

Thrombosis of the Superior Mesenteric and Portal Veins after Splenectomy and/or Distal Splenorenal Shunts

Katsuhide ITO¹⁾, Akira NAITO¹⁾, Tomoko SAITO¹⁾, Kumiko NAITO¹⁾, Haruhito FUKUOKA¹⁾,
Koichi FUJIKAWA¹⁾, Masaki MORI¹⁾, Shoko ITO¹⁾, Shizutomo KATSUTA¹⁾,
Osamu KODAMA²⁾, Toshimasa ASAHARA²⁾, Kiyohiko DOHI²⁾, Komei TODANI³⁾,
Toshio ICHIKI³⁾, Hirofumi YUKAYA⁴⁾ and Yuichiro OGAWA⁴⁾

1)Department of Radiology, Hiroshima University School of Medicine, 1-2-3, Kasumi, Minami-ku,
Hiroshima 734, Japan

2)The Second Department of Surgery, Hiroshima University School of Medicine, 1-2-3, Kasumi,
Minami-ku, Hiroshima 734, Japan

3)Department of Radiology, Hiroshima Red Cross Hospital, Naka-ku, Hiroshima 730, Japan

4)Department of Surgery, Hiroshima Red Cross Hospital, Naka-ku, Hiroshima 730, Japan

ABSTRACT

During the past ten years, 180 patients with liver cirrhosis and hematologic disorders underwent splenectomy and distal splenorenal shunts, 56 of whom were evaluated by CT, ultrasonography (US) and/or angiography, postoperatively at Hiroshima University Hospital and Hiroshima Red Cross Hospital. Five patients were asymptomatic; thrombosis was observed in 10; and two died of liver failure. Three patients each complained of postprandial epigastralgia with diarrhea, fever, and general malaise.

Angiography was performed to evaluate the patency of the shunts; CT and US, to follow up the liver cirrhosis - not the portal vein thrombosis. Since thrombosis of the portal venous system occurs frequently during surgery, and since it causes marked changes in portal vein hemodynamics, the portal and superior mesenteric veins must be observed by US and CT for the early postoperative detection and treatment, to avoid major surgery.

Key words: Splenectomy, Distal splenorenal shunt, Portal vein thrombosis

Thrombosis is a well-known complication post splenectomy in portal hypertension and hematologic disorders in the systemic venous system, including pulmonary embolism and deep-vein thrombosis of the lower extremities or portal veins^{10,15}.

Portal vein thrombosis is not rare; it may be more frequent than previously reported^{18,25}. Injury to the vascular endothelium of the portal venous system, changes in hemodynamics of the portal venous system and changes in the components of the blood including postsplenectomy thrombocytosis are among its causes¹¹.

Such postoperative portal vein thrombosis usually does not affect the postoperative course significantly. Symptoms sometimes resolve spontaneously; however, they can be fatal^{1,19} and aggressive therapy may be indicated. This is a report of correlations between clinical symptoms, thrombosis sites and the degree of obstruction of the portal venous systems with or without cavernous transformation (CTF).

SUBJECTS AND METHOD

Subjects of this study were 180 patients who underwent splenectomy or distal splenorenal shunts (DSRS) at Hiroshima University Hospital, and

Table 1. Study Subjects with Diagnosis

Liver cirrhosis		53
Splenectomy	21	
Distal splenorenal shunt	32	
Idiopathic portal hypertension		2
Splenectomy accompanied by gastrectomy and pancreatectomy		1
total		56

Hiroshima Red Cross Hospital during the past decade. They consisted of 149 cases with liver cirrhosis, 12 with idiopathic portal hypertension, 7 blood discrasias, 1 splenic tumor and 17 splenectomies associated with gastrectomy and pancreatectomy. The portal venous systems of 56 of them were evaluated by angiography, CT and/or US (Table 1). The obstructions were categorized as complete and partial, and the sites were classified as portal vein (PV), superior mesenteric vein (SMV) and splenic vein (SV). Ultrasonographically, portal thrombosis was assessed by CTF of the portal vein, by the presence of echogenic material within the portal system and by nonvisualization of a patent portal system^{7,20}. With enhanced CT, intraluminal low density areas and ring enhancements of the venous walls were evaluated²⁴. Angiographically, this was

Table 2. Study Subjects by Sex, Age, Symptoms, Site, Surgery, Findings, and Course.

