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Original research article

Prevalence and pattern of splanchnic vein thrombosis in cases of acute pancreatitis in a tertiary care hospital

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Abstract

Background: Splanchnic veins thrombosis (SVT) is one of the rare but known complications of Acute Pancreatitis (AP). It usually involves Splenic vein (SPV), Superior mesenteric vein (SMV) and often Portal vein (PV) either individually or together. Though the cause is multifactorial, a direct inflammatory process is implicated in the pathogenesis of SVT in AP. SVT can be sometimes an incidental finding in the radiological evaluation, but it also presents as severe pain, portal hypertension, hepatic dysfunction and bowel ischemia in a life-threatening scenario. Present study aims at understanding incidence, pattern, complications of SVT in AP.

Materials and Methods: A serial study of 64 cases of acute pancreatitis who underwent Contrast Enhanced Computed Tomography was conducted at tertiary care center, department of general surgery, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur, Karnataka from the period of June 2022 to January 2023.

Results: Out of 64 cases studied, 45 were male, 19 were female with mean age of 38.5 years. Most common cause of AP was Ethanol abuse, followed by Gallstones, Main pancreatic duct (MPD) anomaly, Anomaly of duodenal papillae and idiopathic. 13 (20.31%) Patients had SVT. 11 (85.71%) were male, 2 (14.29%) were female.

Conclusion: SVT is one of the known complications in AP with increase in incidence, severity and with presence of peripancreatic collection.

Keywords: Acute pancreatitis, splanchnic vein thrombosis, computed tomography

Introduction

Splanchnic veins thrombosis (SVT) is common among patients with cancer, liver cirrhosis and systemic inflammation. SVT can influence course of many pancreatic diseases. Pancreatitis-induced splanchnic vein thrombosis (PISVT) found in 22.6% of AP cases and 12.4% of chronic pancreatitis (CP).

SVT is one of the rare but known complications of Acute Pancreatitis (AP).

It mostly involves Splenic vein (SPV), Superior mesenteric vein (SMV) and often Portal vein (PV) either individually or in combination.

Though the cause is thought to be multifactorial, a direct inflammatory process is implicated in the pathogenesis of SVT in AP.

SVT can be sometimes an incidental finding in the radiological evaluation, but it also presents with severe pain, portal hypertension, hepatic dysfunction and bowel ischemia in a life-threatening scenario.

In many cases, the clinical consequences of SVT depend on the number of affected vessels and the potential to produce collateral circulation. The latter applying to all chronic processes. SVT can lead to life threatening complications such as gastrointestinal bleeding, intestinal ischemia, necrosis and those related to portal hypertension like ascites, splenomegaly, encephalopathy.

Taking into consideration the clinical significance of these issues, we examined the connection between SVT and the severity of AP at understanding incidence, pattern, complications and outcome of SVT in AP.

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Need for the Study

Acute Pancreatitis is an acute inflammatory process of pancreas with variable clinical presentation.
About 80-85% of acute pancreatitis patients require only clinical monitoring and supportive care as it is self-limiting.
About 15-20% evolves to moderate-severe disease leading to activation of pancreatic enzymes leading to cascade of reactions resulting in auto digestion of pancreas, necrosis, peri-pancreatic collection.
These peri-pancreatic collection cause vasculitis of the surrounding vessels, external compression effects along the systemic activation of coagulation and hypercoagulable state. This is thought to cause SVT.
As SVT is not that common, chances of missing out the diagnosis is more, hence in this study we have seen the prevalence of SVT in cases of moderate to sever acute pancreatitis cases.
In this study we have also seen the pattern of SVT in cases of moderate to severe acute pancreatitis cases.

Objectives

- To determine the prevalence of SVT in cases of acute pancreatitis.
- To determine the pattern of SVT in cases of acute pancreatitis.

Material and Methods

Study Design

This was a retrospective study which was carried out among 64 patients diagnosed with acute pancreatitis who underwent Contrast Enhanced Computed Tomography at tertiary care hospital, department of General Surgery, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur, Karnataka from the period of June 2022 to January 2023.

Duration of the study: 8 months.

Source of Data: Patients diagnosed with Acute pancreatitis who underwent CECT abdomen and pelvis at SIMS&RH Tumkur.

Inclusion criteria: Acute pancreatitis who underwent Contrast Enhanced Computed Tomography (CECT)

Exclusion criteria

- Chronic pancreatitis.
- Known malignancy.
- Liver cirrhosis or portal hypertension.
- Established prothrombotic states.
- Pre-existing splanchnic vein thrombosis.

Sampling method: Purposive sampling method.

Method of collection of data: Data of 64 cases of acute pancreatitis who underwent CECT was conducted at tertiary care center, Department of General surgery, Shridevi Institute of Medical Sciences and Research Hospital, Tumkur, Karnataka from the period of June 2022 to January 2023 was collected.
AP was diagnosed and divided into severity grades based on the Atlanta 2012 criteria as follows: mild (MAP), moderately severe (MSAP) and severe acute pancreatitis (SAP).
SVT was divided into 3 groups:

1. Portal vein thrombosis (PVT).
2. Splenic vein thrombosis (SpVT).
3. Superior mesenteric vein thrombosis (SMVT).

Data related to SVT with respect to etiology, incidence, pattern, complications and outcome were noted on IP basis.
Analysis using statistics are done inference drawn.

Statistical Method: Appropriate descriptive statistical analysis done on purposive samples using SPSS software. P values are derived with significance level of 0.05.

Observation and Results

64 Patients with acute pancreatitis were included in the present study. 40 (63%) patients were in the age group of 20-40 years. With 45 males and 19 females with mean age of 38.5 years (Table No. 01) (Fig no. 01).

