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STUDY OF SPLANCHNIC VENOUS THROMBOSIS IN PATIENTS WITH ACUTE PANCREATITIS IN A TERTIARY CARE HOSPITAL

Gastroenterology

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ABSTRACT

Background: Acute pancreatitis (AP) is an acute inflammatory process affecting the pancreas, which, when severe, can lead to a multitude of local and systemic complications. Splanchnic vein thrombosis (SVT) is a recognised vascular complication of moderate-to-severe AP. SVT can involve the portal vein, the splenic vein, and the superior mesenteric vein, in isolation or in combination. Life-threatening complications such as gastrointestinal bleeding, intestinal ischaemia, necrosis, and those related to portal hypertension can manifest. **Aims:** Incidence of SVT in patients with acute pancreatitis, its relation with severity of pancreatitis, other local complications, and the outcome of SVT. **Study Design:** Prospective observational study. **Methods:** This study was conducted at the Department of Medical Gastroenterology, Madras Medical College, between June 2024 and June 2025. One hundred and two patients fulfilling the revised Atlanta criteria for the diagnosis of acute pancreatitis were consecutively included. Demographic, clinical, and laboratory data were collected. SVT was diagnosed if an actual thrombus was detected in the vein, or if imaging showed the vein compressed or non-visualisation of veins with collaterals formation. Scoring systems, such as the Bedside Index of Severity in Pancreatitis (BISAP) and CT Severity Index (CTSI), were used to assess severity. Patients with SVT were followed up after 3 months with a portal venous (PV) Doppler and an esophago gastroduo denoscopy (EGD). Statistical comparisons were performed using the chi-square test and t-test. $P < 0.05$ was considered statistically significant. **Results:** Among 102 AP participants, 77% were male. Alcohol constituted the predominant etiology (51.96%). SVT was seen in 20 patients (19.6%). The splenic vein was the most commonly involved vessel, seen in 14 patients (70%). A BISAP score of ≥ 3, CTSI ≥ 7, and the presence of acute necrotic collection, pleural effusion, and ascites were significantly associated with SVT. The average length of stay in the hospital was much longer for the SVT group (32.8 ± 7.27 days) than for the non-SVT group (8.89 ± 2.97 days). Percutaneous drainage was needed in 40% of SVT patients. Two patients with SVT died during their hospital stay. Three patients experienced upper gastrointestinal bleeding: one due to fundal varix and two attributable to pseudoaneurysms, managed via endoscopic and radiologic interventions, respectively. At the three-month follow-up, 33.34% (6 of 18 patients) achieved recanalisation, regardless of anticoagulant administration. Varices were identified in three patients (16.66%) during follow-up EGD. **Conclusion:** SVT in acute pancreatitis is more frequently encountered than is usually thought. In patients with severe pancreatitis and associated local complications, evaluation for SVT should be done. Prompt management is needed to prevent long-term complications of portal hypertension. In our study, anticoagulation did not affect the recanalisation rate. Multicenter longitudinal studies are required to confirm this.

KEYWORDS

Acute pancreatitis, splanchnic venous thrombosis, BISAP, CTSI, local complications, anticoagulation, recanalisation

INTRODUCTION:

Acute pancreatitis (AP), an inflammatory condition of the pancreas, is linked to considerable morbidity and mortality.[1] The inflammatory process may be confined to the pancreas or extend to regional or distant organs, resulting in local and systemic complications.[2] In acute pancreatitis (AP), splanchnic vein thrombosis (SVT) is a recognised vascular complication. [3] SVT may affect the portal vein (PV), splenic vein (SV), and superior mesenteric vein (SMV), either individually or together. [4]The prevalence of SVT in AP, as indicated by research, ranges from 1.8% to 36.5%. [5-7] Factors contributing to SVT encompass the anatomical link of the large mesenteric arteries to the pancreas, the prothrombotic characteristics of the acute inflammatory process, the systemic reaction to injury, hypovolemia, and fluid shifts. Peri-pancreatic inflammatory changes and fluid accumulations facilitate the onset of splanchnic vein thrombosis (SVT). [6,8,9]Complications such as hepatic decompensation, bowel ischaemia, and upper gastrointestinal haemorrhage from gastro-oesophageal varices may arise owing to SVT. [10] To prevent these problems, prompt anticoagulant therapy is essential. The existing evidence indicates the use of anticoagulation for portal vein involvement, multivessel involvement, and thrombus expansion. [4,11,12] This study aimed to assess the incidence of SVT in acute pancreatitis, its correlation with the severity of pancreatitis, local complications, and the outcomes of SVT in a tertiary care hospital.