Case	Sex	Age	Dx	Symptoms	Surgery	Duration to detect	Site and Degree	Cavernous transformation	Course
1.	M	45	L.C	diarrhea epigastralgia	Spn.	2Y 2M	SMV complete	(+)	alive
2.	F	60	IPH	liver failure	Spn.	1M	PV, intrah.pv complete	(-)	2M dead
3.	M	51	L.C	liver failure	Spn.	3M	PV occlusion	(-)	dead
4.	F	54	L.C	(-)	Spn.	7Y	SMV partial	(-)	alive
5.	M	66	L.C	(-)	Spn.	4Y 1M	PV occlusion SMV partial	(-)	1M dead
6.	M	47	L.C gastric ca.	fatigue	Spn.	2M	PV, SMV partial	(-)	alive
7.	F	61	L.C	(-)	Spn.	3Y	PV, SMV partial	?	alive
8.	M	62	L.C	fever	DSRS	2Y	PV, SV occlusion	(+)	alive
9.	M	66	L.C	(-)	DSRS	1Y 2M	PV occlusion	(+)	1Y dead
10.	M	50	L.C	(-)	DSRS	4Y 2M	SMV occlusion	?	alive

L.C : liver cirrhosis, IPH : idiopathic portal hypertension PV : portal vein, SMV : superior mesenteric vein, SV : splenic vein, intrah. pv : intrahepatic portal vein, partial: partial occlusion, Spn. : splenectomy, DSRS : distant splenorenal shunt, course : duration after diagnosis, M : month, Y : year

diagnosed by nonvisualization of the portal vein or filling defects in the portal venous system, with or without CTF¹⁷⁻¹⁹). Portal vein thrombosis extending from the intrahepatic portal vein, and associated with hepatoma, were excluded because of their being indistinguishable from tumor thrombi.

RESULTS

Thrombosis of the SMV, SV, and/or PV were observed in 10 patients (Table 2). Ten patients with thrombosis of the portal venous system were comprised by nine liver cirrhotics, and one idiopathic portal hypertension, corresponding to 17.9% (10/56). Eight patients were post splenectomy and 2 patients were post DSRS. The intervals between surgery and the evaluations for portal thrombosis of ten patients ranged from 1 month to 7 years, the average being 28 months. Patients who were more than 1 year postoperative were not always asymptomatic; two patients had symptoms and signs including abdominal pain, diarrhea, fever, and general malaise and both of them had complete occlusion of their SMV and SV with CTF (Table 2 and 3). Five patients had no symptoms, but one had complete occlusion of the PV without CTF. He was deceased one month later.

Table 3. Portal Thrombosis and the Presence of Symptoms

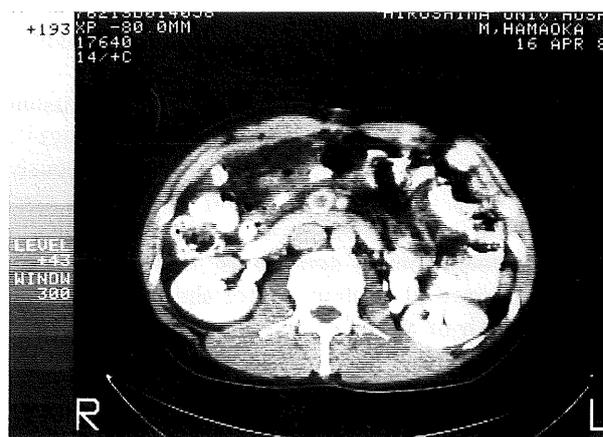
		symptomatic	asymptomatic
Less than 3 months	complete occlusion	2	0
	partial occlusion	1	0
	occlusion		
more than 1 year	complete occlusion	2	2
	partial occlusion	0	3
	occlusion		
		5	5

Symptoms occurred within 3 months postoperatively in three patients. All had complete or partial occlusion of PV without CTF. They had hepatitis and liver failure, and two of them died of the latter.

