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The Incidence and Natural History of Thrombus in the Portal Vein Following Distal Splenorenal Shunt

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The incidence of thrombus formation in the portal vein following distal splenorenal shunt was 4% occlusive and 14% nonocclusive from 1974 to 1977, and 6% occlusive and 22% nonocclusive in 1980. The increased incidence was probably due to more aggressive ligation of collaterals on the portal vein. Ten patients with this complication were evaluated prospectively with clinical and biochemical parameters, angiography, and nutrient hepatic perfusion. In this group, one thrombus was occlusive immediately after operation, and nine were nonocclusive: eight of the latter resolved by six months, but one progressed to total thrombosis. There were no demonstrable adverse clinical or biochemical sequelae. Angiography showed continuing portal perfusion in the face of nonocclusive thrombus, but at six months there was increased collateral formation and significant ($p < 0.05$) reduction in portal vein diameter, from 20 ± 4 mm to 14 ± 5 mm. Nutrient hepatic perfusion at six months, 896 ± 257 ml/min, was not significantly different from that seen prior to operation, 848 ± 92 ml/min. It is concluded that the natural history of nonocclusive portal vein thrombus after distal splenorenal shunt is resolution, and management should be expectant.

THROMBOSIS OF THE PORTAL VEIN following variceal decompression by the distal splenorenal shunt (DSRS) is a sufficiently common occurrence to warrant full evaluation. How common and how serious is this complication? Rotstein et al.[1] reported a 10% incidence of occlusive portal vein thrombosis in 48 shunts and concluded that it contributed to the death of two of their patients. Maillard et al.[2] and Uribe et al.[3] found thrombosis a benign complication in one of 19 and three of 98 patients, respectively. Two distinct entities can be recognized angiographically: nonocclusive clot in the portal vein and totally occlusive thrombosis. What are the precipitating factors in thrombus formation? Anecdotally, it has been noted that the incidence of this complication has increased since portal-azygos disconnection has been performed more aggressively, by identifying and ligating the coronary and umbilical veins as they leave the portal vein. The method of closure of the distal splenic vein stump may influence the incidence of thrombosis, as this is the most common site of thrombus origin. What is the natural history of nonocclusive thrombus? The possible consequences are progression to occlusive thrombosis, with a detrimental effect on hepatic function, and acceleration in the development of portaprival collaterals. Finally, what therapeutic measures should be undertaken when this complication occurs?

In this study, the incidence of both occlusive and nonocclusive thrombus has been defined, and ten patients with this complication have been studied in more detail, with a view to answering the above questions.

Materials and Methods

All patients having DSRS for variceal bleeding at Emory University Hospital are studied by visceral angiography prior to and one week after surgery.[4] One component of this study is visualization of the superior mesenteric and portal veins following superior mesenteric artery injection of contrast; during this phase of

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TABLE 1. *Clinical and Biochemical Parameters of Study Group Prior to and After Operation*

	Age	Sex	Etiology	Bleeding Episodes	Ascites			Encephalopathy			Bilirubin			Prothrombin Time			Albumin		
					1*	2*	3*	1	2	3	1	2	3	1	2	3	1	2	3
												<1.0			<2s.			<3.5	
1	40	M	Alcohol	4	−	−	−	−	−	−	1.9	2.8	2.0	2.5	2.8	2.5	4.3	4.3	3.6
2	51	M	Alcohol	3	+	+	+	−	−	−	0.9	2.0	1.9	1.7	1.7	1.6	2.6	3.4	3.4
3	47	F	Alcohol	4	+	+	−	−	−	−	0.84	2.3	2.4	2.3	1.7	1.0	3.9	3.4	3.0
4	50	M	Alcohol	2	+	+++	+	−	−	−	0.89	4.0	1.2	2.3	2.3	1.6	3.9	3.5	3.4
5	65	F	Alcohol	2	−	+	+	−	−	−	1.2	2.2	1.5	2.1	2.3	3.1	3.5	3.0	3.5
6	62	F	Hepatitis	2	−	+	−	−	−	−	0.37	1.7	0.6	1.2	2.1	1.6	3.9	3.9	3.6
7	44	F	Hepatitis	1	−	+	−	−	−	−	0.39	1.9	2.4	1.2	1.0	2.1	3.3	3.7	3.6
8	51	F	Alcohol	3	−	+	−	−	−	−	0.84	3.7	0.9	1.0	1.5	2.0	4.0	4.6	3.6
9	60	M	Alcohol	2	+	+	+	−	−	−	0.93	3.2	4.7	3.4	2.5	2.9	3.3	4.5	3.3
10	25	F	Hepatitis	3	−	−		−	−		1.64	4.9	2.3	2.7	3.9	2.1	3.3	3.0	3.4