Objective Of The Study:

To estimate the incidence of Splanchnic venous thrombosis in Acute Pancreatitis, its association with severity, local complications and the outcome of SVT

MATERIALS AND METHODS

This prospective observational study was conducted at the Madras Medical College, Department of Medical Gastroenterology, over one year. The consecutive sampling method was used, and data were collected. The Institutional Ethical Committee of the Madras Medical College approved the study, IEC-MMC/Approval/05062024

Study Population:

Between June 2024 and June 2025, 102 patients aged 12 years or older, fulfilling the Revised Atlanta Criteria for Acute Pancreatitis, were consecutively recruited. [13]

Exclusion Criteria

1. Chronic liver disease
2. Chronic kidney disease
3. Malignancy
4. Pregnancy
5. Chronic Pancreatitis
6. Patients with a known thrombotic disorder

Data Collection

Information on demographic, clinical, and laboratory features and imaging parameters was collected prospectively during admission. Severity scoring systems like Bedside Index of Severity in Pancreatitis (BISAP) and CT Severity Index (CTSI) were used to assess the severity. [14] Patients were followed up until discharge or death to assess for the severity, local complications, and outcome. Interventions done (radiological and endoscopic) and the use of anticoagulation were recorded.

Diagnosis of splanchnic vessel thrombosis was based on contrast-enhanced computed tomography (CECT) of the abdomen, magnetic resonance imaging (MRI), and or Doppler ultrasonography of the abdomen. SVT was diagnosed if a thrombus was seen in the vein, if the vein appeared compressed, or if the vein could not be visualised, but collateral vessels were present. [5,6,15]

Follow-up and SVT Outcome

All patients with SVT were followed three months after discharge with

a PV Doppler and an EGD to assess recanalisation and the development of varices.

Statistical Analysis

Statistical analysis was performed using SPSS Version 26. Mean, standard deviation, and range were calculated for continuous variables, while frequencies and percentages were determined for categorical variables. The SVT and non-SVT groups, along with the anticoagulant and non-anticoagulant groups, were compared. The independent t-test and chi-square test were employed to compare continuous and categorical variables, respectively. A p-value of < 0.05 was considered statistically significant.

RESULTS

Out of 102 patients with AP, 78 patients (76.47%) were males and 24 patients (23.53%) were females. A total of 20 patients (19.60%) developed SVT, 19 (95%) of whom were male, and the remaining one patient (5%) was female. Mean age was 40.85 ± 13.89 and 40.00 ± 8.57 years in the SVT and non-SVT groups, respectively. Alcohol was the most common aetiology, 15/20 (75%) in patients with SVT (Table 1). Two patients with biliary pancreatitis and one patient each with hypertriglyceridemia, drug-induced, and idiopathic pancreatitis had SVT. The most involved vessel was the splenic vein (9 of 20), along with PV + SMV and PV + SMV + SV (3 each), SMV and SMV + SV (2 each), and one patient who had PV thrombosis (Table 2). In the SVT group, 18 out of 20 (90%) and 15 out of 20 (75%) patients had pleural effusion and ascites, respectively, while in the non-SVT group, only 6 out of 82 (7.31%) and 7 out of 82 (8.53%) patients had these conditions, respectively (Table 3). Local complications like acute necrotic collection (ANC) were seen in 16 out of 20 patients with SVT, and acute peripancreatic fluid collections (APFC) in 1 out of 20 SVT patients (Table 3). A BISAP score of ≥ 3 ($P = 0.001$) and CTSI ≥ 7 ($P = 0.001$) were significantly associated with the presence of SVT (Table 1). Interventions in the form of percutaneous drainage (PCD) were required in 8 out of 20 (40%) patients and radiological embolisation of splanchnic arterial pseudoaneurysms in 2 out of 20 (10%) patients in the SVT group, whereas no patients in the non-SVT group received such treatments. Three patients had upper gastrointestinal bleeding during their hospital stay. Two were due to splanchnic arterial pseudoaneurysms, which were managed by embolisation, and one was due to a gastric fundal varix, which was managed endoscopically. Two patients (1.96%) died during their hospital stay, both belonging to the SVT group (10%), due to multiorgan dysfunction. The mean duration of hospital stay was 32.80 ± 7.27 in the SVT group and 8.89 ± 2.97 in the non-SVT group ($P=0.001$) (Table 1).

