# ORIGINAL ARTICLE



# Portomesenteric vein thrombosis after gastric surgery

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### Abstract

Background Postoperative portomesenteric venous thrombosis (PMVT) is a rare but potentially serious complication of gastric surgery. This study analyzed the incidence, characteristics, risk factors, and outcomes of PMVT following gastric surgery.

Methods Medical records of patients who underwent gastric surgery between January 2007 and December 2012 were reviewed retrospectively. The risk factors of PMVT were analyzed by a logistic regression analysis with control group matched 1:4 by age, sex, and cancer stage and by a Poisson regression analysis with unmatched control group. The resolution rate of PMVT in 12 months was compared between the treatment group and the nontreatment group. Results The total incidence of PMVT after gastric surgery was 0.67 % (31/4611). Most (54.84 %) PMVT cases were detected within 1 month postoperatively. No accompanying deep vein thrombosis (DVT) was noted. Multivariate comparison with 1:4 matched control showed that combined splenectomy, synchronous malignancy, and intra-abdominal complication were independent risk factors. Advanced stage, combined splenectomy, and synchronous malignancy were independent risk factors in Poisson regression analysis using unmatched controls. The resolution rate of PMVT was not different from patients Conclusions PMVT after gastric surgery was associated with advanced cancer stage, combined splenectomy, and synchronous malignancy, but it was not related to laparoscopy or DVT. Significant differences in the natural course of PMVT were not found between the treatment group and observation group.

**Keywords** Portal vein · Venous thrombosis · Gastrectomy · Splenectomy

# Introduction

Portomesenteric venous thrombosis (PMVT) is a rare but potentially lethal complication of abdominal surgery [1]. PMVT occurs after several surgeries, such as liver transplantation, splenectomy, portosystemic shunt surgery, and gastric surgeries, including bariatric surgery [1]. Such cases exhibit a wide spectrum of clinical manifestations, including as an incidental finding in asymptomatic patients, as nonspecific abdominal pain, and as life-threatening bowel infarction [2].

Several studies investigated PMVT after laparoscopic or laparoscopic bariatric surgery, because pneumoperitoneum during laparoscopic surgery may induce pressure on the portomesenteric vasculature, which leads to thrombus formation [2, 3].

We suggest the following possible risk factors for PMVT based on Virchow's triad. Pneumoperitoneum during laparoscopic surgery may induce venous stasis [1, 3], which may be associated with prolonged operation



treated with anticoagulation (n = 6) or antiplatelet therapy (n = 1) and were not significantly different with those of the untreated group [85.7 % (6/7) vs. 82.3 % (14/17), p = 0.935] during 1-year follow up.

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time in a fixed position and obesity [4]. Perigastric vessel wall injury may be caused by a more aggressive dissection of lymph nodes during cancer surgery [2]. Advanced-stage cancer, a history of deep vein thrombosis (DVT) [5], and a combination of hereditary defects of anticoagulation factors may all be associated with a hypercoagulable state [6]. These factors suggest that gastric cancer surgery is more closely related to PMVT than bariatric surgery. However, little is known about the incidence or clinical course of PMVT after general gastric surgery. Therefore, this study analyzed the incidence and clinical manifestations of PMVT after gastric surgery to identify the risk factors of PMVT after gastric surgery.

## Methods

We retrospectively reviewed the medical records of patients who underwent gastric surgery at Seoul National University Hospital between January 2007 and December 2012 (n = 4611). PMVT was identified by the presence of 'portal vein (PV)/superior mesenteric vein (SMV) thrombosis with/without splenic vein (SV) thrombosis' on computed tomography (CT) or ultrasonography (USG) taken within 1 year after gastric surgery (excluding patients who presented with splenic vein thrombosis only). An expert radiologist reviewed the CT images. Clinicopathological characteristics, including age, sex, body mass index (BMI), diagnosis of stomach and the stage of disease, underlying diseases, operation method (laparoscopy vs. open), operation time, extent of surgery, and intra-abdominal complications corresponding to ss≥grade II by Clavien-Dindo classification, were compared between PMVT patients and non-PMVT patients.

