Research Article

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Can Upper Abdomen Pain Waning Over Time Hide Something More Sinister and Serious? Acute Portal Vein Thrombosis (APVT) is One of the Culprits

Gowhar Nazir[1], Minsha Hameed1, Inzamam Wani[2]*, Irfan Robbani[3], Shadab Maqsood[4] and Zahoor Paul[5]

[1]MBBS, MD, radiodiagnosis and imaging skims Srinagar

[2]MBBS, MD, radiodiagnosis and imaging (ongoing) skims Srinagar

[3]MBBS, MD, professor department of radiodiagnosis and imaging skims Srinagar

[4]MBBS, MD, assistant professor department of radiodiagnosis and imaging skims Srinagar

[5]MBBS, MD, assistant professor department of radiotherapy, GMC baramulla

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*Corresponding Author

* Inzamam Wani, MBBS, MD, radiodiagnosis and imaging, Skims Srinagar, Tel.: +91 6006408264, E-mail: inzywani786@gmail.com

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Abstract

Background: An obstruction or constriction of the portal vein caused by a blood clot is known as portal vein thrombosis (PVT). Thrombosis may form in the main body of the portal vein, its intra-hepatic branches, or even the splenic or superior mesenteric veins (SMV). Cirrhosis of the liver and PVT commonly co-occur.

Aims and Objective: The aim was to study the distribution of possible risk factors and clinical presentation in patients of Acute Portal Vein Thrombosis (APVT).

Method: The study was performed in the Department of Radiology, SKIMS Srinagar between July 2019 and January 2022. Forty patients who were admitted with Acute Portal Vein Thrombosis to the hospital were studied.

Results: Local risk factors were found in 19 patients (47.5%), 15 patients had CLD (37.5%) and 15 patients had a general pro-thrombotic risk factor (37.5%) and no risk factor was found in two patients (5%). The disease was symptomatic in 75% and incidental in 25% of patients. Among the symptomatic patients, the majority (37.5%) presented with acute upper abdominal pain.

Conclusion: Acute Portal Vein Thrombosis presents as upper abdominal pain which is severe initially and tends to fade in intensity over a week with fever and abdominal distension due to a high incidence of ascites. 25% of the patients are asymptomatic which makes screening important in high-risk patients, CLD patients being the most common high-risk group.

Keywords: Portal vein thrombosis (PVT); superior mesenteric vein (SMV); chronic liver disease (CLD); risk factors

Introduction

The second most frequent cause of portal hypertension in the west is portal vein thrombosis (PVT). PVT is due to obstruction of the lumen of the portal vein by a clot. In its chronic stage, there is a replacement of the portal vein by cavernomas, which are newly created tortuous arteries with hepatopetal flow that replace the portal vein lumen.

The extent of the thrombosis, the rate at which it develops, and the presence or absence of cirrhosis affect the clinical presentation differently. When the thrombosis is restricted to one of the portal branches, there is lobe atrophy on the affected side and hypertrophy of the contralateral lobe on the unaffected side [1].

Thrombosis in the portal venous system can involve the intra or extrahepatic tract or occur in the splenic and the superior mesenteric veins too. This occurs secondary to disturbances in one of the elements of Virchow's Triad [2].

The symptoms of PVT range from accidental, asymptomatic findings to serious ones like portal hypertension and variceal haemorrhage. Presentations and responses to anticoagulation depend on the number of vessels affected and the level of thrombosis (partial vs. total). The correlation of the PVT with the presence of cirrhosis, procoagulant haematological conditions in non-cirrhotic individuals (mainly chronic myeloproliferative conditions), abdominal infections/surgery, and intra or extrahepatic malignancy, all of which induce hypercoagulable states, adds to the PVT's complexity [3].

The major risk factors causing Portal Vein Thrombosis are hereditary and acquired pro-thrombotic risk factors, local risk factors, cirrhosis, tumours and surgeries involving the portal venous system [4-14]. Recently, there have been a few documented cases of Acute Portal Vein thrombosis in patients with COVID-19 infection [15-17].

