Long-term Outcomes of Splanchnic Venous Thrombosis in Acute Pancreatitis

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Background: Splanchnic venous thrombosis (SVT) involving veins in the vicinity of the pancreas is a significant complication of acute pancreatitis (AP). The natural history of SVT, especially the rates of recanalization, is poorly understood.

Aim: This study aimed to evaluate the natural history of SVT in AP, with a focus on recanalization rates and identifying predictors of nonrecanalization.

Materials and Methods: This was an observational study in which patients with SVT in the setting of AP were included. Patients were followed for at least 6 months. Recanalization was assessed using Doppler ultrasound or CT imaging, and outcomes were classified as complete recanalization, partial recanalization, or nonrecanalization. Statistical analysis was done to identify predictors of nonrecanalization.

Results: Among 814 patients with AP, 92 (11.3%) developed SVT. Of these, 70 met the inclusion criteria. The mean age was 38.1 years, with 92.8% male predominance. Alcohol was the most common etiology (62.8%). The retropancreatic splenic vein was the most commonly affected vessel. At follow-up, complete recanalization was observed in 54.3% of cases, partial recanalization in 2.9%, while 42.8% showed no evidence of recanalization. Therapeutic anticoagulation was administered to 20% of patients without significantly influencing recanalization rates. A BISAP score ≥ 2 was a significant predictor of nonrecanalization ($P = 0.007$).

Conclusion: Most patients with SVT following AP demonstrate spontaneous recanalization. A key predictor for nonrecanalization is the severity of pancreatitis.

Key Words: acute pancreatitis, vascular thrombosis, pancreas, splenic vein, portal vein, superior mesenteric vein

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Splanchnic venous thrombosis (SVT) is a common complication of acute pancreatitis (AP) affecting the splenic vein (SV), portal vein (PV), and superior mesenteric vein (SMV), either isolated or involving several venous segments.[1] Global estimates of the incidence of SVT in AP vary widely (1.8% and 30.8%).[2] SVT is often an incidental finding when imaging is done to assess symptoms or complications of acute pancreatitis. The pathophysiology of SVT remains poorly understood. It is hypothesized that endothelial dysfunction, inflammation-mediated activation of coagulation, and vascular stasis secondary to local inflammation and edema may lead to thrombus formation.[3] SVT is usually noted in patients with moderately severe or severe AP with peripancreatic necrosis or fluid collections.[4] The presence of ongoing vascular occlusion within the splanchnic circulation can lead to many problems, including but not limited to portal hypertension, small bowel ischemia, and hepatic failure.[5,6] Despite its clinical importance, there is limited literature on the natural history of SVT in AP patients and the role of anticoagulation in its management. This gap affects clinicians' ability to manage SVT in patients with AP. This study aimed to assess the natural history of SVT in patients with AP, with a focus on recanalization rates. By prospectively following a cohort of AP patients with SVT, we sought to identify factors influencing recanalization.

MATERIALS AND METHODS

This prospective single-center observational study was conducted in the Department of Gastroenterology at a tertiary referral center in India. It was approved by the Institutional Review Board and the Ethics Committee (IRB Approval No. 15787). The study was performed in accordance with the Declaration of Helsinki, and written informed consent was obtained from all patients or their next of kin before inclusion in the study.

Study Population

All patients diagnosed with AP and admitted to the hospital between October 2018 and September 2023 were eligible for inclusion in the study. The inclusion criteria for the study were as follows: (1) First episode of acute pancreatitis (the diagnosis of AP was established using the revised Atlanta criteria).[7] (2) Imaging showing presence of SVT. Patients younger than 18 years of age, chronic pancreatitis, recurrent acute pancreatitis, cirrhosis, malignancy, patients who were on anticoagulants for an alternate cause and those who had a follow-up period of < 6 months were excluded from the study.

Outcome Measures

The primary objective of the study was to assess the natural history of SVT in patients with AP, with a focus on recanalization rates. The secondary objective was to determine the predictors of recanalization of SVT in AP.

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Data Collection

We collected comprehensive baseline data for each patient. This included demographic variables such as age and sex, as well as the etiology and severity of AP. Laboratory parameters were recorded, comprising hematocrit, white blood cell count, platelet count, C-reactive protein (CRP), glycated hemoglobin (HbA1c), calcium, triglycerides, and prothrombin time. We also noted clinical outcomes, including the median duration of hospital and ICU stay. Imaging findings were documented, detailing involved vessels, colocalized collections, infected collections, and any therapeutic drainage of collections. Finally, we recorded anticoagulation details, distinguishing between therapeutic and prophylactic anticoagulation administered to patients. Severity of AP was assessed using the Revised Atlanta Classification[7] and the Bedside Index for Severity in Acute Pancreatitis (BISAP) score.[8]

Follow-up and Outcome Assessment

Patients were followed up for a minimum period of 6 months. During follow-up, a Doppler ultrasound of the abdomen was performed to assess for recanalization of thrombosed vessels. The Doppler ultrasound was done by 2 senior radiologists (B.S. and P.D.) who were blinded to the anticoagulation data. Doppler ultrasound was not done in patients who had a contrast-enhanced computed tomography (CECT) abdomen as part of ongoing clinical care. The findings of the CECT abdomen were evaluated by the same 2 radiologists mentioned above, and the status of the thrombosis was determined. Recanalization was classified as complete recanalization, partial recanalization, or nonrecanalization.