* 1 = preoperative; 2 = 7–10 days after operation; 3 = 3–6 months after operation.

the study, the presence or absence of thrombus in the portal vein is determined. For the purpose of this review, postoperative portal vein thrombus has been defined as occurring if the preoperative angiograms show a normal superior mesenteric and portal vein and the 7–10-day postoperative films show thrombus in this venous segment.

Review Group

Two groups of patients undergoing DSRS were evaluated: each consisted of 50 consecutive patients reviewed to determine incidence. Group 1 comprised all patients having this operation from January 1975 to September 1977, during which time the DSRS and portal-azygos disconnection was performed as originally described.[5] The main site of collateral interruption was suprapancreatic at the gastroesophageal junction. Group 2 consisted of 50 consecutive patients operated on from May to December 1980; during this period, the portal-azygos disconnection was more extensive[6] and included dissection of the portal vein and flush ligation of the coronary vein as it joined the portal vein.

Study Group

A subset of ten patients from group 2 has been studied more extensively and comprises all patients with this complication from August to December 1980.

Preoperative Evaluation

Clinical and biochemical data are summarized in Table 1. Needle liver biopsy, for diagnosis and assessment of the activity of cirrhosis, was done on all patients: all showed minimal or no activity of their disease at the time of surgery. Angiography included superior mesenteric and splenic artery, plus hepatic vein and left renal vein studies, as previously described.[4]

Five patients had further hemodynamic evaluation by measurement of the clearance of low plasma concentrations of galactose. This measures effective—or nutrient—liver plasma flow.[7] Five per cent galactose was given intravenously as a 500-mg loading bolus, followed by continuous infusion at 40 mg/minute (i) for 100 minutes. The steady state plasma concentration (C_{ss}) was measured from 60 to 100 minutes and clearance calculated from i/C_{ss}. For comparison, five other patients who had DSRS and the same hemodynamic evaluation, but who did not have portal vein thrombus after operation, have been included.

Operative Procedure

A DSRS with portal-azygos disconnection was performed using the technique currently advocated.[6] The points of emphasis of the surgical procedure in respect to this study are 1) that the splenic vein stump was ligated with a single black silk ligature flush with the superior mesenteric vein, and 2) that the coronary and umbilical veins, which act as significant portal deprivation collaterals, were ligated as close to the portal vein as feasible.

Postoperative Evaluation

The study group was evaluated prior to discharge (7–10 days after operation) and again 3–6 months after surgery. Clinical assessment and the biochemical data base were as in the preoperative evaluation. Superior mestenteric and splenic artery studies—carried through the venous phase—were performed at both times. The superior mesenteric artery study documents portal venous perfusion and portal systemic collaterals; the splenic study demonstrates shunt patency. Thrombus seen in the portal vein on the venous phase of the SMA study at 7–10 days identified the study population. Four

indices were measured on all three angiograms for each patient to quantitate the following features: portal venous perfusion,[4] portal vein diameter 2 cm above splenic vein insertion, thrombus size, and the site and size of portal systemic collaterals. The galactose clearance was repeated in the five patients with preoperative studies at both time intervals.

Results

Incidence

In the period from 1975 to 1977, there were nine episodes of thrombus in the portal vein in the 50 patients reviewed, giving an incidence of 18%. Two of these were occlusive, giving a portal vein thrombosis incidence of 4%. In the more recent period, May to December 1980, there were 14 episodes of thrombus, an incidence of 28%; three were occlusive, giving a thrombosis incidence of 6%.

