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ORIGINAL SCIENTIFIC REPORT

# Benign Portal Vein Stenosis After Pancreaticoduodenectomy

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## Abstract

*Background* The long-term patency of the portal vein (PV) in patients who survive after pancreaticoduodenectomy (PD) remains unclear. The aim of the present study was to investigate the clinical features and risk factors for benign PV stenosis after PD.

*Methods* We retrospectively analyzed the patients who underwent PD from September 2002 and December 2015 at our institution without intraoperative radiation therapy or concomitant PV resection. The postoperative computed tomography of each patient was evaluated, and PV stenosis was defined as the shortest diameter of the PV being <3 mm. The patients with PV stenosis due to local recurrence were excluded.

*Results* Of the 458 patients, PV stenosis occurred in 57 (12.4%), including benign PV stenosis in 28 (6.1%) and PV stenosis due to local recurrence in 29 (6.3%). Of the 28 patients with benign PV stenosis, 7 (25%) developed symptoms related to portal hypertension, namely recurrent gastrointestinal bleeding in 5 and refractory ascites in 2. Six patients were treated with percutaneous transhepatic PV stent placement, and all of their symptoms improved. A multivariate analysis found that a postoperative pancreatic fistula was an independent risk factor for benign PV stenosis after PD (odds ratio, 4.36; $p = 0.005$).

*Conclusions* Postoperative pancreatic fistula was a significant risk factor for benign PV stenosis after PD. Stent placement for benign PV stenosis was effective for the patients with symptoms due to portal hypertension.

## Introduction

Portal vein (PV) stenosis is a late complication after pancreaticoduodenectomy (PD) that is likely to cause portal hypertension [1–3]. Portal hypertension secondary to PV stenosis can lead to gastrointestinal bleeding due to varices or refractory ascites, which may threaten the health and life of patients [4, 5]. Several reports have shown that local recurrence of the periampullary tumor often causes PV stenosis after PD [6, 7]. Intraoperative radiation therapy (IORT) and concomitant PV resection with PD can directly affect the portal system and have also been reported to be associated with PV stenosis after surgery [8–12]. In contrast, the etiology of benign PV stenosis without these evident factors remains unclear, often developing a long time after surgery [2, 3].

The long-term outcomes after surgical resection for periampullary tumors had been poor for the last several decades [13]. Recent advances in perioperative management approaches and adjuvant therapies have markedly improved the survival of patients after PD for periampullary malignancies [14, 15]. Little is known, however,

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about late complications in patients who survive after PD, such as stenosis of the PV. It has become important to investigate the long-term patency of the PV in patients who survive for a long period after PD and to evaluate the etiology of benign PV stenosis.

The purpose of the present retrospective study was to clarify the clinical features and risk factors of benign PV stenosis after PD. The details of related symptom due to portal hypertension and the efficacy of treatment with PV stent placement were also investigated.

## Materials and methods

### Patients

A retrospective review was conducted using a prospectively collected database of patients who underwent PD at the Shizuoka Cancer Center between September 2002 and December 2015. The exclusion criteria included (1) IORT, (2) concomitant PV resection and reconstruction, (3) death or loss of follow-up within 1 year after surgery, and (4) lack of postoperative imaging follow-up. The present study was approved by the institutional review board of the Shizuoka Cancer Center.

### Operation and surgical outcomes

Our standard surgical procedure was conventional PD with the modified Child's method [16]. Postoperative morbidity was defined as complications occurring within 30 days after surgery or during the hospital stay. Pancreatic fistulas (PFs) were defined as grade B or C according to the definition of the International Study Group of Pancreatic Surgery [17]. Bile leakage was defined as grade B or C according to the definition by the International Study Group of Liver Surgery [18]. Intra-abdominal abscess was defined as the presence of fluid collection confirmed by computed tomography (CT) or syringography and requiring radiological intervention and/or antimicrobial therapy. Patients with PF were mostly treated with antibiotics, persistent drainage, and percutaneous drainage. Somatostatin analog was not used.