CASES REPORTS

Case 1. This 45-year-old man underwent splenectomy because of repeated hematemesis and melena secondary to liver cirrhosis. Drainage of pancreatic juice occurred after surgery. Ten months after splenectomy, he underwent removal of a cerebral abscess.

Twenty-six months postsplenectomy, he was admitted for the evaluation of postprandial diarrhea and abdominal pain. CT revealed thrombosis of the SMV (Fig. 1A), and angiography revealed the SMV to be completely obstructed about 5 cm from the second ileal tributary to the middle colic vein, and CTF transformation was noted (Fig. 1B). During the venous phase following injection of the inferior mesenteric artery, the proximal portion of the

**Fig. 1A.** Ring enhancement of superior mesenteric vein and intraportal low density mass.

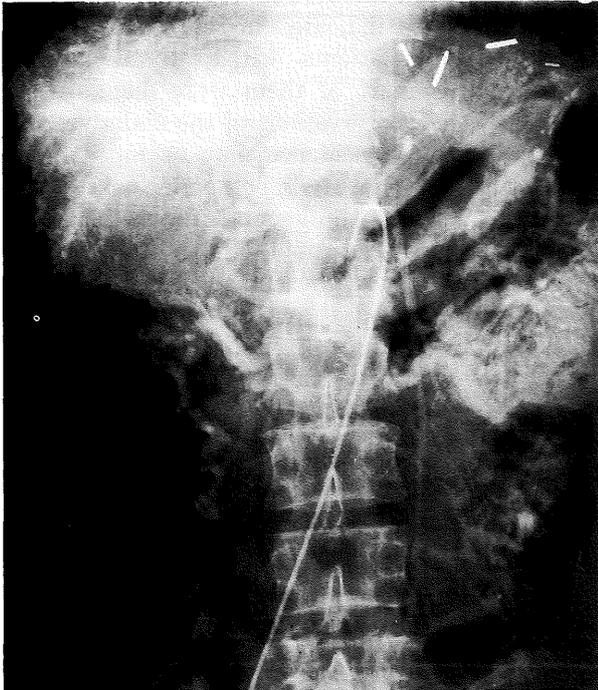


Fig. 1B. Proximal portion of superior mesenteric vein, completely occluded, with cavernous transformation.



Fig. 2A. Echogenic mass within portal vein (arrow), by ultrasonography.

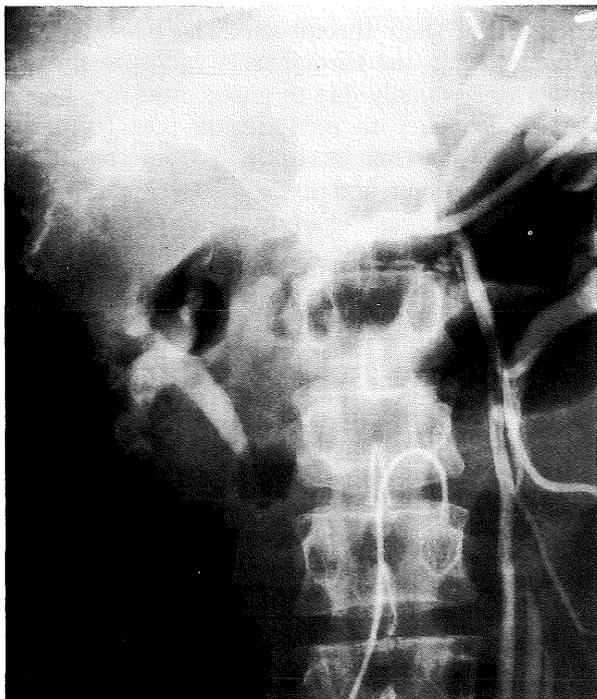


Fig. 1C. Patent splenic vein stump during venous phase of inferior mesenteric arteriography.