Both symptomatic and asymptomatic people can have APVT diagnosed. In asymptomatic, accidentally discovered APVT, a detailed evaluation of the patient is required to identify risk factors, but in symptomatic individuals, imaging can quickly confirm or rule out the diagnosis of APVT. According to the findings of a recent investigation, the same treatment approaches should be used since the prognosis of incidentally identified splanchnic vein thrombosis is similar to that of clinically diagnosed splanchnic vein thrombosis [18].

Acute PVT is accompanied by severe, protracted abdominal pain, which can start suddenly or develop gradually. The loin may experience the most intense pain. A lack of guarding contrasts with the intensity of pain. Although intestinal ileus is widespread, there are no overt indications of intestinal obstruction. At this early stage, an enlarged spleen should point towards myeloproliferative neoplasms. Even when there is no specific inflammatory focus that is producing it, high undulating fever is common in patients with APVT. Septic Pyle phlebitis is characterized by chills and a spike in temperature [19]. It might be challenging to separate the clinical characteristics of acute PVT from those of the local trigger. Hematochezia and bloody diarrhoea indicate mucosal ischemia in the absence of inflammatory bowel disease. Unless they are bleeding from ruptured gastric or oesophagal varices, patients with portal cavernoma often show no symptoms.

The presence of a portal cavernoma is suggested by portal hypertension in the absence of liver disease. Ascites is seen after bleeding or any acute inflammatory insult and is easy to treat. Portal cavernoma cholangiopathy is substantially less common.

Aims and Objective

The aim was to study the distribution of possible risk factors and clinical presentation in patients of Acute Portal Vein Thrombosis (APVT).

Materials and Methods

The study was performed in the Department of Radiodiagnosis and Imaging, SKIMS Srinagar in collaboration with the Departments of Medical Gastroenterology and Surgical Gastroenterology. Between July 2019 and January 2022, forty patients were enrolled prospectively for this study and all of them consented to be included in the study. These patients had clinical suspicion of APVT. A Duplex Doppler scan of the patients was done and the partial/complete absence of colour flow and spectral waveform in the portal vein was a criterion for inclusion of patients into this study while the presence of collateral flow was a criterion for exclusion [Figure 1]. Multiple parameters related to the patient were collected and recorded which included age, gender, clinical presentation, and the presence of underlying risk factors (historically).

Cross-sectional imaging was done by using CECT and multiple imaging parameters from colour Doppler and CECT were recorded. Once acute PVT was confirmed on CECT, patients underwent various investigations to look for possible risk factors. The clinical presentation of these patients was assessed and recorded.