### The evaluation of PV stenosis

All patients underwent periodic postoperative CT after PD in which the assessments were biphasic (unenhanced and equilibrium phases) and reconstructed at a thickness of 5 mm. Follow-up CT examinations were generally performed at 3- to 12-month intervals. An experienced radiologist (TA) blinded to the clinical course of the patients analyzed the CT images. The diameter of the PV was measured on CT images at the equilibrium phases at least 2 months after surgery, as the natural stenotic changes in the PV shortly after PD had almost disappeared by the eighth postoperative week [19]. PV stenosis was defined as the shortest diameter of the PV being <3 mm (Fig. 1a) [20]. PV occlusion was defined as the cessation of enhanced PV lumen. Collateral veins and jejunal varices around the hepaticojejunostomy were also evaluated (Fig. 1a, b). PV stenosis due to local recurrence of the tumor was defined as malignant PV stenosis. Local recurrence was confirmed by radiographic evidence for recurrent disease in the operative and regional area with positive findings on positron emission tomography and/or increased serum levels of tumor markers. PV stenosis not due to local recurrence was defined as benign PV stenosis. The first onset of these findings was defined as the time of PV stenosis.

### Management of PV stenosis

The patients who had PV stenosis without the presence of clinical manifestations underwent follow-up observation. The indication of treatment for PV stenosis was the refractory symptoms due to portal hypertension, such as gastrointestinal bleeding or ascites. A percutaneous transhepatic procedure was performed for PV stent placement under local anesthesia [6, 7, 21]. A self-expandable metallic stent (E-Luminexx [Bard, Tempe, AZ, USA] or Zilver [Cook, Bloomington, IN, USA]) was placed into the PV across the constricted part by an experienced interventional radiologist (TA) (Fig. 1c, d). Anticoagulation (heparin and subsequent oral warfarin) after the procedure was routinely administered. Stent patency was confirmed by follow-up ultrasound or CT examinations at 3- to 6-month intervals.

### Statistical analyses

All continuous variables were expressed as the medians with ranges and were compared using the Mann–Whitney *U* test. Comparisons between the categorical variables were analyzed by Fisher's exact test. The univariate factors showing a $p$ value of <0.05 were entered into a multivariate logistic regression analysis to identify the independent risk factors for benign PV stenosis. All statistical analyses were performed using the SPSS 25.0 software program (SPSS Inc., Armonk, NY, USA). A $p$ value of <0.05 was considered to be statistically significant.

