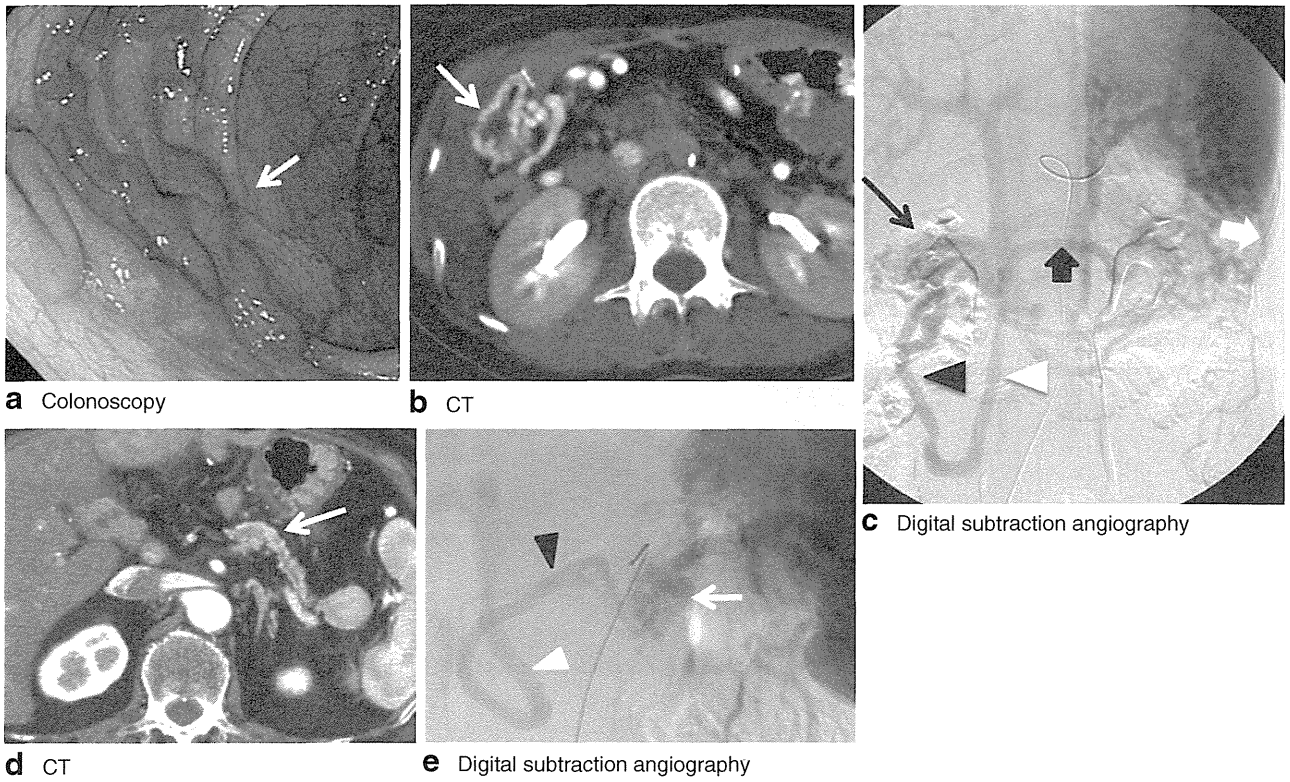

Fig. 5 Colonic and pancreatojejunostomy varices. **a** Colonoscopy revealing tortuous transverse colonic varices (white arrow) near the hepatic flexure. **b** CT image showing colonic varices (white arrow) on right flexure of the colon. **c** Selective splenic artery digital subtraction angiography (venous phase) showing blood flow from the spleen draining into the arc of Barkow (large white arrow) and marginal vein along the transverse colon (large black arrow), colonic varices (black arrow), then finally towards the superior mesenteric vein (white arrowhead) via the right colic marginal vein (black arrowhead). **d** CT image showing pancreatojejunostomy varices (white arrow). **e** Coeliac artery digital subtraction angiography (venous phase) showing blood flow from the splenic vein draining into the anastomosis of the pancreas and jejunum (white arrow), then finally toward the superior mesenteric vein (white arrowhead) via the jejunal vein (black arrowhead)

Angiography and further inspection of CT images revealed the venous flow patterns: colonic and pancreatojejunostomy varices in one patient, and all four types of varicose pattern in the second (*Figs 5* and *6*). These two patients finally underwent splenectomy 1·5 and 2 years after the first operation. Their splenic volume approximately doubled after PD and all varices disappeared after splenectomy. The patient with oesophageal varices, which were diagnosed 3 years after PD, was treated by endoscopic variceal ligation.

Splenic hypertrophy ratio

The splenic hypertrophy ratio was significantly higher in patients with SV ligation than in those without: median 1·37 (0·65–2·59) and 0·96 (0·59–1·83) respectively ($P < 0·001$).