We used descriptive analysis for the clinical features of PMVT patients, and chi-square and t tests for comparison between the patient and control group. Considering a rare incidence of the PMVT, the Poisson regression analysis was performed to identify risk factors of PMVT among all the subjects. Additionally, a control group of 1:4 matched by age (range, patient's age  $\pm 1$  year), sex, and cancer stage was compared for logistic regression analysis to increase statistical power. All risk factors that were found significant (p > 0.05) in univariate analyses were included in the multivariate analysis. The Kaplan-Meier and log-rank tests were used for comparisons of the efficacy of treatment plans. All statistical analyses were performed using SPSS version 21. (IBM, Sommers, NY, USA) All tests were considered statistically significant at p < 0.05.

This study was conducted following IRB approval (IRB number 1509-080-705).



PMVT was detected in 31 patients (0.67 %) of the 4611 patients who underwent gastric surgery (Table 1). The incidence of PMVT was not associated with age (p = 0.671) or sex (p = 0.855). No comorbid DVT was found in the PMVT group. Significantly more PMVT was found in cases of open surgery (n = 28/3256, 0.86%) than laparoscopic surgery (n = 3/1355, 0.22 %) (p = 0.016). The incidence of PMVT was correlated with disease stage in a univariate analysis of all the gastric cancer patients (n = 4579, p < 0.001). Only patients who were diagnosed with early gastric cancer (EGC) preoperatively underwent laparoscopic surgery during the study period; according to the principles of research and analysis, they were classified according to pathological criteria. Limited analysis of the EGC patients (n = 2608) revealed no significant differences in the incidence of PMVT between the laparoscopy and open groups [0.25 % (3/1190) vs. 0.35 % (5/1418)] (p = 0.462). Analysis of advanced gastric cancer (AGC) patients also did not reveal a significant difference (p = 0.169). Because laparoscopic surgery was only indicated for EGC during the study period, the majority of AGC patients who underwent laparoscopic surgery were assessed as EGC preoperatively but were found to be AGC in the postoperative pathology report.

Two cases of PMVT were identified in non-gastric cancer surgeries, a gastrectomy for complicated peptic ulcer and a combined gastrectomy with another malignancy. Both cases were combined with hepatocellular carcinoma (HCC) and underwent open surgery.

Table 2 shows information about diagnosis and treatment of the 31 patients with PMVT. Thrombosis was found at various locations in the portomesenteric vasculature with involvement of one or more vessels (Fig. 1). The most common types were involvement of a single peripheral portal vein (n = 13, 41.94 %) and combined involvement of the main, right, and left portal veins (n = 13, 41.94 %). Seventeen (54.84 %) asymptomatic cases were detected incidentally on routine imaging studies. Eleven patients displayed clinical symptoms and signs such as fever (n = 7), abdominal pain (n = 2), change of drain color (n = 1), and altered mental state (n = 1), but whether these signs and symptoms were directly related to PMVT was not clear.

Six (19.35 %) patients received prophylactic anticoagulation therapy using low molecular weight heparin perioperatively, and 7 patients were treated using anticoagulation or antiplatelet therapy after the detection of PMVT.

Table 3 shows the univariate analysis of risk factors between patients and the 1:4 matched and unmatched



**Table 1** Incidence of portomesenteric vein thrombosis (PMVT)

	Total n	PMVT, n (%)	p value <sup>a</sup>
Incidence	4611	31 (0.67)	
Age, mean $\pm$ SD (year)	$59.61 \pm 11.92$	$60.52 \pm 8.33$	0.671
-40	306	1 (0.33)	0.746
41–60	2021	14 (0.69)	
61–	2284	16 (0.70)	
Sex			
Male	3052	21 (0.69)	0.855
Female	1559	10 (0.64)	
Diagnosis			
Gastric cancer	4579	29 (0.63)	
Stage I	2888	9 (0.31)	< 0.001
Stage II	661	6 (0.91)	
Stage III	640	8 (1.25)	
Stage IV	390	6 (1.54)	
Non-gastric cancer	32	2 (6.25) <sup>b</sup>	
Underlying disease			
Hypertension	763	7 (0.92)	
Diabetes	187	5 (2.67)	
Cardiac disease	61	1 (1.64)	
Cerebrovascular accident	25	0 (0)	
BMI, mean $\pm$ SD (kg/m <sup>2</sup> )	$23.72 \pm 3.14$	$23.14 \pm 3.65$	
<24	2477	22 (0.89)	0.054
≥24	2134	9 (0.42)	
Operation method			
Open	3256	28 (0.86)	0.016
Laparoscopy	1355	3 (0.22)	
Operation method in early gastric cancer	2608	8	
Open	1418	5 (0.35)	0.462
Laparoscopy	1190	3 (0.25)	
Operation method in advanced gastric cancer	1971	21	
Open	1812	21 (1.16)	0.169
Laparoscopy	159	0 (0)	
Operation method in non-gastric cancer	32	2	
Open	26	2 (7.69)	0.655
Laparoscopy	6	0 (0)	