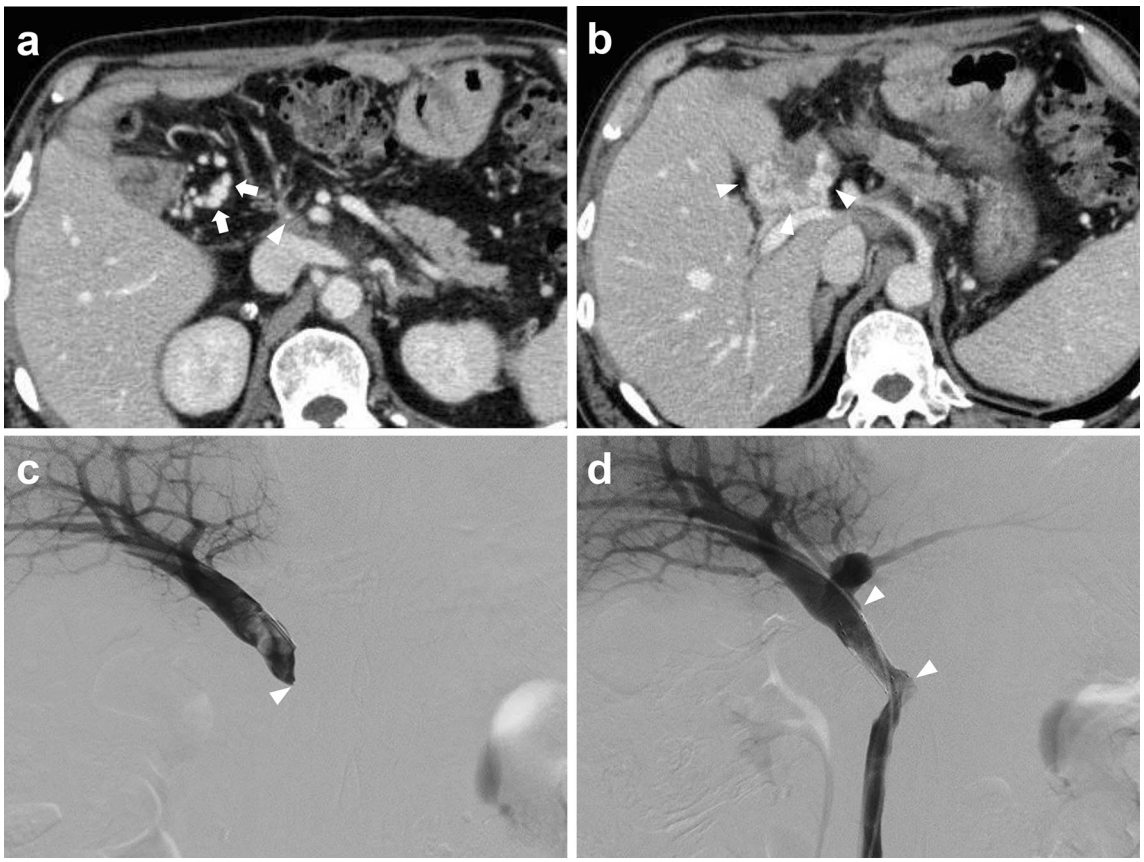


**Fig. 1** **a** Contrast-enhanced computed tomography shows severe stenosis of the superior mesenteric vein (arrowhead) and the formation of collateral vessels (arrows). **b** Computed tomography shows jejunal varices around the hepaticojejunostomy (arrowheads). **c** Percutaneous transhepatic portography shows the occlusion of the main portal vein (arrowhead). **d** Portography shows a metallic stent in the main portal vein (between the arrowheads). The portal vein is patent after stent placement

## Results

Between September 2002 and December 2015, 779 consecutive patients underwent PD at the authors' institution. Of these patients, 19 who underwent IORT, 186 who underwent concomitant PV resection, 75 who died or were lost to follow-up within 1 year, and 41 without available postoperative CT images were excluded. We selected the remaining 458 patients for the analyses (310 [68%] males and 148 [32%] females, with a median age of 68 [36–88] years). Of these 458 patients, 131 (29%) had pancreatic cancer, 119 (26%) had bile duct cancer, 59 (13%) had ampullary cancer, 51 (11%) had intraductal papillary mucinous neoplasm, 37 (8.1%) had duodenal cancer, and the remaining 61 (13%) had other diseases. The median follow-up period of the censored 302 patients was 61.3 (12.2–163.3) months. PV stenosis occurred in 57 patients (12.4%), including benign PV stenosis in 28 (6.1%) and malignant PV stenosis in 29 (6.3%).

Table 1 shows the demographics of the 28 patients with benign PV stenosis after PD. The median interval from surgery to the development of PV stenosis was 5.4 (2.1–27.1) months. Of these patients, 19 (68%) had collateral veins, and 12 (43%) had jejunal varices around the hepaticojejunostomy. Only one patient developed colonic varices; however, it did not cause hemorrhage. Twenty-one patients (75%) had been asymptomatic, whereas 7 (25%) developed symptoms related to portal hypertension, including recurrent gastrointestinal bleeding in 5 and refractory ascites in 2. No patient suffered from hemorrhagic shock due to melena. The median interval from surgery to the development of symptoms was 32.5 (3.4–88.9) months.

