Case Report

Portal Vein Stenting for Jejunal Variceal Bleeding after Recurrence of Pancreatic Adenocarcinoma: A Case Report and Review of the Literature

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Abstract

A 73-year-old woman with portal vein stenosis caused by tumor recurrence after pancreatoduodenectomy was treated with stent placement without embolization of the jejunal varix. Anticoagulation therapy using heparin followed by rivaroxaban was administered after the procedure. She continued to receive systemic chemotherapy as an outpatient. Neither restenosis nor stent thrombosis was observed after 7 months. Based on the presented case and literature review, portal vein stenting is an effective treatment option for jejunal variceal bleeding caused by malignant portal venous stricture after pancreaticoduodenectomy. Antithrombotic therapy following portal venous stenting is required to prevent stent thrombosis in the majority of cases, although it has a risk of inducing recurrent variceal bleeding. Adjunctive jejunal variceal embolization can possibly be omitted in selected cases to obtain sufficient portal-SMV flow reconstruction.

Key words: Portal vein, Constriction, Stents

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Introduction

Recurrent pancreatic cancer can cause portal venous stenosis, resulting in symptoms of portal hypertension, such as hemorrhagic tendencies and liver dysfunction. Pancreatoduodenectomy renders patients susceptible to jejunal varices that often form at the choledochojejunostomy site because the formation of hepatopetal collaterals is precluded after this surgery. Portal vein stenting has been used to treat jejunal variceal hemorrhage. However, no large cohort study to confirm the efficacy of portal venous stenting has yet been reported, and therefore, there is no consensus on the technical details relevant to portal vein stenting, such as the utility of adjunctive embolization of the jejunal varices and anticoagulant/antiplatelet therapy following stenting. This report describes a case of successful stenting for a patient with portal venous stenosis and bleeding from jejunal varices after pancreatoduodenectomy, along with the relevant literature.

Case Presentation

Institutional review board approval was not required at our institution for this type of report.

A 73-year-old Japanese woman suddenly experienced melena and went into hemorrhagic shock (Hb 6.0 g/dL; systemic blood pressure, <60 mmHg). She had undergone subtotal stomach-preserving pancreatoduodenectomy (SSPPD) for pancreatic head adenocarcinoma without portal vein resection 4 years earlier. The final pathological diagnosis was pTNM stage III (pT3N0M0) disease. She had received che-

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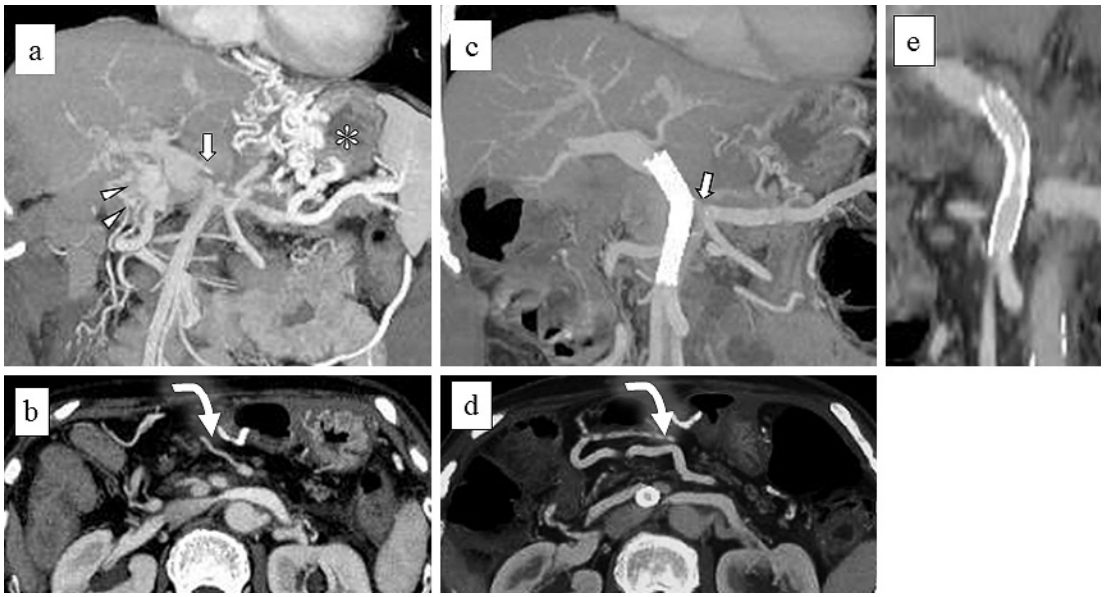