SD standard deviation, BMI body mass index

control group. Mean patient age was 60.52 years (age range, 52.19–68.85 years), and the male-to-female ratio was 2.1:1.0. Operative time, splenectomy, synchronous malignancies, and intra-abdominal complications were significantly associated with the development of PMVT in both unmatched and 1:4 matched analysis. However, statistical significance in the relationship of PMVT with operation method, combined resection (except spleen), and radicality was only identified in the unmatched analysis, but not in the 1:4 matched analysis that stratified the stage

of the gastric cancer. Intra-abdominal complications included intra-abdominal fluid collection (n=5), duodenum stump leakage (n=2), tractitis in drain insertion site (n=1), and IVC thrombosis (n=1) in the PMVT group, and intra-abdominal fluid collection (n=5), anastomosis leakage (n=4), duodenum stump leakage (n=1), and gastrointestinal luminal bleeding (n=1) in the 1:4 matched control group.

Multivariate risk factors analysis (Table 4) demonstrated that splenectomy and synchronous malignancy were



<sup>&</sup>lt;sup>a</sup> Comparison with patients without PMVT

<sup>&</sup>lt;sup>b</sup> Both (2) patients had hepatocellular carcinoma

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**Table 2** Diagnosis and treatment of PMVT (n = 31)

	n (%)
Type	
Peripheral PV	13 (41.94)
Main PV + Rt PV + Lt PV combined	13 (41.94)
SMV	2 (6.45)
RAPV + RPPV + Lt PV combined	1 (3.23)
Rt PV + SMV + SV combined	1 (3.23)
Peripheral PV + SV combined	1 (3.23)
Postoperative period of detection (days)	
0–30	17 (54.84)
31–60	5 (16.13)
61–90	1 (3.23)
91–120	2 (6.45)
121–150	2 (6.45)
151–	4 (12.90)
Reason for identification of PMVT	
Routine follow up	17 (54.84)
Fever	7 (22.58)
Changes in laboratory finding	3 (9.68)
Abdominal pain	2 (6.45)
Change of drain color	1 (3.23)
Altered mentality	1 (3.23)
Prophylactic anticoagulation	
Yes	6 (19.35)
No	25 (80.65)
Treatment	
Anticoagulation therapy	6 (19.35)
Antiplatelet therapy	1 (3.23)
No treatment	24 (77.42)

PV portal vein, SMV superior mesenteric vein, RAPV right anterior portal vein, RPPV right posterior portal vein, SV splenic vein

independent risk factors in both matched analysis and unmatched analysis. Intra-abdominal complication was a significant risk factor in the 1:4 matched analysis but not in the unmatched analysis using Poisson regression model. Advanced cancer stage was identified as a significant risk factor in unmatched analysis also.

We followed up the clinical course of 24 patients for 1 year (mean, 209 days) after PMVT detection and excluded 7 patients who had tumor thrombi in PV, SMV, and SV (Fig. 2). Fourteen of the 17 patients experienced thrombosis resolution without treatment, and 6 of the 7 patients who received anticoagulation or antiplatelet therapy experienced resolution during the 1-year follow-up period (Fig. 3). There was no significant difference in the percentage of remaining thrombosis between the two groups (p = 0.935).