Table 2 shows the results of univariate and multivariate analyses to identify the risk factors for benign PV stenosis after PD. The univariate analyses showed that the population with a high body mass index ($\geq$25 kg/m$^2$) was significantly greater in the patients with benign PV stenosis than in those without PV stenosis. The incidences of

**Table 1** Demographics of the patients with benign PV stenosis after PD

| | $n = 28$ |
|---|---|
| Age* (years) | 67 (44–81) |
| Sex (male) | 23 (82) |
| Histology | |
| Pancreatic cancer | 8 (29) |
| Bile duct cancer | 8 (29) |
| Ampullary cancer | 5 (18) |
| Duodenal cancer | 3 (11) |
| IPMN | 2 (7.1) |
| Others | 2 (7.1) |
| Period from surgery to development of PV stenosis* (months) | 5.4 (2.1–27.1) |
| Location of stenosis | |
| PV | 9 (32) |
| SMV | 15 (54) |
| PV–SMV | 4 (14) |
| Collateral veins | 19 (68) |
| Jejunal varices around the hepaticojejunostomy | 12 (43) |
| PV occlusion | 4 (14) |
| AST* (U/L) | 27 (16–87) |
| ALT* (U/L) | 24 (10–115) |
| Symptoms | 7 (25) |
| Recurrent gastrointestinal hemorrhaging | 5 |
| Refractory ascites | 2 |
| Period from surgery to development of symptom* (months) | 32.5 (3.4–88.9) |
| Stent placement | 6 (21) |

Values in parentheses are percentages unless indicated otherwise

*PV* portal vein, *PD* pancreaticoduodenectomy, *IPMN* intraductal papillary mucinous neoplasm, *SMV* superior mesenteric vein, *AST* aspartate aminotransferase, *ALT* alanine aminotransferase

*Median (range)

postoperative PF, bile leakage, and intra-abdominal abscess were significantly higher in the patients with benign PV stenosis than in those without PV stenosis. The multivariate analysis including these four variables showed that a postoperative PF (odds ratio, 4.36; $p = 0.005$) was an independent risk factor for benign PV stenosis after PD.

Of the 83 patients who developed intra-abdominal abscess after surgery, 70 underwent CT for the diagnosis (10 with PV stenosis and 60 without PV stenosis). The location of the fluid collection was evaluated in these patients (11 had more than 2 lesions), and 31 had abscess around the pancreaticojejunal anastomosis (44%, Fig. 2a), 25 had abscess in front of the inferior vena cava (IVC) (36%, Fig. 2b), 13 had abscess around the liver surface (19%, Fig. 2c), and 7 had abscess around the left caudate lobe (10%, Fig. 2d). The incidence of abscess in front of the IVC was significantly higher in the patients with PV stenosis than in those without PV stenosis (90% vs. 27%, $p < 0.001$). In contrast, the other locations of abscess were not significantly associated with the incidence of PV stenosis.

Table 3 shows the details of six patients who underwent percutaneous transhepatic PV stent placement for benign PV stenosis (one patient with refractory ascites was observed without intervention, considering his age and general condition). All patients were male, three had bile duct cancer, and the remaining three had ampullary cancer. Postoperative pancreatic fistula developed in five patients. The median interval from surgery to stent placement was 43.4 (8.2–95.1) months. Over the long term, all of their symptoms improved after stent placement, although one patient (Case 2) experienced recurrent bleeding from jejunal varices at 3 years after stent placement. In that case, oral warfarin was stopped and there has been no further recurrence. The median interval of stent patency was 69.9 (14.5–103.9) months. The median duration of anticoagulation after stent placement was 55.6 (30.6–112.3) months.

## Discussion

The present study investigated the clinical features and risk factors for benign PV stenosis after PD using a large patient cohort who underwent long-term surveillance after surgery at a single high-volume center in Japan. The results showed that the rate of benign PV stenosis after PD was 6.1%, and postoperative PF was significantly associated with benign PV stenosis after PD. The location of intra-abdominal abscess (in front of the IVC) was associated with the incidence of benign PV stenosis. Most patients with benign PV stenosis were asymptomatic, whereas the six patients with symptoms due to portal hypertension were successfully treated with percutaneous transhepatic PV stent placement.