Definitions

Splanchnic Venous Thrombosis

It was defined by the presence of an actual thrombus in the vein, or the vein appeared compressed or was not visualized with the presence of collaterals.[9]

Complete Recanalization

It is defined as full restoration of blood flow through a previously occluded vessel, with the complete dissolution or removal of the thrombus or obstruction by Doppler abdomen/CT abdomen.[10]

Partial Recanalization

Partial recanalization often refers to more than 50% reduction of the previous thrombus without thrombus extension.[11]

Therapeutic Anticoagulation

Patients receiving therapeutic anticoagulation were initially treated with either subcutaneous LMWH (1 mg/kg twice daily) or intravenous unfractionated heparin (in patients with acute kidney injury, 5000 U q6h) followed by oral warfarin with target INR maintenance between 2 and 3.

Prophylactic Anticoagulation

Patients were anticoagulated with subcutaneous LMWH (1 mg/kg once daily) or intravenous unfractionated heparin (in patients with acute kidney injury, 5000 U q12h) till discharge of the patient from the hospital.

Statistical Analysis

For continuous variables, we reported the mean and SD for normally distributed data, while the median and interquartile range (IQR) were used for non-normally distributed data and ordinal data. For categorical variables, we presented frequencies and percentages. Independent *t* test and χ^2 test were used for comparison of continuous variables and categorical variables, respectively. Univariate analysis was performed to identify risk factors for nonrecanalization of SVT. All tests were 2-sided at an alpha = 0.05 level of significance. Data was analyzed using SPSS software version 21.

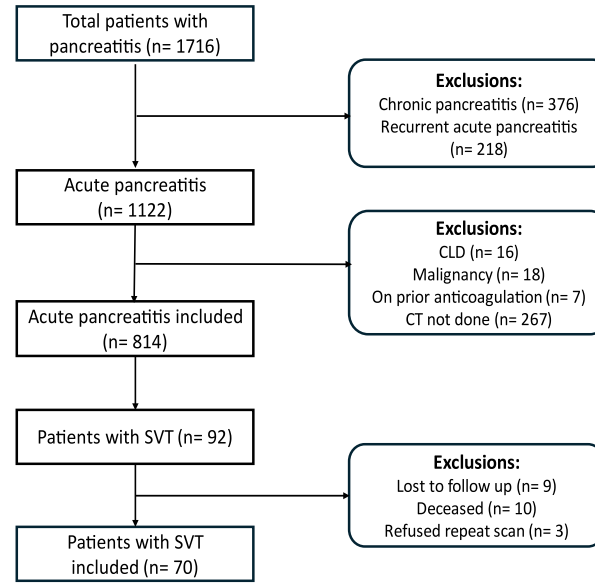

FIGURE 1. Flow chart of patients included in the study. CLD indicates chronic liver disease; SVT, splanchnic venous thrombosis.

RESULTS

Baseline Characteristics

Of 814 patients with AP, 92 patients (11.3%) developed SVT (Fig. 1). Among them, 9 patients were lost to follow-up, 10 died, and 3 refused repeat scans. Seventy patients with SVT who met the inclusion criteria were included in the study (Table 1). Sixty-seven (95.7%) patients had direct visualization of thrombus within the vein, while 3 (4.3%) patients were diagnosed based on vein attenuation/compression with the presence of collaterals. The mean age of the cohort was 38.1 years (SD = 12), with a strong male predominance of 65 (92.8%). The most common etiology of AP was alcohol consumption, accounting for 44 (62.8%) cases. According to the Revised Atlanta Classification, most patients 41 (58.6%) had moderately severe AP. Necrotizing pancreatitis was observed in 64 (91.4%) of patients. The retropancreatic splenic vein was the most involved vessel, affected in 47 (67.1%) of cases (Table 2). Three patients (4.3%) with SVT demonstrated concurrent deep vein thrombosis, comprising one case of isolated RPSV, one case with concomitant RPSV + PV involvement, and one case with extensive thrombosis affecting the RPSV+PV +SMV. None had pulmonary embolism. Therapeutic anticoagulation was administered to 14 (20%) of patients, based on clinical judgment of the treating team (Supplemental Tables 1 and 2, Supplemental Digital Content 1, http://links.lww.com/MPA/B395).