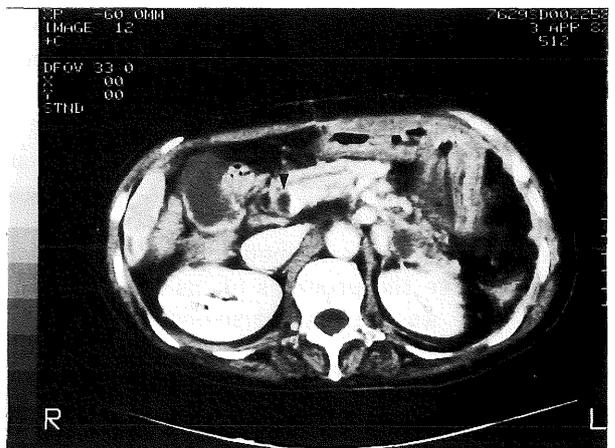


Fig. 2B. CT showing filling defect in portal vein (arrow head).

SV was intact (Fig. 1C).

Thrombectomy using a Fogarty catheter and partial jejunostomy were performed, because effective fibrinolytic therapy was not anticipated. At surgery, the SMV was hard, had a tubular appearance with no flow, and the pulsations of the SMA were diminished. An organized yellow-brown thrombus was partially removed.

Case 2. Splenectomy was performed for this 60-year-old woman whose diagnosis was idiopathic portal hypertension. Abnormal liver function and portal thrombosis were noted one week and one month post splenectomy, respectively, by US, and CT (Fig. 2A, and B) without CTF. She died three months after surgery. At autopsy, the portal vein

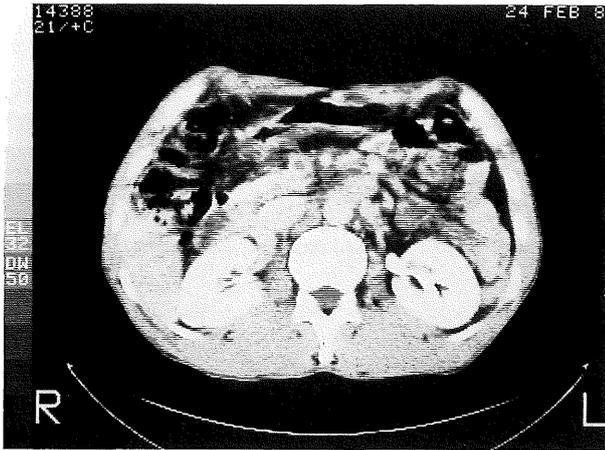


Fig. 3A. Complete occlusion of superior mesenteric vein

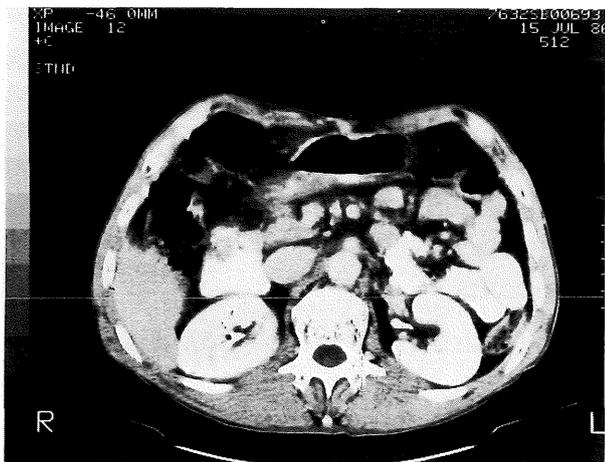


Fig. 3B. Same patient as in Fig. 3A. Superior mesenteric vein is recanalized 5 months later without anticoagulation and antiplatelet therapy.

was completely occluded from its origin to its intrahepatic portion, but no thrombosis was detected in the SMV or SV.

Case 10. CT was performed for this 53-year-old man to observe the progress of his liver cirrhosis (Fig. 3A). He underwent splenectomy 4 years and 2 months ago.

Complete occlusion of the short segment of the SMV was observed. Collateral circulation of the SMV was not observed because no angiography was performed. He was asymptomatic, but his severe anemia persisted. Five months later, his occluded SMV had recanalized following sclerosing therapy for esophageal varices, and blood transfusions (Fig. 3B).