The long-term patency of the PV after PD has not been fully investigated, and the etiology for benign stenosis of the PV remains unclear. Because the present study focused on the benign stenotic change of the PV, the patients who underwent IORT or PV resection and reconstruction were excluded, as these factors directly affect the PV patency [8–12]. The multivariate analysis showed that postoperative PF was an independent risk factor for benign PV stenosis. This etiology may be biologically feasible, as local inflammation due to PF may induce the development of hard fibrotic adhesions around the PV. Previous reports have suggested that fibrosis and granulation tissue that developed after abdominal surgery were associated with PV stenosis [2, 21]. The patients with benign PV stenosis had a higher BMI and higher probability of postoperative

**Table 2** Univariate and multivariate analyses of the risk factors for benign PV stenosis after PD

| Variables | Univariate | | | Multivariate | | |
|---|---|---|---|---|---|---|
| | Benign PV stenosis | | *p* | OR | 95% CI | *p* |
| | (+) *n* = 28 | (–) *n* = 401 | | | | |
| Characteristics of patients | | | | | | |
| Age ≥ 70 years | 11 (39) | 173 (43) | 0.844 | | | |
| Sex (male) | 23 (82) | 265 (66) | 0.097 | | | |
| BMI ≥ 25 kg/m$^2$ | 8 (29) | 50 (12) | 0.039 | 1.73 | 0.68–4.40 | 0.247 |
| ASA-PS ≥ 3 | 3 (11) | 35 (8.7) | 0.728 | | | |
| Diabetes mellitus | 8 (29) | 93 (23) | 0.496 | | | |
| Preoperative biliary drainage | 15 (54) | 201 (50) | 0.846 | | | |
| Histology (pancreatic cancer) | 8 (29) | 106 (26) | 0.826 | | | |
| Neoadjuvant therapy | 1 (3.6) | 2 (0.4) | 0.184 | | | |
| Operative details | | | | | | |
| Operative time ≥ 480 min | 5 (18) | 99 (25) | 0.500 | | | |
| Blood loss ≥ 1000 ml | 10 (36) | 137 (34) | 0.840 | | | |
| Blood transfusion | 2 (7.1) | 46 (11) | 0.756 | | | |
| Soft pancreatic texture | 24 (86) | 279 (70) | 0.086 | | | |
| Surgical outcomes | | | | | | |
| Intra-abdominal abscess | 12 (43) | 71 (18) | 0.005 | 1.74 | 0.72–4.17 | 0.217 |
| Intra-abdominal hemorrhaging | 1 (3.6) | 11 (2.7) | 0.560 | | | |
| Bile leakage | 3 (11) | 9 (2.2) | 0.037 | 3.80 | 0.86–16.80 | 0.078 |
| Pancreatic fistula | 23 (82) | 181 (45) | < 0.001 | 4.36 | 1.55–12.30 | 0.005 |
| Reoperation | 1 (3.6) | 8 (2.0) | 0.458 | | | |

Values in parentheses are percentages

*PV* portal vein, *PD* pancreaticoduodenectomy, *OR* odds ratio, *CI* confidence interval, *BMI* body mass index, *ASA-PS* American Society of Anesthesiologists physical status

bile leakage and intra-abdominal abscess than those without PV stenosis in the present study. These factors may also be associated with PF and local inflammation. A high BMI is a well-known risk factor for PF [22–24]. The combination of bile with pancreatic juice exacerbates the inflammation caused by the PF [25], resulting in the subsequent development of intra-abdominal abscesses [16]. The results of the present study therefore strongly suggest that benign PV stenosis may be associated with PF.

In the subgroup analysis of the patients with postoperative intra-abdominal abscess, the location of the abscess was correlated with the development of benign PV stenosis. An abscess in front of the IVC was significantly associated with a high incidence of PV stenosis. Effusion from the surgical site (including pancreaticojejunal anastomosis) is likely to collect on the anterior surface of the IVC due to gravity. An abscess between the IVC and PV (Fig. 2b) can cause inflammation and the development of fibrosis around the PV, which leads to PV stenosis. Locating an anatomical approach window for percutaneous drainage of abscesses in front of the IVC may be difficult due to their deep location [26]. Placement of a drain for this area is considered to be important in patients at high risk for pancreatic fistula, given the risk of subsequent PV stenosis. At our institution, drains have been routinely inserted since 2011 via the left flank and placed posteriorly to the pancreaticojejunal anastomosis, extending to the anterior surface of the IVC. The incidence of benign PV stenosis actually decreased from 9.7% (22 of 227 patients) before 2010 to 3.0% (6 of 202 patients) after 2011 ($p = 0.006$) in the present study. Appropriate drain placement may prevent fluid collection around the PV and the development of benign PV stenosis.