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TABLE 1. Baseline Demographic and Clinical and Laboratory Characteristics of AP Patients With SVT Divided into 2 Groups Based on Recanalization

Parameters	Total (n = 70)	Recanalization (n = 40)	Nonrecanalization (n = 30)	*P*
Age, y	38.1 (12)	39 (12.3)	37 (12.3)	0.5
Males	65 (92.8)	37 (92.5)	28 (93.3)	1.00
Etiology				
Alcohol	44 (62.8)	26 (65)	18 (60)	0.55
Idiopathic	13 (18.7)	8 (20)	5 (16.7)	
Gallstone	12 (17.1)	5 (12.5)	7 (23.3)	
Hypertriglyceridemia	1 (1.4)	1 (2.5)	0	
Severity				
Mild	3 (4.3)	3 (7.5)	0	0.30
Moderate	41 (58.6)	22 (55)	19 (63.3)	
Severe	26 (37.1)	15 (37.5)	11 (36.7)	
Organ failure	36 (51.4)	21 (52.5)	15 (50)	1.00
PCV at admission	42.6 (10.7)	42 (10.1)	43 (11.7)	0.70
Platelet at admission (cells/mm^3)	26,200 (19,900–35,400)	28,300 (21,200–37,500)	25,000 (19,200–33,900)	0.99
Hba1c	6.2 (1.3)	6.3 (1.5)	6.2 (1)	0.80
CRP at admission (mg/dL)	188 (135–311)	199 (135–227)	170 (121–275)	0.34
Prothrombin time at admission (s)	16 (3.2)	17.2 (4.7)	15.7 (2.6)	0.91
Duration of hospital stay in days	9 (6–12)	8.5 (5.2–12)	9 (6–13)	0.70
ICU admission	7 (10)	4 (10)	3 (10)	1.00
Duration of ICU stay in days	8 (7–12)	11 (8–55)	7 (5–8)	0.35
Duration of follow-up				
6–12 mo	35 (50)	21 (52.5)	14 (46.7)	0.81
>12 mo	35 (50)	19 (47.5)	16 (53.3)	
Therapeutic drainage	18 (25.7)	9 (22.5)	9 (30)	0.60

Data are expressed as mean (SD), median (IQR), or number (%), as appropriate.
CRP indicates C-reactive protein; PCV, packed cell volume; WBC, white blood cells.

TABLE 2. Imaging Findings: Parameters Were Expressed Number (Percentage)

Parameters	n = 70	Recanalization (n = 40)	Nonrecanalization (n = 30)	*P*
Pancreatic parenchymal necrosis	64 (91.4)	37 (92.5)	27 (90)	1.00
Initial imaging				
CT abdomen	69 (98.6)	39 (97.5)	30 (100)	1.00
USG abdomen	1 (1.4)	1 (2.5)	0	
Vessels involved				
RPSV	47 (67.1)	22 (55)	25 (83.3)	0.16
PV	6 (8.6)	5 (12.5)	1 (3.3)	
SMV	1 (1.4)	1 (2.5)	0	
RPSV PV	6 (8.6)	5 (12.5)	1 (3.3)	
RPSV SMV	3 (4.3)	3 (7.5)	0	
RPSV PV SMV	6 (8.6)	3 (7.5)	3 (10)	
SMV PV	1 (1.4)	1 (2.5)	0	
Colocalized collections				
AFC	5 (7.1)	3 (7.5)	2 (6.7)	0.16
ANC	21 (30)	16 (40)	5 (16.7)	
Pseudocyst	3 (4.3)	2 (5)	1 (3.3)	
WON	41 (58.6)	19 (47.5)	22 (73.3)	
Infected collections	21 (30)	12 (30)	9 (30)	1.00
Initially collaterals	21 (30)	10 (25)	11 (36.7)	0.31
Collaterals at follow-up	32 (45.7)	5 (12.5)	27 (90)	<0.001
Deep venous thrombosis	3 (4.3)	1 (2.5)	2 (6.7)	0.57
Portohypertensive ascites	1 (1.4)	0	1 (1.4)	
Follow-up imaging				
CT abdomen	38 (54.3)	19 (90.4)	19 (63.3)	0.23
Color doppler	32 (45.7)	21 (52.5)	11 (36.7)	
Pseudoaneurysm	3 (4.3)	1 (2.5)	2 (6.7)	0.57
Gastric varices	1 (1.4)	1 (2.5)	0	

AFC indicates acute fluid collection; ANC, acute necrotic collection; PV, portal vein; RPSV, retropancreatic splenic vein; SMV, superior mesenteric vein; WON, walled-off necrosis.