Two patients did not experience thrombosis resolution during the follow-up period. One patient was a 60-yearold woman who underwent laparoscopy-assisted distal gastrectomy with a diagnosis of EGC. The final pathological result was stage Ia. Portal vein thrombosis at S5 and pulmonary thromboembolism (PTE) at the right lower lobe were found on CT, with no evident symptoms on postoperative day 95. The PTE had disappeared on the CT that was performed 5 months later, but the portal vein thrombosis at S5 was still present. The other patient was a 48-year-old woman who underwent extended total gastrectomy followed by chemoradiation therapy for the treatment of AGC. A main portal vein thrombosis was incidentally found on CT 4 months later. Ascites increased gradually, and peritoneal seeding and metastasis to the left ureter and ascending colon were found on CT 8 months postoperatively. This patient expired 1 year postoperatively despite conservative management with double J catheter insertion and percutaneous nephrostomy.

No significant gastrointestinal bleeding events were identified during the follow-up period. The average levels of preoperative hemoglobin in the treated and untreated groups were 12.84 (6.10–15.40) and 12.85 (8.90–15.70), respectively, without statistical significance (p = 0.991). The average levels of hemoglobin 1 year post surgery in the treated and untreated groups were 10.84 (9.50–12.00) and 11.01 (8.60–13.90), respectively, which was not significantly different between groups (p = 0.761).

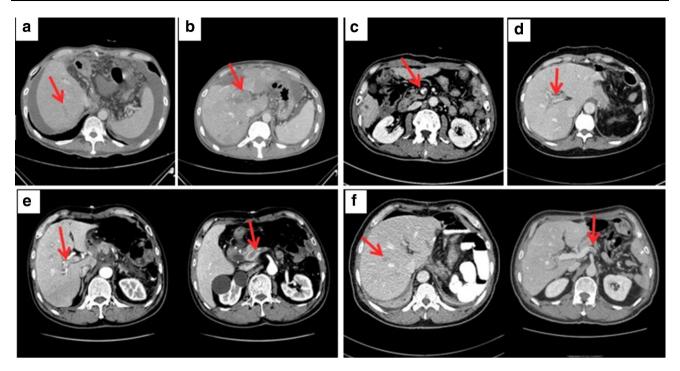
# **Discussion**

This study analyzed the incidence, clinical manifestations, and risk factors of PMVT after gastric surgery. Previous studies investigated the incidence rate of PMVT after laparoscopic bariatric surgery (0.3 %) [1] or splenectomy (12.3 %) [7, 8], but few reports have examined PMVT incidence after other types of gastric surgery. The PMVT incidence rate in our study was 0.6 %.

Significantly more PMVT developed in open surgery than laparoscopic surgery (p=0.016) in univariate unmatched analysis, and laparoscopic surgery also did not increase the incidence of PMVT in stratified analyses for EGC and AGC patients in this study. However, most AGC cases in this series were diagnosed as EGC preoperatively and found to be AGC postoperatively. Therefore, the effect of laparoscopic surgery for AGC in PMVT requires further validation in laparoscopic gastrectomy series, which was indicated only in preoperatively staged AGC patients.

The relatively higher PMVT incidence (6.25 %) in the non-gastric cancer subgroup was the result of two patients who also presented with HCC. No further PMVT was identified in the non-gastric cancer subgroup regardless of operative type.





**Fig. 1** Type of portomesenteric vein thrombosis (PMVT). **a** Peripheral portal vein thrombosis (41.94 %). **b** Main portal vein, right portal vein, and left portal vein thrombosis combined (41.94 %). **c** Proximal superior mesenteric vein thrombosis (6.45 %). **d** Right anterior portal

vein, right posterior portal vein, and left portal vein thrombosis combined (3.23 %). e Right portal vein, superior mesenteric vein, and splenic vein thrombosis combined (3.23 %). f Peripheral portal vein and splenic vein thrombosis combined (3.23 %). *Arrow* thrombosis

The clinical manifestations of PMVT are nonspecific epigastric pain, nausea, vomiting, diarrhea, fever, and gastrointestinal tract bleeding [1]. In this study, 54.8 % of cases were asymptomatic, and PMVT was detected using routine imaging studies. The routine imaging follow-up process in our institute include alternative CT and sonography every 4 months post surgery for EGC and every 3 months post surgery for AGC. The detection of PMVT on unscheduled imaging studies was caused by specific symptoms, including fever, abnormal laboratory findings, and abdominal pain (Table 2). Three of the 7 patients with fever had fluid collection around the anastomosis on the same CT scan, but the other 4 of 7 patients did not have any other cause of fever. PMVT could not be excluded as the cause of fever in these 3 patients. One patient with abdominal pain had underlying HCC, but we could not confirm whether the HCC caused the abdominal discomfort.