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Table 1

Gender ratio	n	Percentage
Male	45	70.31
Female	19	29.69
Total	64	100

Gender Ratio

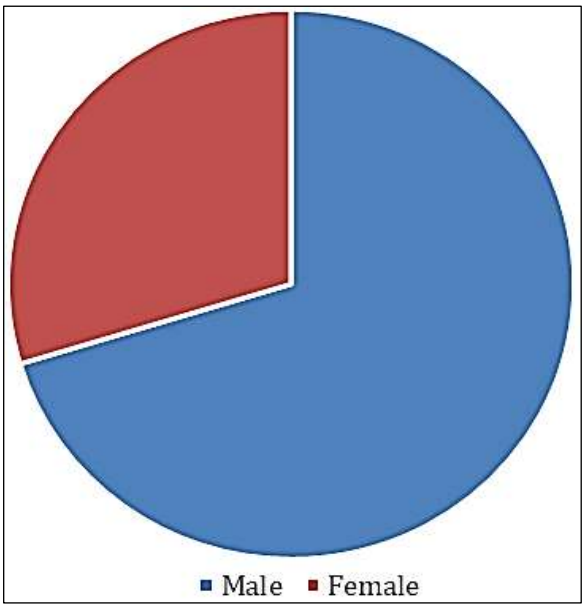

Fig 1

Chronic ethanol consumption was the most common etiology in 65.16% (n=42) patients. Followed by gallstones as cause with 20.22% (n=13) incidence. 14.6% (n=09) had idiopathic cause. (Table No.02) (Fig no.02)

Table 2

Etiology	n	Percentage
Alcohol	42	65.16
Gallstones	13	20.22
Idiopathic	09	14.60

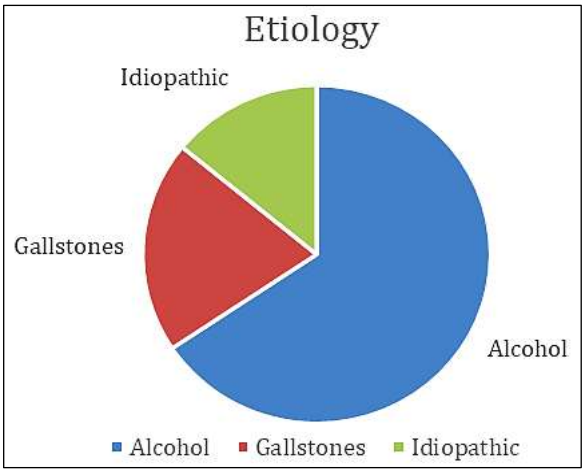

Fig 2

In this study 28 patients were diagnosed with acute severe pancreatitis, 10 were diagnosed moderately severe pancreatitis and remaining 26 were mild pancreatitis.
13 (20.31%) of the patients in this study had SVT, in which 8 were acute severe pancreatitis, 4 were moderately severe acute pancreatitis and 1 was mild pancreatitis. In that 11 were males (85.71%) and 2

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females (14.29%). (Table No.3) (Fig no.03)

Table 3

SVT-gender wise	n	Percentage
Male	11	85.71
Female	02	14.27

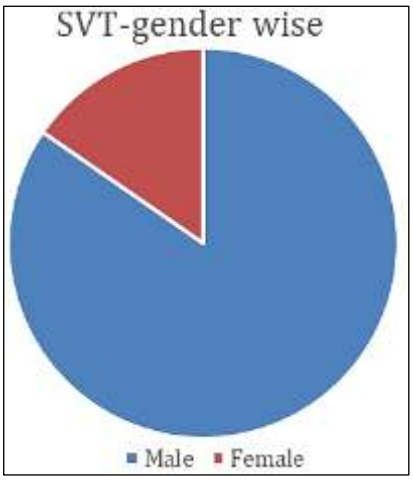

Fig 3

1 patient had isolated splenic vein thrombosis, 1 patient had splenic vein + portal vein thrombosis, 2 patients had isolated splenic vein thrombosis + portal vein thrombosis along with portal vein hypertension and 9 patients had involvement of all three vessels with varied features of bowel ischemia/hepatic dysfunction. (Table No.04)

Table 4

Thromosis of veins	N
SPV	1
SPV + PV	1
SPV + PV + Portal HTN	2
Triple vessel thrombosis	9

Conclusion

Splanchnic vein thrombosis is a relatively common observation in patients with severe AP; in the vast majority of patients, it is associated with pancreatic necrosis and peripancreatic collections.

Localization of peripancreatic collections and splanchnic vein thrombosis suggests that compression and perivascular inflammation are important mechanisms.

SVT is seen more commonly in alcohol-induced AP. It is associated with the presence of local complications of AP.

Among SVT in AP cases, involvement of all 3 vessels is common.

Discussion

In view of limited literature available, natural history of SVT in acute pancreatitis remains elusive. Incidence of SVT is widely variable ranging from 1% to 24%.

This is due to heterogeneity of the study subjects (mild vs. severe AP, acute vs. chronic pancreatitis), etiologies and imaging modality used for diagnosis (ultrasonography vs. CT scan) SVT remains a frequent complication in cases of acute pancreatitis.

Though the pathogenesis of SVT remains multifactorial, pancreatic and peripancreatic inflammation plays a key role. Inflammation leads to cellular infiltration, edema and systemic activation of hemostasis with consequent deposition of platelets and fibrin clot formation.

Compression of local vessels by peripancreatic collection along with cellular disruption of pancreas leads to venous stasis and activates coagulation.

There is also hypercoagulable state in acute pancreatitis due to increased effects of inflammatory mediators and increased synthesis of prothrombotic factors by liver resulting in SVT.

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