Fig. 1. Contrast-enhanced CT before (a, b) and after (c–e) stent placement. (a) Oblique maximum intensity projection (MIP) image showing portal vein (PV) stenosis (*arrow*). Markedly dilated vasculature clearly demonstrating the short gastric and jejunal veins. The tortuous jejunal vein connects with the paracholedochal vein. As a result, esophageal and gastric varices (EGVs) (*asterisk*) and jejunal varices (JVs) (*arrowhead)* are formed. (b) The marginal vein along the transverse colon and the middle colic vein was not dilated (*curved arrow*) before stenting. (c) Oblique MIP image demonstrating that the JVs and EGVs have disappeared. The SMV was stenosed (*arrow*) after stent placement. (d) The marginal vein is dilated (*curved arrow*). (e) Coronal multiplanar reformation image showing PV stent patency.

motherapy for local recurrence and lung metastasis for 2 years.

Computed tomography (CT) on the patient's admission to our hospital showed portal venous stenosis due to local recurrence, which revealed jejunal varices of the jejunal limb and esophageal and gastric varices (**Fig. 1a**). Gastrointestinal endoscopy revealed coagula in the remnant stomach and colon, and no obvious bleeding points were detected. These findings suggest that the bleeding was due to a rupture of the jejunal varices caused by extrahepatic portal hypertension, and we thus speculated that portal venous stenting for the emergence of lethal portal hypertension would be beneficial. After considering the case by our hospital's institutional review board and the patient's acceptance of portal venous stenting after receiving a thorough explanation of the procedure and its risks, we performed the palliative procedure of a transhepatic insertion of a vascular stent into the portal venous stenosis.

With the patient under local anesthesia, a 6-Fr sheath was inserted at the umbilical portion of the portal vein under sonographic guidance. Portography was performed (**Fig. 2a**). After the tip of a 0.035-inch guidewire (Radifocus® E type, Terumo, Tokyo, Japan) was shaped manually, we passed through the portal venous stenosis using the upper 0.035-inch wire to insert a catheter (#C2, 4.2 Fr, Medikit, Tokyo, Japan) into the superior mesenteric vein (SMV). Venography showed portal venous stenosis, jejunal varices, and esophageal and gastric varices due to portal hypertension (**Fig. 2b, c**). The stenosis detected on venography was measured as approximately 5 cm from the portal vein to the splenoportal confluence stenosis. The pressure of the portal vein was 8 mm Hg, but the corresponding pressure of the SMV was 13 mmHg.

The stenotic lesions were dilated with two balloon catheters (Mustang™, 6×40 mm and 8×40 mm, Boston Scientific, Marlborough, MA, USA); however, the stenoses did not improve. Self-expanding stents (Epic™, 9×40 mm and 10×50 mm, Boston Scientific, Marlborough, MA, USA) were placed across the stenosis. After stenting, the jejunal varices disappeared quickly (**Fig. 2d**). Therefore, the jejunal varices were not embolized. The pressure of the SMV decreased from 13 to 8 mmHg, and the pressure gradient between the portal vein and SMV improved. To prevent bleeding from vascular access, the transhepatic parenchymal route was embolized with a gelatin sponge (Spongel®, Astellas Pharma, Tokyo, Japan) through a 6-Fr sheath. We used heparin for anticoagulant therapy with a bolus of 2,000 IU administered at stent insertion followed by continuous infusion at approximately 10,000 IU per day after stenting; 2 days later, the patient was switched to oral administration of rivaroxaban.