Because of the rare incidence of PMVT, we used two methods to identify risk factors of PMVT. First, we used Poisson regression analysis, which is known to be useful in rare events. Also, we matched the PMVT subjects with the control group using a ratio of 1:4 to offset the effect of a different stage, because the number of subjects in the control group was too large compared to the patients with PMVT, and a matching ratio larger than 1:4 does not significantly increase the statistical power [9].

We matched the patients by cancer stage, because it was shown to be a strong disease factor related with PMVT in univariate analysis (Table 3), but the distribution of the PMVT group and the unmatched control group was different (Table 1) Graham J. Caine reported that tumor cells produce their own procoagulant factors, which activate the coagulation cascade directly and stimulate the prothrombotic properties of other blood cell components [10]. Therefore, they interact with all parts of the hemostatic system. We also used age and sex as matching parameters although there was no correlation in univariate analysis, because advanced age (>60 years) has been shown as an independent risk factor of venous thromboembolism in previous studies [11, 12], and some reports suggest that sex hormones affect thrombosis [13, 14].

Virchow's triad provides that increased intra-abdominal pressure during laparoscopic surgery may induce thrombosis in portomesenteric vessels, which was found in previous studies. Insufflation of the abdomen and increased intra-abdominal pressure decrease mesenteric and portal venous flow via direct pressure-induced compression. This decrease in venous flow varies from 35 % to 84 %. Insufflation with carbon dioxide induces a more substantial decrease in venous flow than with other inert gases [2, 3]. This study revealed no significant difference in the incidence of PMVT between laparoscopic and open surgeries in cases of EGC.



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**Table 3** Univariate analysis of risk factors

	PMVT	1:4 matched control		Unmatched control	
	$n = 31 \ (\%)$	$n = 124 \ (\%)$	p value	$n = 4580 \ (\%)$	p value
Age	$60.52 \pm 8.33$	$61.32 \pm 7.87$	Matched	$53.60 \pm 11.94$	0.671
Sex					
Female	10 (32.26)	40 (32.26)	Matched	1549 (33.82)	0.855
Male	21 (67.74)	84 (67.74)		3031 (66.18)	
Operation metho	od				
Laparoscopy	3 (9.68)	21 (16.94)	0.319	1352 (29.52)	0.016
Open	28 (90.32)	103 (83.06)		3228 (70.48)	
Operation name	a				
PPG	0 (0.00)	5 (4.03)	0.471	455 (9.93)	0.604
PG	1 (3.22)	4 (3.23)		214 (4.67)	
TG	13 (41.94)	43 (34.68)		836 (18.25)	
DG	17 (54.84)	72 (58.06)		3075 (67.14)	
Stage					
I	9 (31.03)	46 (37.10)	Matched	2879 (63.27)	< 0.001
II–IV	20 (68.97)	78 (62.90)		1671 (36.73)	
Operative time	(min)				
>240	10 (32.26)	20 (16.13)	0.016	769 (16.81)	0.022
≤240	21 (67.74)	104 (83.87)		3810 (83.19)	
BMI (kg/m <sup>2</sup> )					
≥24	9 (29.03)	59 (47.58)	0.063	2122 (46.40)	0.054
<24	22 (70.97)	65 (52.49)		2455 (53.60)	
Prophylactic and	ticoagulation				
Yes	6 (19.35)	32 (25.81)	0.455	876 (19.23)	0.975
No	25 (80.65)	92 (74.19)		3703 (80.87)	
Splenectomy					
Yes	11 (35.48)	13 (10.48)	0.001	163 (3.56)	< 0.001
No	20 (64.52)	111 (89.52)		4417 (96.44)	
Combined resec	etion (except spleen) <sup>b</sup>	` '		` ,	
Yes	10 (32.26)	25 (20.16)	0.492	700 (15.28)	0.014
No	21 (67.74)	99 (79.84)		3880 (84.72)	
Lymph node dis	ssection				
<d2< td=""><td>11 (35.48)</td><td>47 (37.90)</td><td>0.803</td><td>1918 (41.88)</td><td>0.472</td></d2<>	11 (35.48)	47 (37.90)	0.803	1918 (41.88)	0.472
≥D2	20 (64.52)	77 (62.10)		2662 (58.12)	
Radicality					
R2	4 (12.90)	13 (10.48)	0.701	139 (3.03)	0.015
R0 & R1	27 (87.10)	111 (89.52)		4441 (96.97)	
Synchronous ma	` '	` '		` ,	
Yes	7 (22.58) <sup>c</sup>	3 (2.42) <sup>d</sup>	< 0.001	24 (0.52) <sup>e</sup>	< 0.001
No	24 (77.42)	122 (97.58)		4556 (99.45)	
Intra-abdominal	` '	· · · · · · · ·		X = /	
Yes	9 (29.03)	11 (8.87)	0.006	657 (14.34)	0.027
No	22 (70.97)	113 (91.13)		3923 (85.66)	