DISCUSSION

The incidence of portal venous thrombosis post-splenectomy reportedly ranges from 4.4 to 17.9%, based on autopsy findings and clinical experiences^{4,8,14}; however, it is reportedly about

70% as imaged by systemic angiography^{18,25}.

Our experience indicated an incidence of 38%. Since half the patients with thrombosis were asymptomatic, many of them were diagnosed incidentally during observations of the underlying disease process. The incidence may be much higher than clinically appreciated. The underlying disease processes causing portal venous system thrombosis post splenectomy were mainly portal hypertension and blood dyscrasias. There have been few reports concerning portal vein thrombosis following splenectomy for trauma, pancreatectomy or gastrectomy^{3,14,22}; such cases were not included in the present study. The incidence of thrombosis after DSRS is reportedly 4 to 28%^{9,19}; and by our experience, 7%. The more porto-systemic collaterals ligated, the higher the rate of thrombosis⁹.

It is considered that portal venous thrombosis occurred within 1 month after surgery in most patients because of the acceleration of coagulation, regardless of the primary disease¹⁶.

In our series, portal thrombosis discovered in the relatively early postsurgical period, accompanied clinical symptoms, and cases with complete occlusion of PV without CTF had poor prognoses. In general, the prognosis of patients with portal thrombosis depends on the speed and extent of the formation of their thrombosis. The mortality rate is higher when the thrombus is widespread¹³. Fatalities were mainly due to gastrointestinal hemorrhages secondary to increases in portal pressure and intestinal ischemia resulting from venous outflow obstructions and liver failures^{1,8}. However, cases with minimal changes in their portal vein hemodynamics had favorable prognosis; their portal thrombosis occurred later postoperatively and were mainly nonocclusive; their thromboses subsided spontaneously; and they developed collateral circulations^{9,13}. The significance of CTF or collateral circulations may lie in decreasing portal hypertension and maintaining portal flow.

Conventionally, the mechanism of portal vein thrombosis has been based on three major factors; 1) injury to the vascular endothelium, 2) changes in hemodynamic flow, and 3) changes in the blood components¹¹. Increased platelet counts were previously considered the main factor in postoperative thromboembolism; but this is now regarded as only one of a number of factors in the development of thrombosis^{5,8}. Correlations between platelet counts and thromboembolism have been refuted by some investigators^{3,18}. More recently, attention has been directed to changes in hemodynamic flow and to traumatized endothelium as causative factors^{8,18}. Thrombi reportedly originate in the splenic vein stump, and the operative method of dealing with this problem may influence the risk of developing a thrombus^{4,9}. During surgery, the SV should be ligated as close as possible to its junction with the inferior mesenteric vein⁴. We ob-

served only one case of thrombosis of the splenic vein, and surgical trauma to the portal venous system which also tended to cause portal thrombosis. Portal thrombosis is reportedly more frequent after catheter intervention, as in intraoperative portography and the measurement of portal pressures during surgery²¹). However, if injury to the endothelium occurs by infection or surgery, spontaneous resolution of thrombosis long after surgery, as in case 10, is difficult to explain. In case 10, there may have been correlations between congestion of the blood, changes in the blood components, and thrombosis associated with persistent severe anemia long after surgery¹⁶). The brain abscess occurred in case 1 is attributable to vulnerability to infection; specifically, overwhelming postsplenectomy infection¹²). Postsplenectomy infection is another potentially lethal complication in splenectomized patients and it requires immediate, precise treatment.

The successful treatment of portal thrombosis includes early surgical exploration, resection of the involved bowel and thrombectomy^{2,6,23}).

A mortality rate of nearly 100% has been reported in unoperated patients^{6,17,23}), with CTF, the mortality rate would have been greatly reduced. The presence of CTF and collateral circulations must be diagnosed, because their presence indicates a good prognosis. However, portal thrombosis may be prevented using anticoagulation and antiplatelet therapy prophylactically before and after surgery.

After splenectomy or DSRS, there is a high rate of portal thrombosis, and ascites or abdominal pain are signals to suspect portal vein thrombosis^{1,4,7,19,24}). When suspected, must be evaluated by US and CT for early detection and treatment, and long-term follow-ups are indicated with attention to anemia because of its occurrence long after surgery.

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