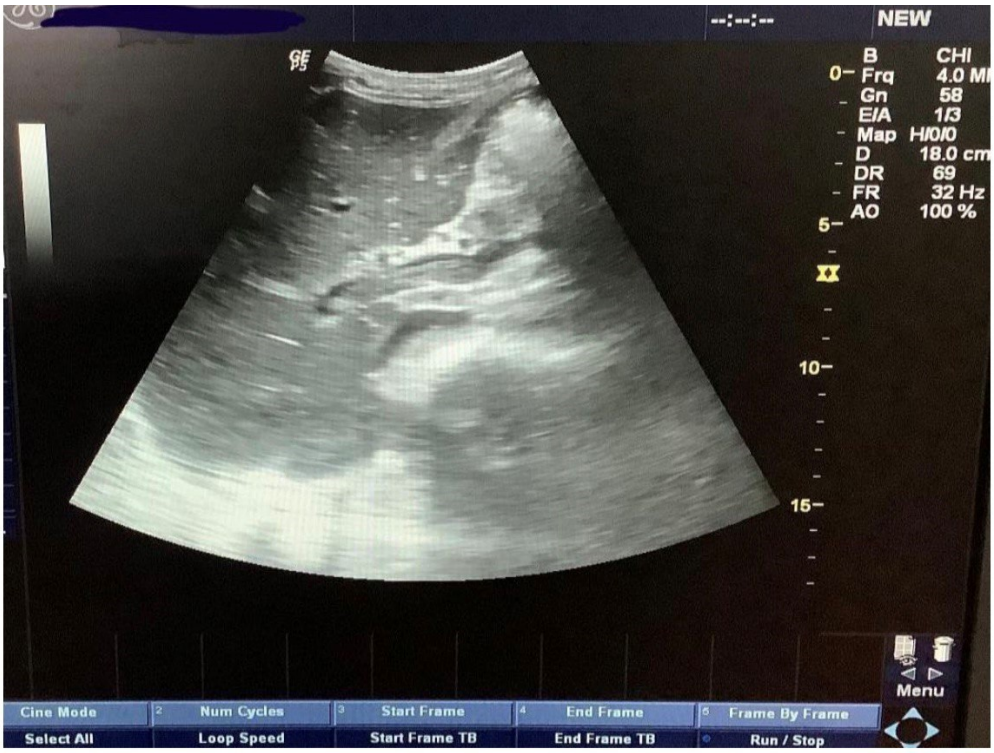

Figure 1: Gray-scale sonogram of the portal vein showing echogenic contents within the lumen suggestive of PVT.

Data Analysis

The recorded data was compiled and entered into a spreadsheet (Microsoft Excel) and then exported to data editor of SPSS Version 20.0 (SPSS Inc., Chicago, Illinois, USA). Continuous variables were expressed as Mean ± SD and categorical variables were summarized as frequencies and percentages.

Ethical Clearance

Before the start of the study, ethical clearance was sought from the Institutional Ethics Committee, Sher-i-Kashmir Institute of Medical Sciences (SKIMS), Soura.

Results

The age of the patients ranged between 32 and 70 years with a mean age of 56.1 years. Out of the 40 patients with acute PVT, the highest incidence was found in the age group of 51-60 years (45%) [Table 1].

Table 1: Age distribution

Age (Years)	Number	Percentage
≤ 40	3	7.5
41-50	8	20
51-60	18	45
61-70	9	22.5
> 70	2	5
Total	40	100

Mean ± SD (Range)=56.1 ± 10.19 (32-75)

In our study, 26 (65%) of the patients were males and 14 (35%) were females [Table 2].

Table 2: Gender distribution

Gender	Number	Percentage
Male	26	65
Female	14	35
Total	40	100

The disease was symptomatic in 75% of the patients and incidental in 25% of them. In the symptomatic patients, the majority (37.5%) presented with acute abdominal pain; followed by abdominal distension (27.5%); fever (25%); vomiting (12.5%); nausea (10%); anorexia (7.5%); upper GI bleed (7.5%) and diarrhoea (5%) [Figure 2]. Most of the patients had a combination of the above-mentioned symptoms [Table 3].

Table 3: Clinical presentation of study patients

Clinical presentation	Number	Percentage
Abdominal pain	15	37.5
Abdominal distension	11	27.5
Fever	10	25
Vomiting	5	12.5
Nausea	4	10
Anorexia	3	7.5
Upper GI bleed	3	7.5
Diarrhea	2	5
Incidental	10	25