Clinical Profile Of SVT

Involvement of a single vessel was seen in 12 (60%), double vessel in 5 (25%), and triple vessel in 3 (15%) patients (Table 2). The most commonly involved vessel was the splenic vein, in 14 out of 20 patients (70%); it was isolated in 9 out of 20 (45%) and in combination with other vessels in the remaining patients. Anticoagulation was started in patients who had double or triple vessel involvement, single vessel (PV or SMV) involvement, and in one patient who had isolated splenic vein involvement, as he had co-existing infrarenal IVC thrombus due to compression by ANC. Patients were initially anticoagulated with unfractionated or low molecular weight heparin, subsequently transitioning to oral warfarin, with dosage adjustments made to maintain an international normalised ratio of 2 to 3.

SVT Outcome

On follow-up, 10 out of the remaining 18 patients were receiving anticoagulation, as 2 patients were lost to follow-up due to in-hospital death (Table 4). Recanalisation was seen in six patients (33.33%), of whom four received anticoagulation and two did not ($P = 0.502$) (Table 5). Collaterals or cavernoma formation were seen in 13 out of 18 (72.22%), 7 of whom were anticoagulated and 6 were not ($P = 0.813$) (Table 5). One patient with a recanalised vessel also showed evidence of collaterals. No bleeding manifestations were reported in either group on follow-up. Varices on follow-up (oesophageal and/or gastric) were present in 3 patients (16.66%), of whom 2 patients were anticoagulated.

DISCUSSION

Splanchnic venous thrombosis is a well-known vascular complication of acute pancreatitis. Literature is limited, and its natural history remains elusive. We have reported our experience on the incidence of SVT and its outcome at a tertiary care referral center. The incidence of SVT in AP in our study was 20 in 102 patients (19.60%). Ahmed et al. previously reported an incidence of SVT in 27.1% of subjects with AP; Easler et al. identified SVT in 18% of AP patients; and Junare et al. found SVT in 22.85% of patients. [5,6,16] The pathogenesis of SVT is multifactorial, with pancreatic and peripancreatic inflammation being a significant contributor. Inflammation results in cellular infiltration, edema, and systemic activation of hemostasis, leading to the deposition of platelet and fibrin thrombi. [8,17] Gonzalez et al. documented local complications in 19 of 20 patients with SVT (95%) in their study. [6] Likewise, Junare et al. reported local complications in all 24 patients with SVT (100%) in their cohort. [4] Local complications like ANC and AFPC were observed in 17 out of 20 patients (85%) in our study, of which 16 were ANC. Only 3 out of the remaining 82 patients (3.66%) without local complications developed SVT. Markers of disease severity, like a BISAP score ≥ 3 and a CTSI ≥ 7, were significantly associated with the occurrence of SVT. Splenic vein involvement was the most common, seen in 14 out of 20 patients (70%) in isolation or in combination, followed by SMV in 10 out of 20 patients (50%) and portal vein in 7 out of 20 patients (35%). Higher splenic vein involvement may be explained by its proximity to pancreatic and peripancreatic inflammation and collections. Previous studies suggest that the advancement of SVT may be complicated by portal hypertension, hepatic decompensation, and intestinal ischaemia due to thrombus extension into the PV and SMV. [5,6,15] Available literature indicates that the rate of spontaneous recanalisation may reach 30%, particularly in cases of splenic vein thrombosis. [8,18] The 10-year recurrence-free survival rate is highest for isolated splenic vein thrombosis; however, this may not apply to PV or SMV thrombosis. [19] Previous research has indicated a significant mortality rate in acute mesenteric vein thrombosis. [20] The available consensus data indicate that anticoagulation is advised in cases of SVT with portal vein, superior mesenteric vein involvement, or thrombus progression. [4,10,11,21]. In our study, patients with PV, SMV or multivessel involvement and one patient with splenic vein thrombosis who had co-existent infrarenal IVC thrombosis were anticoagulated. Regarding the outcomes of SVT, such as the development of varices, collateral vessels, cavernoma formation, recanalisation, and mortality, we did not find a statistically significant difference between the anticoagulated and the non-anticoagulated groups. Three patients had upper gastrointestinal bleeding during their hospital stay. Two were due to splanchnic arterial pseudoaneurysms, which were managed by embolisation, and one was due to a gastric fundal varix, which was managed endoscopically. Bleeding related to anticoagulation therapy was not observed in our study. Given the scarcity of literature, the routine application of anticoagulation in all patients with SVT may not be prudent. Multicentric longitudinal studies are needed based on which recommendations can be made in future.