Mortality/Morbidity

There were no operative deaths (30 days) in patient with this complication. Early morbidity, as judged by length of hospital stay, clinical and biochemical indices, was no greater in patients with thrombus than in those without this complication. Late morbidity is harder to assess but at a clinical level does not appear to be increased.

Angiography

In all patients in this study group, the shunt was patent at both postoperative studies. There was one patient with total portal vein thrombosis and nine episodes of nonocclusive thrombus. Table 2 semiquantitates the degree of thrombus formation in this study group.

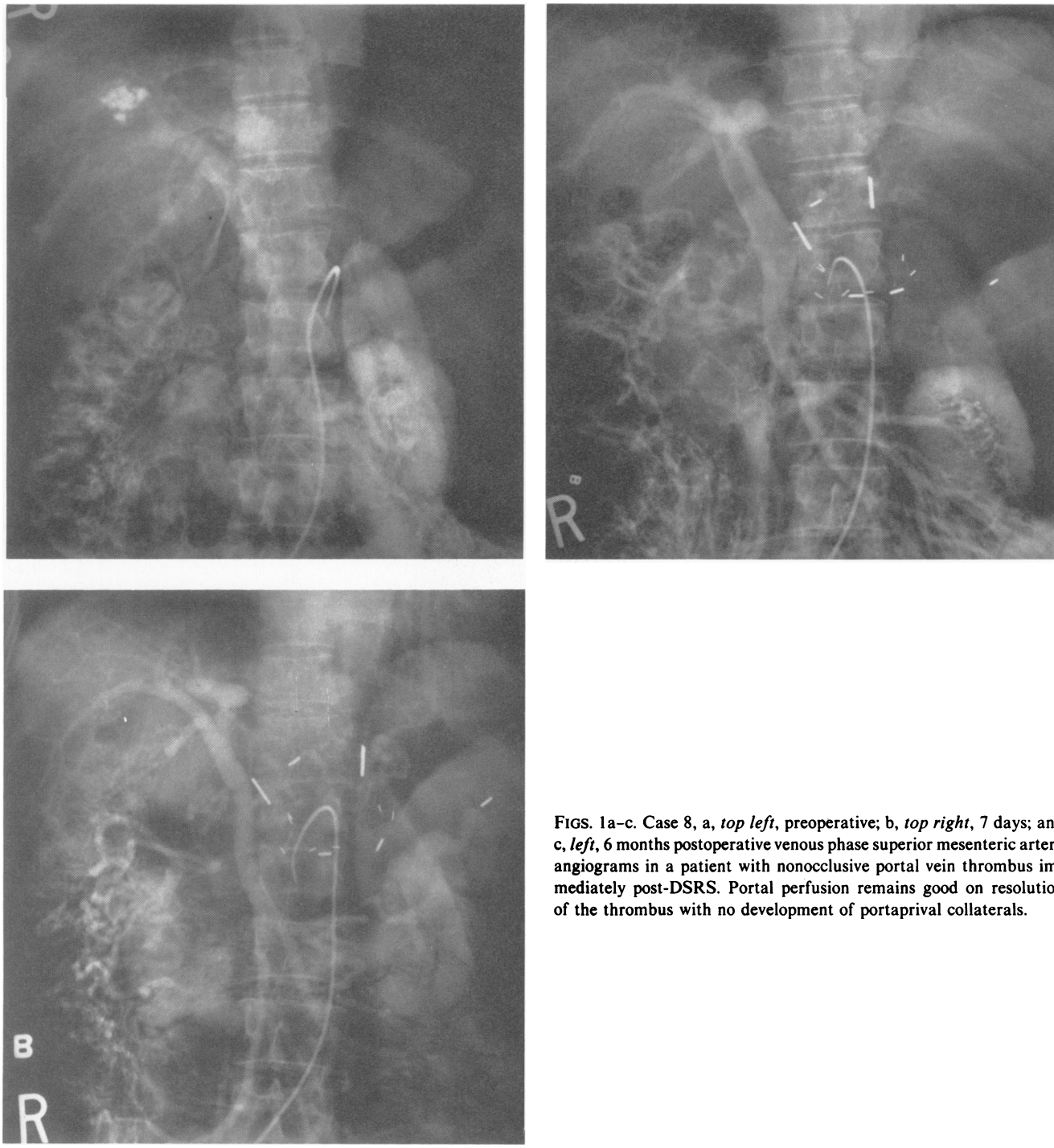
The angiographic grade of portal perfusion does not appear to change significantly, provided the thrombus is nonocclusive. Prior to operation, five patients had grade I portal venous flow and five had grade II; at 3–6 months, five had grade I, two grade II, and one grade III, and two had progressed to total thrombosis. In contrast, the diameter of the portal vein was significantly ($p < 0.05$) less at 3–6 months (14 ± 5 mm) than it was prior to operation (20 ± 4 mm). The thrombus was large, with a mean length of 83 mm. Complete or partial resolution of all but one of the nonocclusive thrombi had occurred by 3–6 months. Figures 1 and 2 illustrate 1) preoperative, 2) 7–10 days postoperative, and 3) three months postoperative in two of these patients. The first patient developed no significant collaterals, but the second showed significant collateral formation to her shunt via colonic vessels by six months. The one patient