The patient was discharged on the 12th postoperative day without any complications. Follow-up CT at 2 months showed portal vein patency (**Fig. 1e**). The esophageal and gastric varices, as well as jejunal varices, also disappeared despite residual splenic venous stenosis (**Fig. 1c**). The pa-

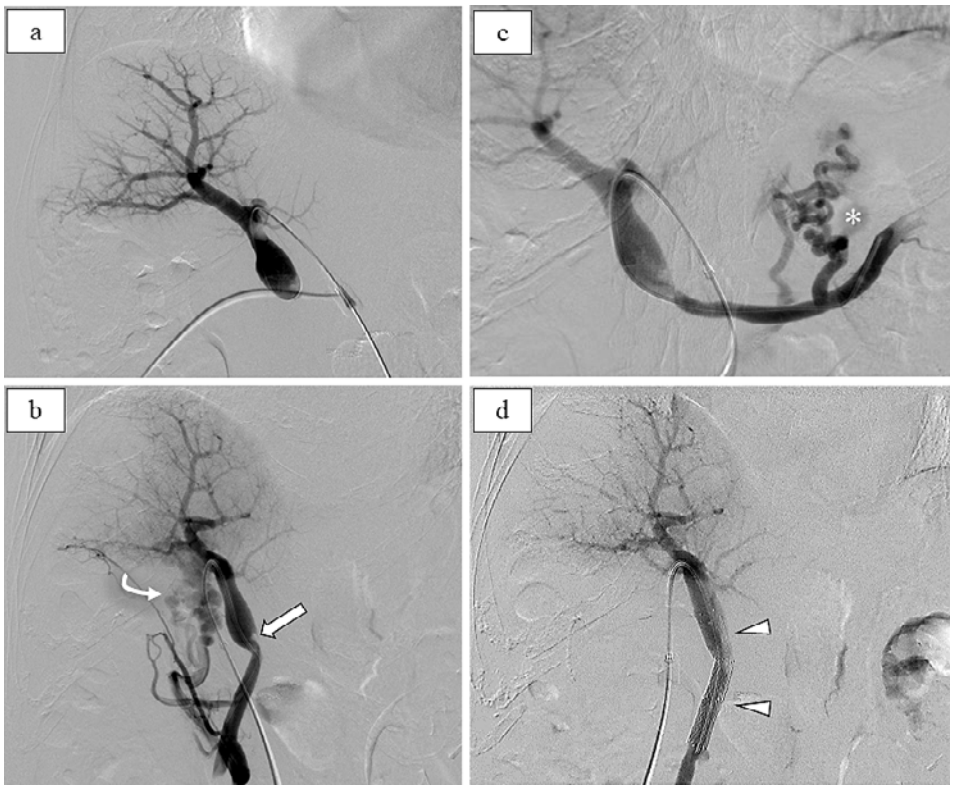

Fig. 2. Venography imaging during stent placement. (a) Portography does not show the SMV or SV because of the splenopoeral confluence stenosis. (b) Superior mesenteric venography shows PV stenosis (*arrow*) and JVs (*curved arrow*). (c) Splenic venography shows a gastrorenal shunt and EGVs (*asterisk*). (d) An expandable-wall stent was placed at the site of PV stenosis (*arrowhead*). At baseline, the pre- and post-stenotic portal venous pressures were 13 mmHg and 8 mmHg, respectively. After the PV stenting, the pre- and post-stenotic portal venous pressures were both 8 mmHg.

tient received chemotherapy in an outpatient setting. Although the tumor was not controlled, tumor ingrowth or overgrowth has not yet been observed. Eventually, the stent remained patent, and no rebleeding was observed for 7 months after the procedure. The administration of rivaroxaban has been continued to date.

Literature Review

We searched for the keywords of "pancreatoduodenectomy" and "portal stent placement" or "portal stenting" in Igaku Chuo Zasshi and PubMed. From 1999 to 2020, there were 26 case reports (**Table 1**) and 14 research articles, including portal venous stenting after pancreatoduodenectomy (**Table 2**) [1-40].