Limitations

Our study was conducted at a single tertiary care center with a relatively small sample size, which may limit the generalizability of the results. Furthermore, thrombophilia testing was not conducted on all patients, and it is possible that some of our patients possessed an underlying thrombophilic disorder. A study assessing the impact of thrombophilia in acute pancreatitis, however, found no significant difference between the SVT and non-SVT groups (16). The follow-up period was restricted to three months; an extended follow-up could have yielded greater understanding of the long-term effects of SVT, such as chronic portal hypertension or recurrent variceal hemorrhage. Despite these limitations, our experience provides a valuable understanding of the prevalence, clinical features, treatment, and outcomes of SVT in AP, which could help clinicians make decisions and provide a basis for further research given the paucity of existing literature.

CONCLUSION

Splanchnic venous thrombosis is a common and clinically relevant complication of acute pancreatitis, especially in its severe and necrotizing variants. Its incidence is significantly associated with disease severity and local inflammatory complications. Despite anticoagulation not affecting recanalisation rates in this study, timely identification and tailored care are essential for enhancing outcomes and reducing long-term complications of portal hypertension.

Demographic Characteristics, Etiology, Severity Scores And Hospital Stay

VARIABLES	SVT		Total	P Value
	Present	Absent		

Age In Years	MEAN AGE	40.85±13.89	40.00±8.57	40.16±9.89	0.729
	LESS THAN 30	4	12	16	0.788
	31-40	7	30	37	
	41-50	7	33	40	
	51-60	1	6	7	
	MORE THAN 60	1	1	2	
SEX	MALE	19	59	78	0.029
	FEMALE	1	23	24	
BISAP Score	0 TO 2 (Low Mortality)	3	78	81	0.001
	3 TO 5 (High Mortality)	17	4	21	
CT Severity Index (CTSI)	0 TO 3 (MILD)	0	73	73	0.001
	4 TO 6 (MODERATE)	6	8	14	
	7 TO 10 (SEVERE)	14	1	15	
Etiology	Idiopathic	1	6	7	0.067
	Alcohol	15	38	53	
	Biliary	2	32	34	
	Drug Induced	1	1	2	
	Hypertryglyceridemia	1	1	2	
	Pancreatic Divisum	0	1	1	
	Post ERCP	0	3	3	
Hospital Stay Duration		32.80±7.27	8.89±2.57	13.57±5.98	0.001

Distribution Of Splanchnic Venous Thrombosis

Splanchnic Vein Thrombosis	**Number Of Patients**	**Percentage**
PRESENT	20	19.60%
ABSENT	82	80.34%
Vessel Involved	**Number Of Patients**	**Percentage**
PORTAL VEIN	1	5%
SUPERIOR MESENTERIC VEIN	2	10%
SPLENIC VEIN	9	45%
PV+SMV	3	15%
SV+SMV	2	10%
PV+SV+SMV	3	15%

Local Complications And SVT Association

Findings		**Splanchnic Vein Thrombosis**		**Total**	**P Value**
		Present	**Absent**		
Local Complications	Acute Necrotic Collection	16	2	18	0.001
	Acute Peripancreatic Fluid Collection	1	19	20	
	NIL	3	61	64	
ASCITES	Present	15	7	22	0.001
	Absent	5	75	80	
Pleural Effusion	Present	18	6	24	0.001
	Absent	2	76	78	
Percutaneous Drainage	Required	8	0	8	0.001
	Not Required	12	82	94	

Collaterals Formation And Recanalisation On Follow- Up

Anticoagulants	**Number Of Patients**	**Percentage**
GIVEN	10	55%
NOT GIVEN	8	45%
Collaterals/Cavernoma	**Number Of Patients**	**Percentage**
PERIGASTRIC	1	6%
PERISPLENIC	6	34%
PERISPLENIC/PERIGASTRIC	5	28%
PORTAL CAVERNOMA	1	6%
ABSENT	5	28%
Recanalisation	**Number Of Patients**	**Percentage**
PRESENT	6	34%
ABSENT	12	66%

Effect Of Anticoagulation

On Follow-up	**Anticoagulants**		**Chi-square Test**
Collaterals/Cavernoma	GIVEN	NOT GIVEN	P Value
Present	7	6	0.813
Absent	3	2	
Recanalisation	GIVEN	NOT GIVEN	P Value
Present	4	2	0.502
Absent	6	6	

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