TABLE 2. *Angiographic Parameters in 10 Patients with Portal Vein Thrombus Following DSRS*

	Perfusion Grade			Portal Vein Diameter (mm)			Thrombus Size (mm)		Collateral Veins		
	Preoperative	1 week	3–6 mos.	Preoperative	1 week	3–6 mos.	1 week	3–6 mos.	Preoperative	1 week	3–6 mos.
1	I	I	I	21	20	12	30 × 15 (n.o.)†	Nil	Nil	Coronary, ++	GI +
2	II	III	IV	18	18	—	80 × 10 (n.o.)	Occ*	Umbilical ++	GI +	GI +++
3	II	I	III	25	24	22	80 × 17 (n.o.)	35 × 8 (n.o.)	Nil	Coronary +	GI ++++
4	II	III	II	22	20	10	120 × 17 (n.o.)	Nil	Nil	Coronary +	Coronary +++
5	I	IV	IV	15	—	—	Occ	Occ	Coronary +	GI +	GI +++
6	II	I	I	19	19	16	30 × 15 (n.o.)	Nil	Coronary +	Nil	Pancreatic +++
7	I	II	I	17	26	8	120 × 10 (n.o.)	Nil	Coronary +	GI +	GI ++
8	II	II	I	14	20	13	70 × 13 (n.o.)	Nil	Nil	Nil	Nil
9	I	II	II	20	20	9	120 × 10 (n.o.)	Nil	Nil	Coronary +	Coronary +++
10	I	I	I	28	29	21	100 × 21 (n.o.)	Nil	Nil	Nil	Pancreatic +++
				Mean ± SD							
				20 ± 4	21.5 ± 4	14 ± 5					

* Occ = occlusive thrombus.

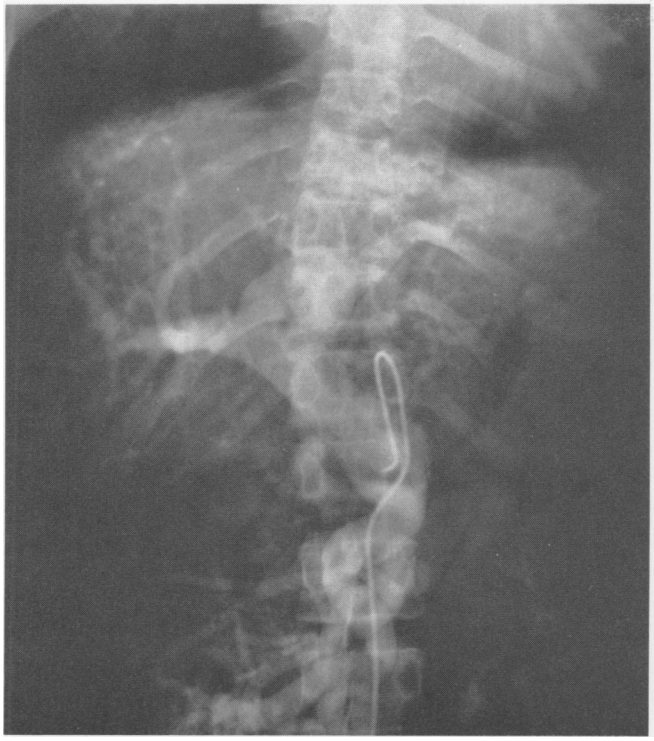
† n.o. = nonocclusive thrombus.