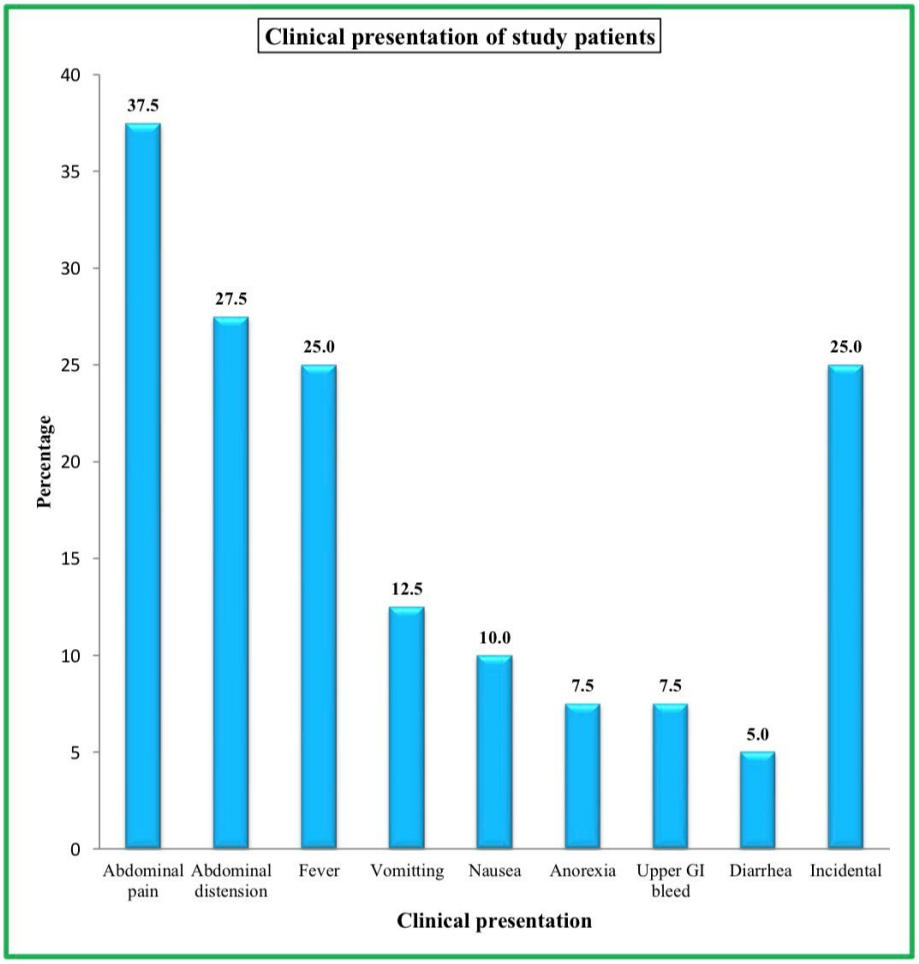

Figure 2: Multiple bar diagram showing clinical presentation of patients with APVT.

Local risk factor was found in 19 patients (47.5%), 15 patients had CLD (37.5%) and 15 patients had a general prothrombotic risk factor (37.5%). Out of these patients, 8 patients had a local risk factor in presence of a general prothrombotic risk factor [Figure 3]. No risk factor could be identified in two patients (5%) [Table 4].

Table 4: Various risk factors among study patients*

Risk factor	Number	Percentage
Local risk factor	19	47.5
CLD	15	37.5
General pro-thrombotic risk factor$	15	37.5
No identifiable cause	2	5
CLD + general pro-thrombotic risk factor	0	0
Local risk factor + general pro-thrombotic risk factor	8	20

*Patients can have more than one risk factor

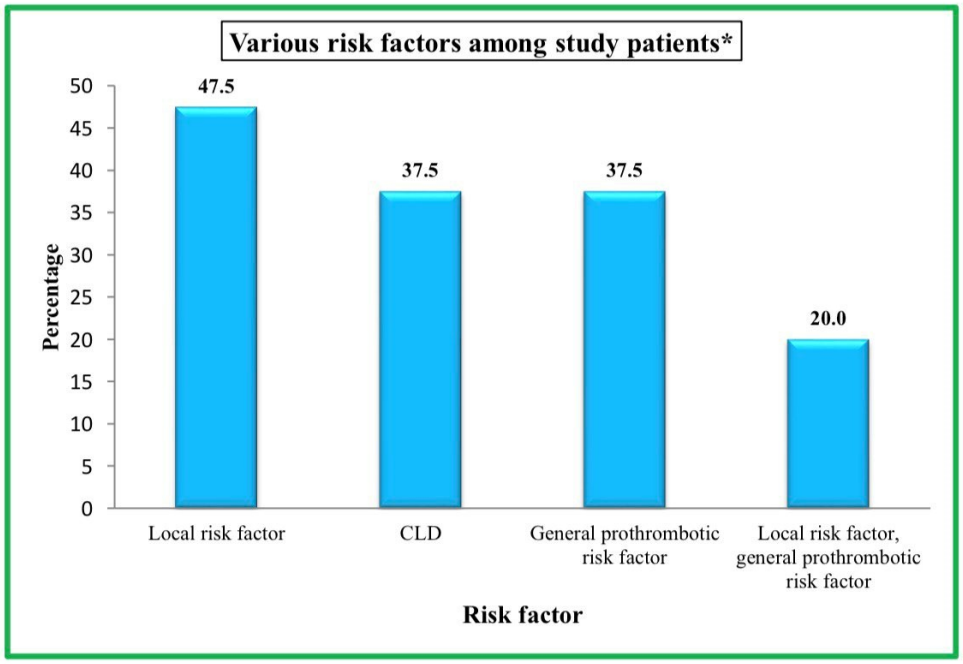

Figure 3: Multiple bar diagram showing various risk factors in patients of APVT.