The technical success rate was > 99 % (failure in one case). The incidence of major complications, such as sepsis, liver abscess, and stent thrombosis, was less than 8% (10 cases). In some cases, bleeding related to the percutaneous transhepatic puncture site occurred, but all recovered conservatively. In most reports, symptoms related to portal hypertension improved without major complications.

In 8 of 18 reports (44.4%), the varices or collateral vessels were embolized at the same time as portal venous stenting. Eight case reports were excluded from this calculation owing to the lack of a statement. In two studies, embolization of collateral vessels was performed in selected cases.

In 15 of the 19 evaluable cases (84.2%), anticoagulation or antiplatelet therapies were administered after stenting. Anticoagulation using warfarin or edoxaban was administered in 8 cases, antiplatelet in 6 cases, and anticoagulation followed by antiplatelet therapy in 1 case. Heparin alone or urokinase was administered to the thrombus in three cases. One patient did not receive any therapy. In seven research articles, anticoagulation or antiplatelet therapies were administered.

Discussion

Portal venous stenosis occurs in approximately 12.4% of cases, and symptoms related to portal hypertension that require treatment occur in approximately 1.5%-2.8 % of cases after pancreatoduodenectomy [34, 39]. Jejunal varices formed at the site of the choledochojejunostomy are often difficult to detect and treat by gastrointestinal endoscopy [3]. CT can be used to evaluate the anatomic variation of the portal vein [41].

In our case, the stenosis was dilated using a balloon catheter prior to stenting. Pre-dilatation was performed in about half of the cases in our literature review. Although

Table 1. Cases of portal vein stenting after pancreaticoduodenectomy (clinical information)