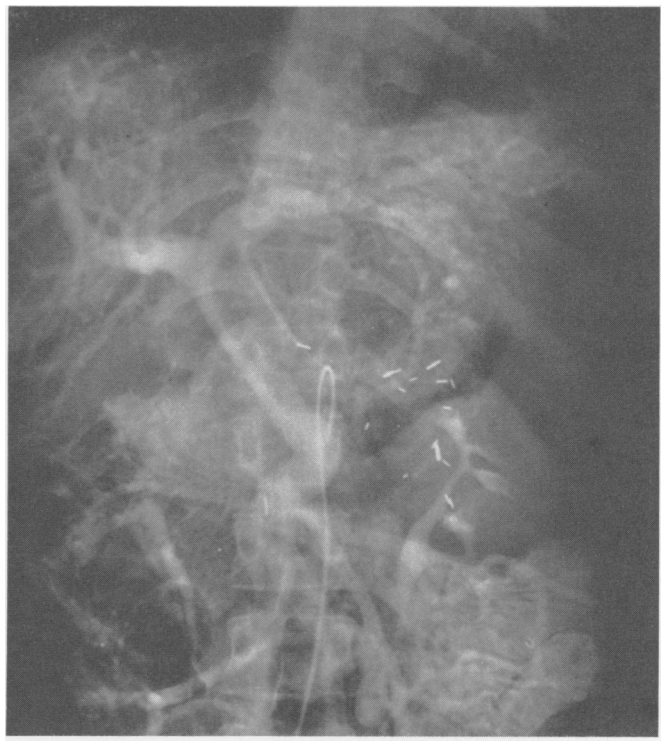


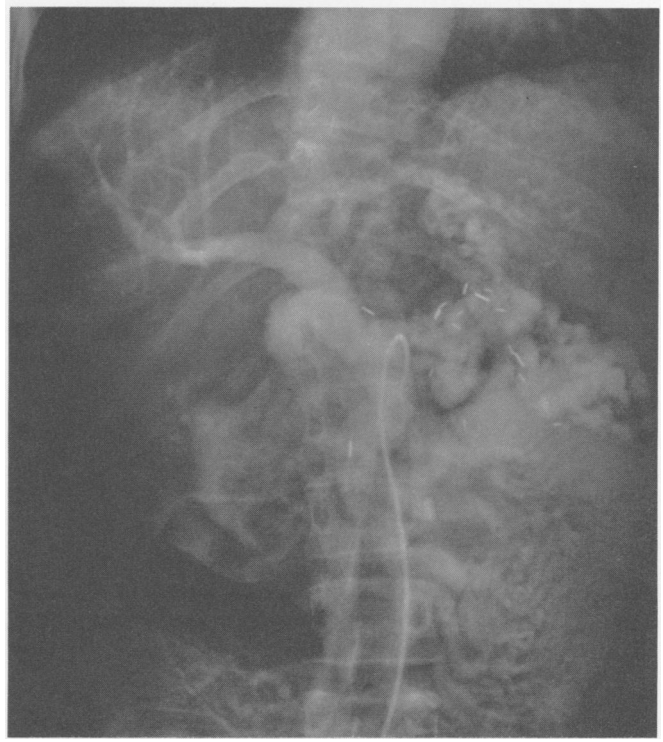
FIGS. 1a–c. Case 8, a, *top left*, preoperative; b, *top right*, 7 days; and c, *left*, 6 months postoperative venous phase superior mesenteric artery angiograms in a patient with nonocclusive portal vein thrombus immediately post-DSRS. Portal perfusion remains good on resolution of the thrombus with no development of portaprival collaterals.

who progressed from partial occlusion to total thrombosis at four months was noted to have a rise in SGOT and alkaline phosphatase at three months. This may have been associated with the progression to thrombosis. The other patient in this group with total portal vein thrombosis has not recanalized, but her clinical course has been satisfactory. However, the two patients with total thrombosis in the 1975–1977 review group both showed recanalization and restoration of portal perfusion at two years and four years, respectively.