The local risk factors included local malignancies (27.5%) like pancreatic Ca (n=4), HCC (n=3), Cholangiocarcinoma (n=3) and Ca stomach (n=1); inflammatory abdominal conditions (17.5%) like acute pancreatitis (n=3), liver abscess (n=2), peritoneal tuberculosis (n=1) and hypertrophic gastritis (n=1); and surgery (splenectomy). All of the patients in whom a general prothrombotic risk factor was found were non-cirrhotics [Figure 4]. Thus, out of the 25 non-cirrhotic patients, 60% (n=15) patients had a general prothrombotic risk factor which included a majority of patients with a Myeloproliferative Disorder (MPD). 32% of the non-cirrhotics with acute PVT had an MPD (n=8). In 12% of the non-cirrhotics, sepsis was found to be the prothrombotic risk factor and 16% of the non-cirrhotics had other thrombophilic conditions like APLA (n=2), Prothrombin gene mutation(n=1), Factor V Leiden mutation(n=1) [Table 5].

Table 5: Distribution of local and general risk factors*

		Number	Percentage
Local risk factor	Local malignancy	11	27.5
	Inflammatory abdominal condition	7	17.5
	Surgery (Splenectomy)	1	2.5
General pro-thrombotic risk factor (in non-cirrhotics)	MPD	8	32
	Sepsis	3	12
	Others (APLA, Prothrombin gene mutation, Fator V Leiden mutation)	4	16

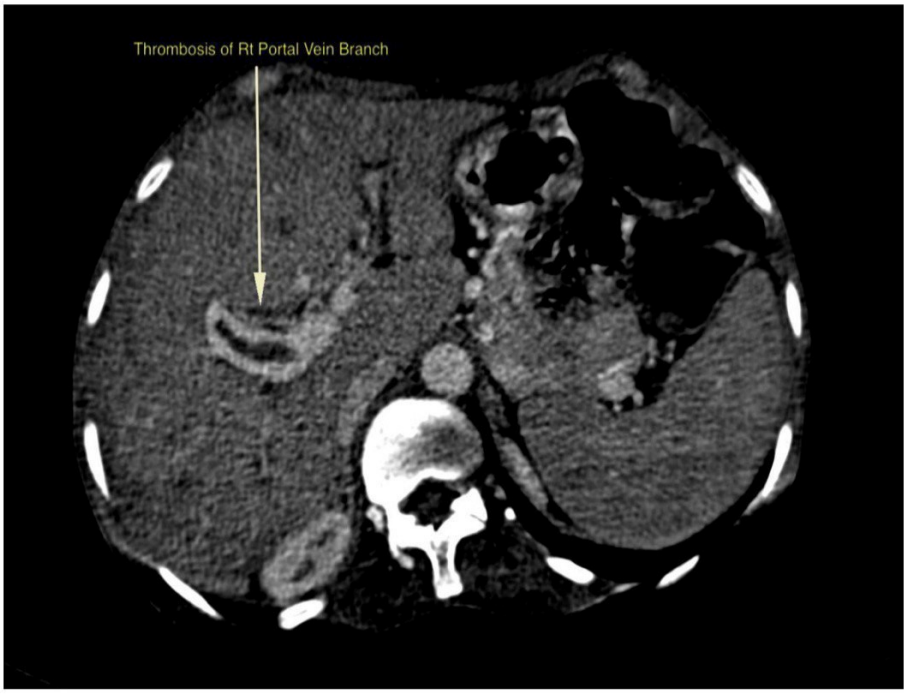

Figure 4: Axial CECT image in the portal phase showing acute thrombosis of the right portal vein branch in a non-cirrhotic liver.

Discussion

APVT has disastrous consequences, if not diagnosed early. The aetiology is diverse and the patients usually have vague signs and symptoms rendering clinical diagnosis difficult. In our study, the clinical features and risk factors for APVT were studied in detail.