 $<sup>^{\</sup>mathrm{a}}$  DG distal gastrectomy, TG total gastrectomy, PG proximal gastrectomy, PPG pylorus-preserving gastrectomy



<sup>&</sup>lt;sup>b</sup> Gall bladder (n = 400), colon (n = 98), pancreas (n = 89), ovary (n = 46), liver (n = 36), mesocolon (n = 29), adrenal (n = 21)

 $<sup>^{\</sup>rm c}$  Hepatocellular carcinoma (n=5), esophageal cancer (n=1), pancreas neuroendocrine tumor (n=1)

<sup>&</sup>lt;sup>d</sup> Prostate cancer (n = 1), papillary thyroid carcinoma (n = 1), hepatocellular carcinoma (n = 1)

<sup>&</sup>lt;sup>c</sup> Colorectal cancer (n = 7), renal cell carcinoma (n = 4), papillary thyroid carcinoma (n = 3), hepatocellular carcinoma (n = 2), lung cancer (n = 2), pancreas cancer (n = 2), prostate cancer (n = 1), bladder cancer (n = 1), ovary cancer (n = 1), chronic myeloid leukemia (n = 1)

Table 4 Multivariate analysis of risk factors

Factors	1:4 matched	1:4 matched $(n = 124)$			Unmatched (Poisson regression) $(n = 4580)$		
	RR	95 % CI	P value	RR	95 % CI	P value	
Operation method	d						
Open				Reference	0.216-2.531	0.630	
Laparoscopy				0.739			
Stage							
I	Matched			Reference	1.017-4.919	0.045	
II–IV				2.236			
Operation time (1	min)						
≤240	Reference	0.353-3.226	0.908	Reference	0.263-2.453	0.701	
>240	1.067			0.804			
Splenectomy							
No	Reference	2.520-20.317	< 0.001	Reference	3.233-22.684	< 0.001	
Yes	7.155			8.563			
Combined resect	ion (except spleen	)					
No				Reference	0.230-2.137	0.533	
Yes				0.702			
Radicality							
R0 and R1				Reference	0.621-7.321	0.229	
R2				2.133			
Synchronous mal	lignancies						
No	Reference	3.493-76.475	< 0.001	Reference	10.451-81.785	< 0.001	
Yes	16.343			29.236			
Intra-abdominal o	complications						
No	Reference	1.527-15.378	0.007	Reference	0.609-3.557	0.390	
Yes	4.846			1.472			

The total incidence rate of PMVT after splenectomy was 12.3 % in a study by Krauth et al. [8]. Combined splenectomy was significantly associated with the development of PMVT in this study, which was consistent with previous studies. This result may be associated with blood stasis at the stump of the splenic vein. Cul de sac formation in the splenic vein stump after splenectomy induces thrombosis that can extend to the portal vein or superior mesenteric vein [7]. Elevated platelet counts after splenectomy are also associated with hypercoagulation [7].