Most patients with benign PV stenosis did not develop any symptoms in the present study, whereas the six patients who underwent PV stent placement suffered from recurrent melena or refractory ascites. The presentation of these symptoms caused by PV stenosis occurred several years after surgery, as described in previous reports [3, 27]. Stenting for the patients with symptoms was effective, and the stent patency was satisfactory during the follow-up period in the present study. Recent reports have described

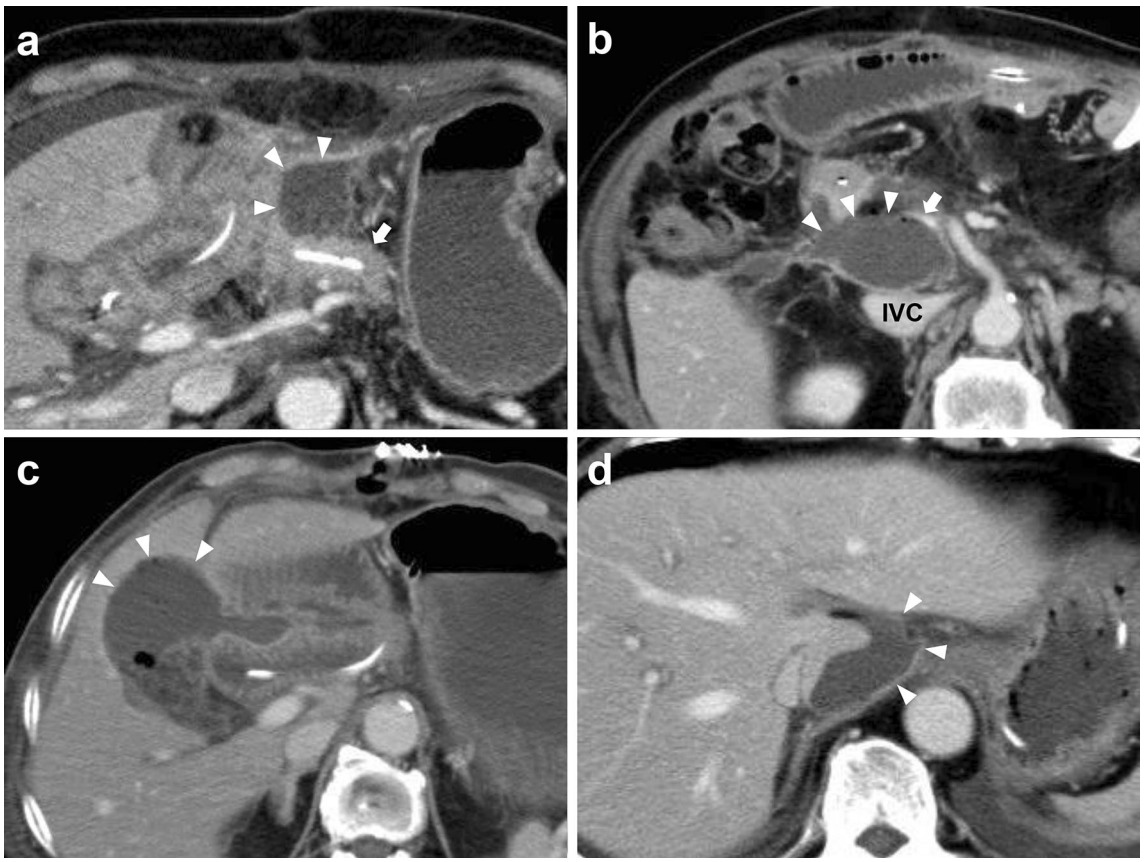


**Fig. 2** **a** Computed tomography shows infected fluid collection around the pancreaticojejunal anastomosis (arrowheads). The arrow indicates the remnant pancreas. **b** Computed tomography shows infected fluid collection in front of the inferior vena cava (arrowheads). The arrow indicates the portal vein touching the abscess. **c** Computed tomography shows infected fluid collection around the liver surface (arrowheads). **d** Computed tomography shows infected fluid collection around the left caudate lobe (arrowheads)