As the splenic volume was increased significantly after PD with SV ligation, the splenic hypertrophy ratio of patients with and without IMV ligation, and those with non-varicose or varicose routes in the SV ligation group, was examined. There was no significant difference in splenic hypertrophy ratio between patients with and without IMV ligation: median 1·34 (0·65–2·59) *versus* 1·49 (0·74–1·97) respectively ($P = 0·451$). By contrast, there was a significant increase in the splenic hypertrophy ratio among patients who had varicose routes compared with those who had non-varicose routes: median 1·52 (0·88–2·59) *versus* 0·94 (0·65–1·67) ($P < 0·001$). The splenic hypertrophy ratio at 12 months after surgery was almost the same as at 4–8 months (data not shown).

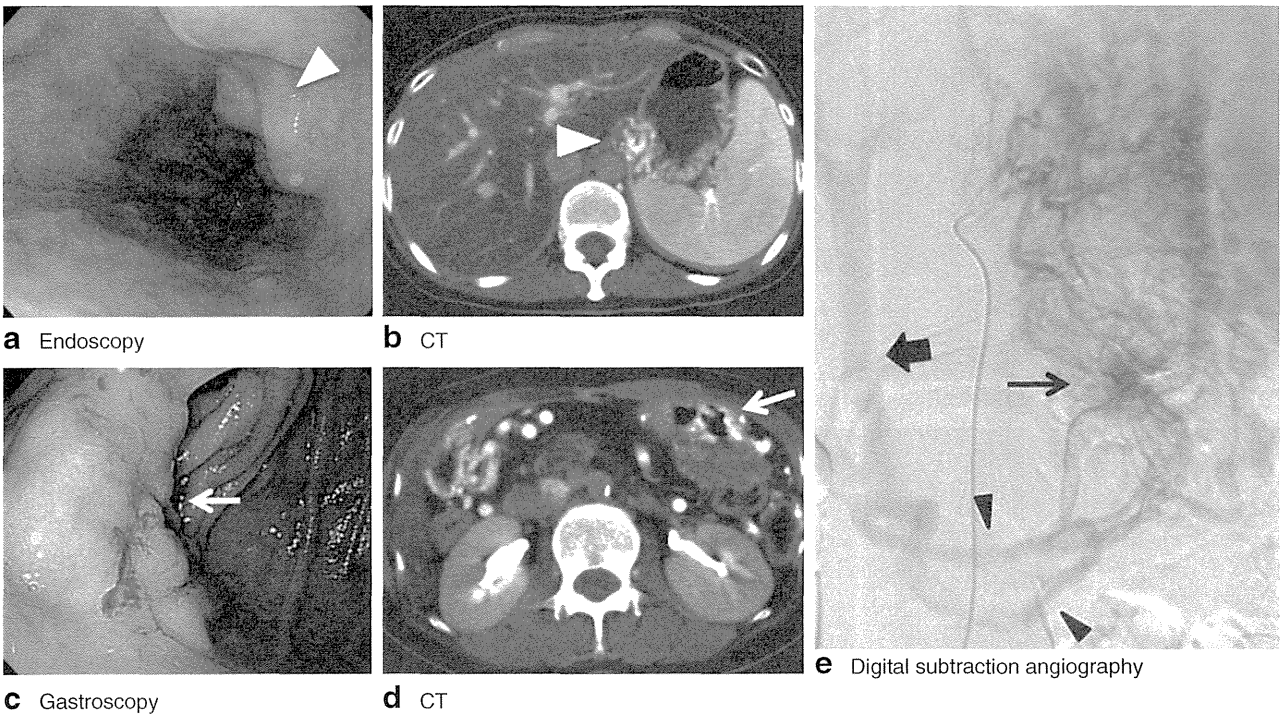

Fig. 6 Oesophageal and gastrojejunostomy varices. **a** Endoscopy showing oesophageal varices (white arrowhead). **b** CT image showing large paraoesophageal varices (white arrowhead). **c** Gastroscopy showing gastrojejunostomy varices (white arrow). **d** CT image showing gastrojejunostomy varices (white arrow). **e** Selective left gastric artery digital subtraction angiography (venous phase) showing blood flow from the gastric veins draining into the anastomosis of the stomach and jejunum (black arrow), then finally toward the superior mesenteric vein (large black arrow) via the jejunal marginal vein (black arrowheads)

Discussion

Sinistral portal hypertension is a clinical syndrome that usually occurs as a result of isolated SV thrombosis, which in turn arises from various aetiologies, mainly pancreatic diseases. Sinistral portal hypertension should be considered in patients with gastrointestinal bleeding who have normal liver function tests and unexplained splenomegaly. Recurrent haemorrhage is not usual[28] and the prognosis depends mainly on the underlying aetiology.