Synchronous malignancy was a significant risk factor for PMVT. Malignancy in other organs, not only HCC, which can present with tumor thrombi, activates the coagulation cascade [10].

Intra-abdominal complication showed statistical significance in association with PMVT by univariate analyses as well as by a logistic regression multivariate analysis in 1:4 matched comparisons, although not in Poisson regression analysis, however. The different result may be caused by association between complications and other factors such as cancer stage, and the limitation of matching analysis is that it cannot sufficiently represent a variety of intra-abdominal complications in the total control group.

The low number of cases with prophylactic anticoagulation was caused by a lack of reliable data on the incidence of postoperative DVT in abdominal surgeries in Asian populations and experts' opinions that the risk of perioperative bleeding is greater than the risk of DVT. Routine prophylactic anticoagulation was adopted in our institution following the suggestion of the 2010 "Korean guideline for the prevention of VTE" [15, 16].

PMVT treatment after laparoscopic surgery is not fully established. Prompt initiation of anticoagulation therapy is the current recommendation for the treatment of acute PMVT [2]. Previous studies recommended a 6- to 12-month period of anticoagulation therapy [2]. One study demonstrated that anticoagulation therapy led to a two-thirds reduction in the risk of thrombotic events [17] and complete or partial recanalization in 90 % of patients [2, 18]. Anticoagulation therapy alone provides a therapeutic benefit in PMVT because it accelerates recanalization of the portomesenteric vein and reduces the risk of further thrombosis [2, 18]. However, anticoagulation therapy increases the risk of gastrointestinal bleeding, especially in patients with portal vein thrombosis and portal vein hypertension [2, 17]. No patients in this study experienced



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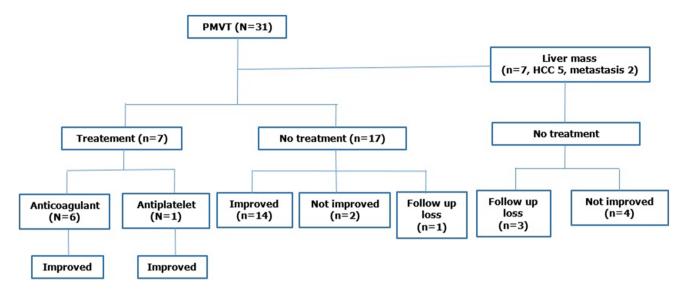
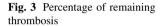
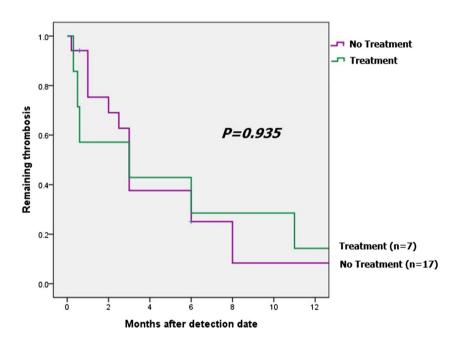


Fig. 2 Clinical course of PMVT





clinical symptoms of gastrointestinal bleeding or signs of a sudden decrease in hemoglobin level after anticoagulation therapy, but the risk of bleeding during anticoagulation therapy should always be considered.

The potential risks and benefits of the administration of anticoagulant agents should be considered [17]. This study demonstrated no significant difference in thrombosis resolution during follow-up of the clinical course of PMVT between the treated and untreated groups. Most of the PMVT cases were detected on routine follow-up imaging studies and clinical symptoms, and signs of PMVT were relatively not severe. Most patients in the untreated group experienced a spontaneous resolution of thrombosis.

In conclusion, the incidence rate of PMVT after gastric surgery was 0.67 %. The development of PMVT was significantly associated with advanced-stage gastric cancer, combined splenectomy, and synchronous malignancy, but the incidence was not associated with opermethod (laparoscopy VS. open), obesity, prophylactic anticoagulation therapy, or the presence of DVT. No difference was found in the resolution course of PMVT between the treated and observation groups in this retrospective series. However, larger prospective studies are needed to confirm the efficacy of anticoagulation treatment for asymptomatic PMVT after gastric surgery.



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