No	Year	Author	Age	Sex	Primary disease	Chief complaint	Postoperative period (month)	Patency period (month)	Improvement of symptoms (Yes/ No)	Approach	Pre-dilatation	Embolization of collateral veins	Antithrombotic therapy		Complication
													During procedure	After stenting	
1	2001	Hiraoka [1]	66	F	PC	Melena, JV	12	84	Yes	PTP	Yes	NS	NS	NS	No
2	2002	Koike [2]	39	F	PNET	Melena, JV	5	21	Yes	PTP	NS	Yes	NS	1) 10000 IU heparin through catheter in the stent for 24 hours 2) 2–5 mg warfarin	No
3	2005	Ota [3]	64	M	AC	Melena, JV	98	32	Yes	PTP	Yes	No	NS	1) 15000 IU day^{-1} heparin for 4 days 2) Warfarin	No
4	2005	Sakai [4]	54	F	BDC	Melena, JV	16	4	Yes	PTP	Yes	No	NS	Warfarin for 4 days	No
5	2005	Shimizu [5]	57	M	PC	Intestinal bleeding	9	54	Yes	PTP	NS	NS	NS	NS	NS
6	2006	Takeuchi [6]	55	F	AC	Melena	8	24	Yes	PTP	Yes	Yes	NS	120000 IU of urokinase through catheter in the stent for 24 hours	NS
7	2006	Yasuda [7]	67	F	PC	Melena, JV	27 (days)	8	Yes	TIC	No	No	4000 IU heparin iv	1) 12000 IU heparin in 24 hours 2) 200 mg day^{-1} ticlopidine hydrochloride	No
8	2007	Hwang [8]	68	M	BDC	Melena, JV	25	14	Yes	PTP	NS	NS	No	No	No
9	2007	Ichihara [9]	64	F	BDC	GB	9	9	Yes	PTP	No	No	NS	240000 IU day^{-1} urokinase through catheter in the stent for 7 days	No
10	2009	Ellis [10]	58	M	PC	GB	15	4	Yes	PTP	No	No	NS	NS	No
11	2009	Kozono [11]	49	M	PC	Melena, JV	24	36	Yes	PTP	Yes	No	3000 IU heparin iv	NS	No
12	2010	Hirota [12]	63	F	BDC	Melena	13	3	Yes	TIC	Yes	No	70 IU kg^{-1} heparin iv	1) 12000 IU heparin in 24 hours 2) 200 mg day^{-1} aspirin and 75 mg day^{-1} clopidogrel for 3 months 3) 100 mg day^{-1} aspirin	No
13	2013	Kubo [13]	49	F	PC	Melena	20	NS	Yes	TIC	No	No	5000 IU heparin iv	1) 12000 IU day^{-1} heparin in a day 2) Warfarin	No
14	2013	Tsuruga [14]	54	M	PC	Ascites	9	60	Yes	TIC	NS	NS	No	1) 10000 IU day^{-1} heparin in 3 days 2) Warfarin 3) The warfarin was switched to aspirin 3 month later	NS
	2013	Tsuruga [14]	68	F	PC	Ascites	5	4	Yes	PTP	NS	NS	No	Same above	NS
15	2014	Sakurai [15]	67	M	PC	Melena, JV	24	7	Yes	TIC	No	Yes	No	1: 10000 IU day^{-1} heparin in 3 days 2: 100 mg day^{-1} aspirin	NS
16	2014	Sawatsubashi [16]	70	M	PC	Melena	8	NS	Yes	TIC	NS	NS	NS	NS	No
17	2014	Wakabayashi [17]	69	M	AC	Melena	36	64	Yes	PTP	Yes	NS	NS	NS	No
	2014	Wakabayashi [17]	75	F	PC	Melena	18	20	Yes	PTP	NS	NS	NS	NS	No
18	2015	Kitajima [18]	72	F	PC	Melena	8	7	Yes	PTP	Yes	NS	NS	7500 IU day^{-1} heparin	Melena (because of stent thrombosis)
19	2016	Matsui [19]	58	F	IPMN	Melena, anemia	49	6	No (Relapse after 6 months)	TIC	NS	Yes	Urokinase	1) Heparin 2) Warfarin	No
20	2018	Asai [20]	70	M	BDC	Melena	27	42	Yes	PTP	NS	Yes	NS	1) Heparin 2) Clopidogrel	No
21	2018	Sakabe [21]	86	M	DC	Melena	20	4	Yes	PTP	Yes	Yes	NS	Warfarin	No
22	2018	Sakamoto [22]	66	M	PC	GB	9	4	Yes	PTP	Yes	Yes	NS	NS	NS
23	2018	Sawai [23]	68	M	CCC	NS	2	NS	Yes	TIC	NS	NS	NS	1) Heparin for a week 2) Warfarin for 6 months	NS
	2018	Sawai [23]	68	F	PC	NS	52	NS	Yes	TIC	NS	NS	NS	Same above	NS
	2018	Sawai [23]	75	M	CCC	NS	3	NS	Yes	TIC	NS	NS	NS	Same above	NS
24	2018	Wakasa [24]	72	F	PC	Hematemesis	4	26	Yes	PTP	No	No	NS	100 mg aspirin	No
25	2019	Nishihara [25]	86	M	PC	Ascites, JV	12	28	Yes	TIC	Yes	Yes	Heparin iv	Antiplatelet drug (unknown)	No
26	2020	Kouzu [26]	75	F	AC	HE	6	3	Yes	PTP	No	No	NS	1) 10000 IU day^{-1} heparin in 6 days 2) Edoxaban	No

AC: ampullary cancer, BDC: bile duct cancer, CCC: cholangio cellular carcinoma, DC: duodenal cancer, EGV: esophageal varices, GB: gastrointestinal bleeding, HE: hepatic encephalopathy, IPMN: intraductal papillary mucinous neoplasm, iv: intravenous Injection, JV: jejunal varices, LD: liver dysfunction, NS: not stated, PC: pancreatic cancer, PNET: pancreatic neuroendocrine tumor. PTP: percutaneous transhepatic portal vein approach, TIC: transileocolic vein approach.

there is no consensus regarding the indication, pre-dilatation should be considered in cases where the stenosis or obstruction did not allow advancement of the stent delivery and sufficient expansion of the stent due to the rigid lesion.