Several clinical issues and reconstruction techniques or venous flow patterns from the spleen have been described after PV–SMV confluence resection during PD. Previously, some surgeons[19–22] reported that reconstruction of the SV was not necessarily required and there were no complications after PD with SV ligation. Strasberg and colleagues[22] examined the pattern of venous collateral development after ligation of the SV in five patients, and concluded that occlusion of the SV did not result in splenomegaly or other symptoms when evaluated after 6–8 months. They described an inferior route, similar to the splenocolonic collateral in the present study, but did not mention a varicose route. In fact, they did not encounter sinistral portal hypertension after SV ligation because they preserved the left gastric vein, middle colic vein or transverse colic marginal vein. Weitz and co-workers[18] reported that the SV may simply be ligated without any negative consequences, such as venous congestion of the stomach, especially when the left gastric vein is preserved. If the middle colic vein is preserved, the flow from the spleen passes through the arc of Barkow and drains into the SMV via the middle colic vein. If the left gastric vein is not divided, the blood flow from the spleen passes through the short gastric vein and drains into the PV via the left gastric vein (*Fig. 3b*). However, when an extended PD with PV–SMV confluence resection is performed for infiltrative pancreatic head cancer, the major vessels are ligated, that is the left gastric vein, right gastric vein, gastrocolic trunk of Henle, middle colic vein and sometimes the IMV if it returns to the SMV or splenomesenteric angle. In this situation, blood flow from the spleen passes through the splenocolonic collateral or the varicose route (*Fig. 2*).

In contrast, many authors describe the importance of preserving the SV to prevent upper gastrointestinal

haemorrhage. Some surgeons have introduced and recommended reconstruction techniques to preserve the SV[8,9,14,18]. It has also been suggested[12,16] that SV–IMV anastomosis or preservation of a natural SV–IMV confluence provides sufficient venous drainage of the spleen and gastric remnant. Routine construction of a direct shunt between the SV and the left renal vein when the natural SV–IMV confluence cannot be preserved has also been reported[17,29]. However, these studies did not examine the cause of sinistral portal hypertension.

Misuta and co-workers[15] evaluated splenic venous flow during surgery by colour Doppler ultrasonography, and constructed an SV–IMV or SV–SMV anastomosis. They advocated a strategy involving an end-to-side anastomosis between the SV and SMV, using a bypass graft when the flow from the IMV drained into the SV after division of the SV. According to examination of the collateral pattern after ligation of the SV in the present study (*Fig. 2*), an SV–IMV anastomosis or preservation of the natural SV–IMV confluence would not stop the development of sinistral portal hypertension because the blood flow from the spleen was the same when the IMV was divided (*Fig. 3b*).

In the present study, 27 patients developed a varicose route and were considered to have sinistral portal hypertension. However, only a minority of them required treatment for gastrointestinal bleeding or oesophageal varices. Treatment for bleeding or oesophageal varices occurred years after surgery. Considering that the median survival time after PD with SV ligation was 21 months in the present study, many patients may die before gastrointestinal bleeding occurs as a result of sinistral portal hypertension. This may explain why sinistral portal hypertension associated with PD and SV ligation has been studied infrequently.

However, understanding the pathogenesis of sinistral portal hypertension is important if extensive surgery is performed. In the present study, all patients with the varicose route developed colonic varices. The colonic varices were most likely caused by ligation of the confluence of the superior right colic vein and transverse colic marginal vein (*Fig. 4*), because the blood flow of the spleen must pass through the confluence to enter the SMV via the right colic marginal vein (*Fig. 2a*). The splenic hypertrophy ratio in patients with a varicose route was significantly higher than that in patients with a non-varicose route. Furthermore, there was no significant difference in the splenic hypertrophy ratio between patients with and without IMV ligation, which suggests that IMV ligation had no association with sinistral portal hypertension.

The superior right colic vein has been defined as the tributary from the marginal veins of the right flexure of the colon to the confluence of the gastrocolic trunk of Henle or the SMV[30]. Unlike the middle and right colic veins, the entire path of the superior right colic vein has no corresponding arteries. During resection of the pancreas, this vein may often be cut and divided to avoid being torn, resulting in unexpected bleeding. However, anticipating the confluence of the superior right colic vein and transverse colic marginal vein is important for keeping the marginal venous flow from the left side of the portal system (*Fig. 4*). This marginal vein is not immediately adjacent to the colon at the hepatic flexure.

The important anatomy of venous flow patterns from the spleen via the colic marginal vein after PD with SV ligation has been described here. In the event of SV ligation, the colic marginal vein should be preserved (with or without IMV preservation) to avoid sinistral portal hypertension. When the marginal vein cannot be preserved because of tumour invasion, there is a potential risk of developing varices associated with critical gastrointestinal haemorrhage owing to sinistral portal hypertension. Reconstruction of the SV may prevent sinistral portal hypertension (for example a distal splenorenal shunt, direct anastomosis of SV and PV). There would be no difference if a pylorus-preserving procedure were performed. When following up patients after PD with SV ligation, it is crucial to confirm the venous flow pattern from the spleen by CT. Detailed knowledge of the vascular anatomy of sinistral portal hypertension is necessary during complex surgical procedures for pancreatic cancer involving the PV–SMV confluence.

Disclosure

The authors declare no conflict of interest.

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