TABLE 3. Univariate Analysis of Risk Factors for Nonrecanalization of SVT

Parameters	n = 70	Recanalization (n = 40)	Nonrecanalization (n = 30)	Odds ratio	*P*
Pancreatic necrosis	64 (91.4)	37 (92.5)	27 (90)	1.37 (0.26–7.32)	1.00
BISAP score					
<2	37 (52.9)	27 (67.5)	10 (33.3)	0.24 (0.1–0.66)	0.007
≥2	33 (47.1)	13 (32.5)	20 (66.7)		
Therapeutic anticoagulation	14 (20)	9 (22.5)	5 (16.7)	1.45 (0.43–4.9)	0.80
Any anticoagulation	31 (44.3)	15 (37.5)	16 (53.3)	0.52 (0.2–1.4)	0.23

Follow Up

At follow-up of 6 months or more, no patients had gastrointestinal bleeding or bowel ischemia. Complete recanalization was observed in 38 (54.3%) of patients, while partial recanalization occurred in 2 (2.9%). Among the patients who had complete recanalization, the majority (30 out of 38) had spontaneous recanalization. In univariate analysis, a BISAP score ≥2 at admission was the only significant risk factor associated with nonrecanalization of SVT ($P = 0.007$) (Table 3).

DISCUSSION

The present study explored the natural history of SVT in patients with AP and aimed to identify the factors influencing recanalization. In our cohort, the overall incidence of SVT in acute pancreatitis was 11.3%, consistent with a recent meta-analysis by Xu et al.[12] This rate falls within the broad range reported in the literature, from 1.8% to 30.8% underscoring the significant impact of SVT as a complication of AP.[2,9]

The retropancreatic splenic vein was the most commonly affected vessel (67.1%) either in isolation or in conjunction with the PV and/or the SMV. Multiple studies have confirmed that the RPSV is the most commonly affected vessel.[2,9,13] The splenic vein is in close approximation to the pancreas, making it particularly vulnerable to the effects of pancreatic inflammation. Endothelial dysfunction, hypercoagulable state, and stasis secondary to inflammation all contribute to the pathogenesis of thrombosis.

Twenty-one (30%) patients had collaterals at the time of diagnosis. Formation of collaterals usually occurs a few days after initial thrombosis, up to 3–5 weeks in splanchnic vessels. Initial imaging in acute pancreatitis is often an abdominal ultrasound, which has limited sensitivity for SVT detection. Contrast-enhanced CT scans, which better detect SVT, are typically performed later in the disease course. This probably explains the presence of collaterals at diagnosis in patients with SVT.[5]

An important finding in our study is a high rate of spontaneous recanalization within 6 months. There is a wide variability in the reported rates of recanalization in SVT. Our finding suggests that in a significant proportion of individuals, the thrombus resolves completely within 6 months. This brings into question the routine use of anticoagulation in SVT secondary to AP. Contrasting evidence exists on the role of anticoagulation for the management of SVT in AP.[14–17] Anticoagulation in the setting of AP presents significant challenges, primarily due to the increased risk of hemorrhage. This risk is further increased by the frequent need for invasive procedures and necrosectomy in these patients.[18] Therefore, anticoagulation should probably be reserved for individuals who are at risk for nonrecanalization.

We evaluated the risk factors associated with nonrecanalization. In our study, BISAP score ≥2 emerged as the only significant risk factor for nonrecanalization of SVT in AP. This strengthens the fact that the severity of pancreatitis correlates with local complications and plays a significant role in long-term outcomes.

Our study has several strengths, including being the largest prospective study to date, standardized severity classifications, and the systematic long-term follow-up with Doppler ultrasound. We acknowledge certain limitations, including the single-center study design and the relatively small sample size, particularly for anticoagulated patients. Anticoagulation was initiated based on individualized physician assessment rather than standardized criteria. A randomized controlled trial design would avoid potential selection bias by standardizing anticoagulation initiation criteria across treatment groups. Cross-sectional imaging is usually not done for patients with mild AP, and therefore, there is a chance that the actual incidence of SVT in AP may be more than what has been reported in this series. The substantial proportion (30%) of patients with collaterals at SVT diagnosis likely reflects the conventional investigation sequence, wherein initial abdominal ultrasonography, with inherently limited sensitivity, precedes the more definitive contrast-enhanced computed tomography performed later in the disease course. Multivariate analysis could not be done due to the relatively small sample size of anticoagulated patients. These findings highlight the need for larger, multicenter studies to define management strategies and to identify subgroups of patients who might benefit most from anticoagulation.

To conclude, the majority of patients with splanchnic venous thrombosis in patients with acute pancreatitis spontaneously recanalize on follow-up. The severity of acute pancreatitis predicted nonrecanalization of SVT.

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