Forty patients who were admitted with acute PVT to the hospital had a mean age of 56.1 years (range 32-70 years). The highest number was found in the age group of 51-60 years (45%). This corroborates with the study done by J. Attali [20] et al in which, the mean age of 23 patients with acute portal vein thrombosis was 57 years. The male: female ratio of patients in our study was 1.9:1.

A local risk factor was found in 47.5% of the patients. CLD was the risk factor in 37.5% of patients and 37.5% of patients had a general prothrombotic risk factor with 32% of patients having a combination of general and local risk factors. Out of the local risk factors, a local malignancy was the most common risk factor (Pancreatic Ca, HCC, cholangiocarcinoma and Ca stomach in that order). The most common inflammatory abdominal condition was acute pancreatitis. The general prothrombotic risk factors included Myeloproliferative Neoplasms, sepsis, APLA, Prothrombin gene & Factor V (Leiden) mutation. Previously, the study by Plessier [2] et al in 2010 had shown that at least one general or local risk factor was found in 52% and 21% of patients respectively. Our research also supplements the study published in 2020 by Jiahao Fan [21] which stated that Myeloproliferative Neoplasms were found to be the risk factor in 28.3% of patients of non-cirrhotic PVT (32% in our study). No identifiable cause could be found in 2 patients. In cirrhotics, no other inherited or acquired thrombophilia was found.

APVT was symptomatic in 75% of the patients and incidental in 25% of the patients. In the symptomatic patients, the majority (37.5%) presented with acute abdominal pain. The pain was acute in onset, mostly located in the upper abdomen, but sometimes described by the patient as generalized abdominal discomfort. The severity of pain was initially reported as >6/10 by all the patients on the Visual Analog Scale but the severity tended to fade over one week. Abdominal distension was the next most frequent complaint (27.5%) and was attributed to the presence of clinically and radiologically detectable ascites. However, all patients who had radiologically detectable ascites did not complain of abdominal distension. Fever was found to be a major presenting complaint (25%) in both the patients who had an inflammatory condition and those who did not. Nausea (12.5%) and vomiting (10%) were notable clinical features and diarrhoea was the presenting complaint in 5% of patients. Upper GI bleed in the form of hematemesis was seen in three patients (7.5%), however lower

GI bleed was not the presenting complaint in any patient. 7.5% of the patients had anorexia. Most of the patients had a combination of the above symptoms.

Conclusion

Most of the PVT cases are seen with combined local and systemic risk factors. CLD is the most common risk factor for acute PVT followed by local malignancy (pancreatic adenocarcinoma, HCC, cholangiocarcinoma, adenocarcinoma stomach). Myeloproliferative Neoplasms are an important risk factor for acute PVT independently and in combination with a local risk factor. Inflammatory abdominal conditions like acute pancreatitis, liver abscess and abdominal tuberculosis pose a risk for acute PVT. APLA, Factor V Leiden mutation and prothrombin gene mutation are other risk factors for the development of PVT and thus, imaging for PVT should be done in these patients if they present with symptoms pertaining to APVT. Conversely, in patients of acute PVT, screening for these risk factors should be done for proper treatment of the root cause. A thrombophilia screen may not be required in the patients of CLD with PVT as none of our cirrhotic patients had a positive thrombophilia screen. Symptoms that point towards APVT include upper abdominal pain which is severe initially and tends to fade in intensity over a week with fever and abdominal distension due to a high incidence of ascites.

Authorship

Conception and design of the study: Irfan Robbani

Acquisition of data: Gowhar Nazir, Minsha Hameed

Analysis and interpretation of data: Inzamam Wani, Zahoor Ahmed

Critical revising: Shadab Maqsood

Final approval: by IEC SKIMS

Ethical standards

Approval by Institutional Ethical Committee SKIMS

Informed Consent

Taken from each patient based on WMA DECLARATION OF HELSINKI – ETHICAL PRINCIPLES FOR MEDICAL RESEARCH INVOLVING HUMAN SUBJECTS.

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None

Conflict of interest

None

Financial Disclosure

None

Data Availability Statement

The authors confirm that the data supporting the findings of this study are available within the article. Raw data that support the findings of this study are available from the corresponding author, upon reasonable request.

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