**Table 3** Cases of benign PV stenosis after PD treated with percutaneous transhepatic PV stent placement

| Case | Age (years) | Sex | Disease | Postoperative complication | Symptom | Postoperative period (months) | | Stent patency (months) |
|---|---|---|---|---|---|---|---|---|
| | | | | | | To PV stenosis | To stent placement | |
| 1 | 69 | Male | Ampullary cancer | Wound infection | Gastrointestinal hemorrhaging | 4.8 | 95.1 | 55.8 |
| 2 | 48 | Male | Ampullary cancer | PF, BL | Gastrointestinal hemorrhaging | 27.1 | 37.1 | 103.9 |
| 3 | 77 | Male | Bile duct cancer | PF | Gastrointestinal hemorrhaging | 9.2 | 49.9 | 92.5 |
| 4 | 57 | Male | Bile duct cancer | PF | Gastrointestinal hemorrhaging | 4.2 | 49.6 | 83.9 |
| 5 | 79 | Male | Ampullary cancer | PF | Ascites | 3.0 | 8.2 | 29.6 |
| 6 | 62 | Male | Bile duct cancer | PF | Gastrointestinal hemorrhaging | 6.3 | 19.5 | 14.5 |

*PV* portal vein, *PD* pancreaticoduodenectomy, *PF* pancreatic fistula, *BL* bile leakage

the efficacy of percutaneous transhepatic stent placement for PV stenosis after hepatobiliary surgery [3, 7, 28]. However, this procedure is invasive and potentially dangerous because of the risk of associated complications such as vascular injury, pseudoaneurysm of the hepatic artery, and bile leakage from the intrahepatic duct [4, 29, 30]. It may be acceptable to follow asymptomatic patients with benign PV stenosis for a long period after PD, whereas PV stent placement should be performed only for those who suffer from refractory symptoms due to portal hypertension. Prophylactic anticoagulation was not performed for asymptomatic patients with benign PV stenosis, because there is no evidence to support its efficacy.

Several limitations associated with the present study warrant mention. First, the retrospective design and single-institution setting may have result in selection bias of the patient cohort. However, a multi-institutional prospective study may be difficult to perform because of the low incidence of events and the need for long-term surveillance with CT. A retrospective cohort study with a large population may be appropriate. Second, the definition of PV stenosis has varied in previous reports. Several reports defined PV stenosis in adults after liver transplantation as >50% narrowing of the PV diameter [31, 32]. However, this relative definition may be inappropriate because the diameter of the PV or SMV can be quite different depending on the part that is measured after PD. Thus, PV stenosis was defined by a shortest PV diameter of <3 mm [20] in the present study, which is approximately one-third of the normal PV diameter in adults [33, 34]. Finally, the rate of PF observed in the present study was relatively high (215 of 458 patients, 47%). There were several possible reasons for this: the ratio of patients with pancreatic head cancer (with a low risk of PF) [23, 24] was appreciably low because they were excluded due to IORT and PV resection. In addition, we placed prophylactic drains very close to the site of anastomosis and frequently exchanged the drainage tubes with the continuous measurement of the drainage fluid amylase level. This strict management might have led to a high incidence of PF.

In summary, benign PV stenosis was an uncommon late complication after PD but not extremely rare. Surgeons should carefully follow patients with postoperative pancreatic fistula after PD, considering the risk of benign PV stenosis. Stent placement for benign PV stenosis may be suitable for patients with refractory symptoms due to portal hypertension.

#### Compliance with ethical standards

**Conflict of interest** The authors have no funding or conflicts of interest to disclose.

**Informed consent** Informed consent was obtained from all individual participants included in the present study.

**Human and animal rights** The present study was approved by the institutional review board of the Shizuoka Cancer Center.

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