Regarding the embolization of collateral veins, Kato et al. found that the presence of a collateral vein was a significant variable related to stent failure [36]. This result suggests that embolization of the collateral veins should be performed for stent patency. Sakurai et al. also recommended the embolization of jejunal varices to prevent re-rupture of the varices due to stent occlusion by tumor growth [15]. After Sakurai's report published in 2014, 6 of 8 evaluable cases (75%) underwent embolization of varices or collateral veins. There have been reports of malignant stenosis or the presence of a collateral vein being associated as a risk factor for stent occlusion [33, 36]. However, in our case, variceal embolization was not performed, stent patency was maintained for 7 months. This could be because sufficient portal-SMV flow was achieved after stenting, and there has been no tumor ingrowth or overgrowth. However, further careful observation is required.

Yamakado et al. reported three factors that were significantly associated with a higher probability of stent occlusion: splenic venous involvement, severe hepatic dysfunction, and portal venous obstruction [42]. This could be explained by a reduction in the portal blood flow after stenting [30]. A recently published study showed that pancreatic fistula was a multivariate derived risk factor [40].

Another factor of stent patency is anticoagulant or antiplatelet therapy [36]. There is controversy regarding therapy after portal venous stenting. Many investigators routinely or selectively use anticoagulant/antiplatelet therapy to prevent stent thrombosis, and heparin is most commonly used during and immediately after stenting. For management with oral medication, the use of antiplatelet and anticoagulant drugs was comparable. It is important to balance anticoagulant therapy with both stent patency and rebleeding from varices. No anticoagulation-related bleeding has been observed in previous studies [40].

Follow-up CT showed an interval decrease in the caliber

Table 2. Research articles including portal vein stenting after pancreaticoduodenectomy

No	year	Author	Total case (PD case)	Approach	Pre-dilatation	Embolization of collateral veins	Antithrombotic therapy		Complication (cases)
							During procedure	After stenting	
1	1999	Morita [27]	8 (1)	PTP	Yes	No	60000 UI of urokinase into the portal vein	200 mg day^{-1} Ticlopidine hydrochloride	Stent thrombosis (1) DIC (1)
2	2005	Takeshita [28]	5 (2)	PTP	Yes	No	No	No	Intraabdominal bleeding (1)
3	2005	Yamazaki [29]	4 (4)	PTP	Yes	NS	5000 IU heparin through catheter before stenting	NS	No
4	2009	Novellas [30]	14	PTP	Yes	No	No	No	Liver abscess (1)
5	2009	Woodrum [31]	18 (6)	PTP	No	No	NS	NS	NS
6	2009	Nio [32]	14 (3)	TIC	Yes	NS	NS	1) 5000 IU day^{-1} heparin for a week 2) Aspirin or warfarin for 1–3 months	NS
7	2011	Kim [33]	19 (9)	PTP	Yes	No	No	No	Sepsis (1) Liver abscess (1) Stent thrombosis (1)
8	2015	Hiyoshi [34]	5 (5)	PTP	No	No	2000–3000 IU heparin iv (2 cases)	1) 500 IU hr^{-1} heparin in a day 2) 100 mg day^{-1} aspirin for a few years	Technically failed (1) Hemorrhage related to PV puncture (2)
9	2016	Jeon [35]	22 (12)	PTP	NS	NS	NS	NS	NS
10	2017	Kato [36]	29	PTP (22) TIC (7)	No	No	No	1) Heparin for 0–7 days 2) Warfarin for at least 1 year	Stent thrombosis (3)
11	2017	Hyun [37]	11 (8)	PTP (10) PSP (1)	Yes	No	No	100 mg aspirin and 75mg clopidogrel for at least 3 months	No
12	2017	Shim [38]	22 (16)	PTP	Yes	5 cases	50 IU kg^{-1} heparin into the portal vein	NS	No
13	2019	Ohgi [39]	6 (6)	PTP	NS	No	NS	1) Heparin 2) Warfarin	No
14	2020	Lee [40]	60 (27)	PTP	Yes (not all)	7 cases	19 cases	100 mg aspirin and 75mg clopidogrel for at least 3 months	No