The development of portal systemic collaterals is harder to quantitate. Gastrointestinal collaterals, usually via the transverse colon, and pancreatic collaterals, both to the decompressed spleen appeared by 3–6 months in seven of the study group of patients. The three patients with extensive coronary collaterization







FIGS. 2a–c. Case 10, a, *top left*, preoperative; b, *top right*, 10 days; and c, *right*, 6 months postoperative angiograms, as for Figure 1. This patient maintains good portal perfusion, but also shows reduction in portal vein size and the development of large portaprival collaterals.

represent a failure of complete portal-azygos disconnection.

Hemodynamics

The galactose clearance data for the five patients and the five controls are given in Table 3. These show that there is no significant difference in flow in the patients with thrombus compared with the control group with no thrombus at any of the time intervals. Of note are the three patients in the control group and two in the thrombus group who had a significant increase in clearance at one week; this is probably secondary to acute

TABLE 3. *Galactose Clearance Data Following DSRS in five "Uncomplicated" Patients and five Patients with Thrombus in Portal Vein*

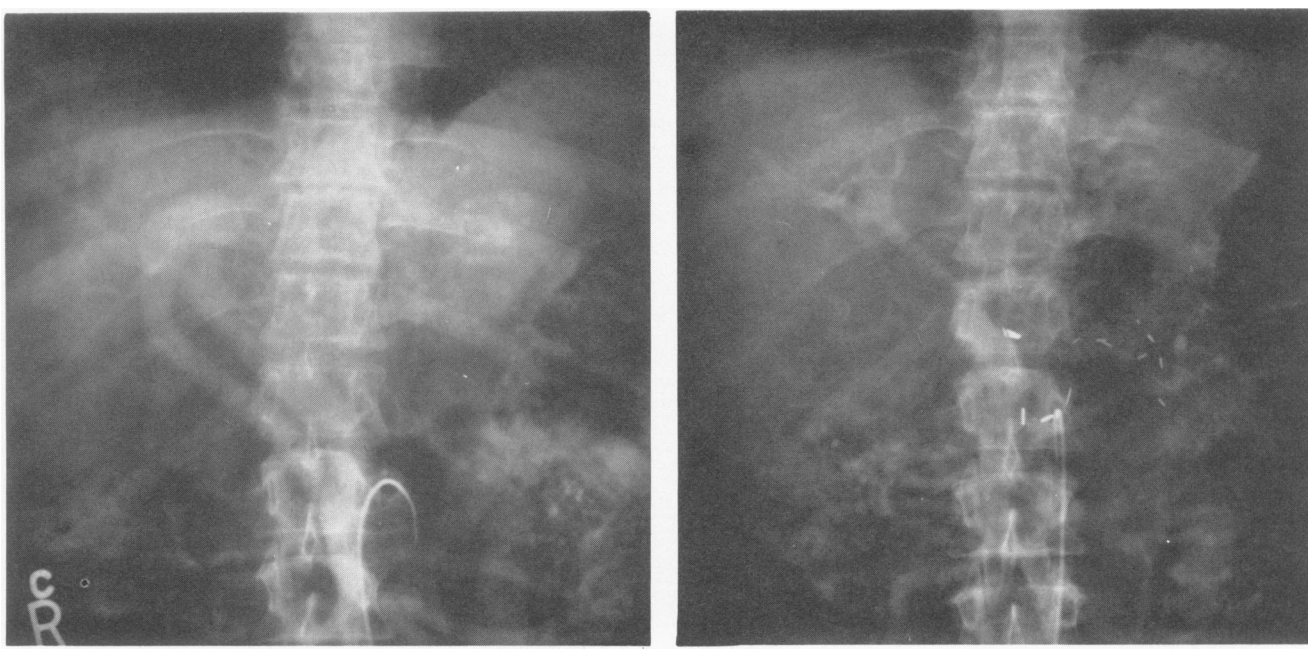
	Clearance (ml/min)		
No Thrombus	Preoperative	7–10 days	3–6 mos.
1	896	2100	857
2	1016	2075	727
3	694	1262	546
4	956	701	830
5	580	404	657
	828 ± 184	1308 ± 775	723 ± 128
Thrombus			
1	880	2042	1008
2	774	1870	1293
3	977	868	804
4	913	656	707
5	776	657	667
	848 ± 92	1219 ± 681	896 ± 257

perioperative hemodynamic changes and has returned to the preoperative level by three months. In neither group is the mean clearance at 3–6 months significantly different from preoperative levels.

Discussion

In the reviewed patients, the incidence of portal vein thrombus following DSRS rose from 18 to 28% concurrently with changes in operative technique. In both groups, however, the majority (7 of 9, and 11 of 14) showed nonocclusive thrombus, the natural history of this being resolution. More aggressive attempts to separate the high-pressure portal venous from the low-pressure splenorenal system by ligation of both major and minor collaterals may have contributed to this rise. Full mobilization of the portal vein behind the pancreas, with identification and ligation of the coronary vein at its origin, may predispose to intraluminal clot. Dissection of the umbilical vein, which may be massive in some of these patients, to its origin on the left portal vein has undoubtedly contributed to thrombus formation in some cases (Fig. 3). The site of origin of thrombus in this series was most commonly the splenic vein stump, and the operative method of dealing with this may influence the risk of thrombus. For the first 50 patients reviewed, the splenic vein stump was oversewn with a vascular suture flush with the superior mesenteric vein, while in the latter 50 it was managed by a single flush ligature. The effect of these two methods cannot be assessed from this review as other operative changes, as indicated above, have accompanied this change. Flush ligation, avoidance of a *cul-de-sac*, and preservation of intimal continuity should minimize this risk, but the ideal cannot always be achieved. Aggressive interruption of collaterals from the portal vein, particularly the coronary and umbilical veins, creates the same risks as potential sites for thrombus, which may then propagate into the vein.

Are there any preoperative predictive indices for thrombus formation? Spontaneous thrombosis of the



FIGS. 3a and b. Case 2, a, *left*, preoperative and b, *right*, 7 days postoperative angiograms as in Figure 1; a, large umbilical vein, as a portaprival collateral, running parallel to the portal vein; b, thrombus in the portal vein originating at the splenic vein stump and in the left portal vein originating from the ligated umbilical vein.

portal vein can occur in cirrhosis; this has been associated with sluggish portal venous flow. However, this factor cannot be implicated etiologically in the present study group, who all had grade I or II portal venous perfusion on preoperative angiography. The clinical, biochemical, and clearance measurements in these patients did not differ in any way from those of patients without thrombus. No preoperative "at risk" factors have been identified in the present study.

The significant reduction in portal vein diameter at 3–6 months is equivalent, by Poiseuille's law, to a reduction in flow in that vessel. However, liver blood flow has two component parts, hepatic arterial and portal venous, and the quantitative measure by galactose clearance incorporates both components. While our angiographic data suggest some reduction of portal venous flow notably reduction in portal vein diameter, effective—or nutrient—flow is maintained. This is probably due to increased hepatic arterial flow and increased cardiac output. Some key as to the mechanism by which this occurs may lie with those patients who show significant increase in their galactose clearance over the operation period.

How should this complication be managed? This study defines the natural history of thrombus in the portal vein induced at surgery and indicates that for the majority, with nonocclusive thrombus, management should be expectant. All but one of the nonocclusive thrombi had totally or partially resolved by six months. In addition, there was no demonstrable early or late morbidity in the patients studied. But what of the patients with total thrombosis? These are the patients reported by Rotstein et al.;[1] two of their five patients with this complication died. This has not been the experience at this institution; there was no mortality in the present series attributable to this complication, which supports the findings of Uribe[3] and Maillard.[2]

Table 2 indicates that rapid development of significant portoprival collaterals follows this complication, with all but one of the ten study patients developing significant collaterals by six months. It is postulated that increased mesenteric pressure secondary to the thrombus may encourage this diversion of flow by collaterals. While this phenomenon is being increasingly described following the DSRS,[2,8] the normal time course for the development of portoprival collaterals is over years, and not the rapid change seen in these patients. This is the major long-term detrimental effect of this complication.

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