DIC: disseminated intravascular coagulation, NS: not stated, PD: pancreaticoduodenectomy, PSP: percutaneous trans-splenic portal vein approach, PTP: percutaneous transhepatic portal vein approach, PV: portal vein, TIC: transileocolic vein approach.

of the short gastric vein, and the gastroesophageal varices had disappeared (**Fig. 1c**). In addition, the marginal vein along the transverse colon and middle colic vein were dilated after stenting (**Fig. 1b, d**). We suspect that the retrograde flow of the splenic vein was altered and drained into the SMV via the marginal vein through the inferior mesenteric vein after stenting (**Fig. 3**). As a result, the portal blood flow increased, which helped maintain the stent's patency.

Several studies have reported that pancreatoduodenectomy with splenic venous transection does not always lead to sinistral portal hypertension, and the preservation of the right colic marginal vein could prevent the development of varices [43, 44]. It is thus speculated that portal venous stenting without preservation of the splenic venous flow is acceptable, as in the present case. However, there is a wide variety of abdominal veins, and the hemodynamic changes due to portal venous stenosis are still unknown.

In conclusion, portal stenting is useful for the management of extrahepatic portal venous stenosis, resulting in portal hypertension and gastrointestinal bleeding. Anticoagulant or antiplatelet therapy following portal venous stenting is required in the majority of cases to prevent acute portal venous stenting, although it has a low risk of inducing recurrent variceal bleeding. Adjunctive jejunal variceal embolization can possibly be omitted in selected cases to obtain sufficient portal-SMV flow reconstruction.

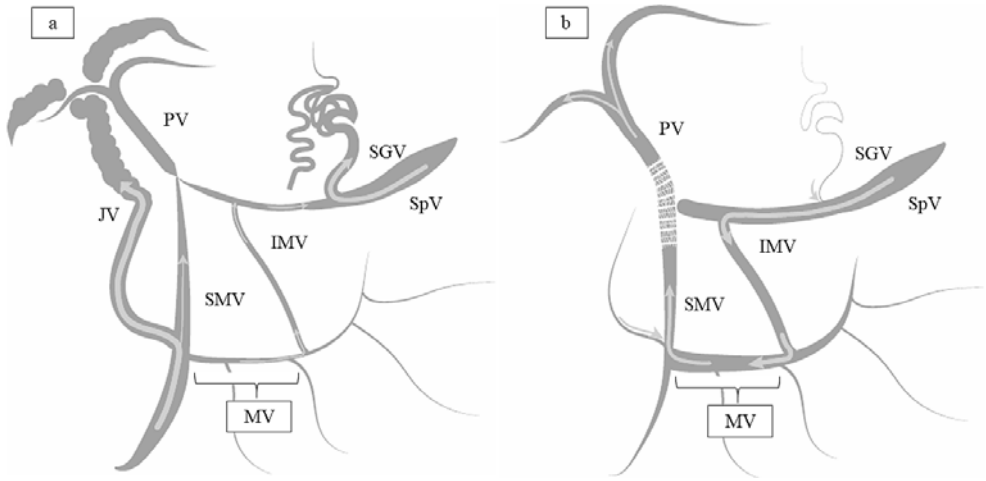

Fig. 3. The scheme of blood flow changes before and after stent placement. (a) There were two collateral pathways before stent placement. The first involved the retrograde flow of the splenic vein (SpV), followed by the short gastric vein (SGV). The second involved the retrograde flow of the jejunal vein (JV), which formed JVs and was followed by the peribiliary plexus. (b) The retrograde flow of the SpV and jejunal vein was changed after stent placement. The blood flow of the SpV drained mainly into the SMV via the marginal vein along the transverse colon through the IMV after stent placement. The blood flow of the jejunal vein drained into the SMV, after which the JVs disappeared. IMV: inferior mesenteric vein, JV: jejunal vein, MV: marginal vein, PV: portal vein, SMV: superior mesenteric vein.

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

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