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DETECTION OF PORTAL VEIN THROMBOSIS ON COMPUTED TOMOGRAPHY IN CIRRHOTIC PATIENTS: CORRELATION WITH SPLENOMEGALY AND ASCITES SEVERITY

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Abstract

Introduction:
Portal Vein Thrombosis is an obstruction in the PV trunk. Symptoms including fever, nausea, diarrhea, and temporary stomach pain appear less in acute PVT. A blood clot forming inside the main branches or trunk of the portal vein is the hallmark of portal vein thrombosis.

Objectives:
The main aim of this study is to detect the presence of portal vein thrombosis in cirrhotic patients using computed tomography scans and to assess its correlation with the severity of splenomegaly and ascites.

Methodology:
Cross sectional Analytical study was conducted at Social Security Hospital, Multan Road Lahore for four months, to evaluate imaging techniques for detecting portal vein thrombosis in cirrhotic patients. A total of 60 patients underwent scans using a computed tomography (128- slice Toshiba), with all observations recorded by radiologists. Data analysis was performed using SPSS 27.0 to determine distribution, frequency, percentage, using chi-square and spearman's tests to see the association of variables.

Results:
This study examined the association between portal vein thrombosis (PVT) and splenomegaly and ascites in 60 cirrhotic patients. Chi-Square ($p = 0.001$) and Spearman correlation ($rho = 0.267$, $p = 0.039$), both corroborated the findings, which showed a strong link between splenomegaly and PVT. In contrast, no significant connection was identified between ascites and PVT (Chi-Square, $p = 0.573$; t-test, $p = 0.137$). Overall, splenomegaly appears to be a prominent

radiographic indication of PVT in cirrhotic patients, although ascites severity lacks prognostic relevance.

Conclusion:

It is concluded that splenomegaly is correlated with portal vein thrombosis in cirrhotic patients. Ascites shows a non-significant result with PVT correlation. Our test analysis results also showed that splenomegaly and PVT are correlated.

INTRODUCTION

Portal Vein Thrombosis is an obstruction in the PV trunk. When symptoms including fever, nausea, diarrhea, and temporary stomach pain appear less than 60 days prior to a medical evaluation, it is deemed acute on the other hand, and partial PVT obstruction might also cause acute PVT to be asymptomatic. When hepatic-petal collateral veins develop, allowing blood to flow through the blocked trunk of the PV, the PVT may reanalyze or develop into a chronic condition. There may be significant hemorrhages, ascites, splenomegaly, and related hypersplenism as a result of portal hypertension. Acute and chronic PVT are diagnosed using a variety of imaging modalities, including color Doppler ultrasonography (US), CT, and MRI.[1]

When portal blood flow ceases, the liver loses around two-thirds of its blood supply. It is likely that the early activation of two compensatory mechanisms will complement the loss of the portal vein's contribution to liver blood circulation. The first process is arterial vasodilation of the hepatic artery, which is identical to what happens when the portal vein is clamped after liver resection. This arterial rescue is a type of vascular response found in all organs with both arterial and venous circulation that can preserve liver function during the acute stages of PVT. Venous rescue (VR) is the second compensatory strategy, which involves the quick creation of collaterals to get around the barrier. [2]

Patients with chronic portal vein thrombosis typically exhibit splenomegaly. Ascites is quite rare in PVT because it is caused by presinusoidal portal hypertension. [3]

A blood clot forming inside the main branches or trunk of the portal vein is the hallmark of portal vein thrombosis, a liver vascular disorder. PVT was highly prevalent in hospitalized patients, and the risk was considerably increased by CLD, HCC, and raised platelets. [4]

The prevalence, pathophysiology, diagnosis, impact on the natural history of cirrhosis and liver transplantation, and management of portal vein thrombosis (PVT) rise with the severity of liver disease: less than 1% in well-compensated cirrhosis, 7.4%-16% in advanced cirrhosis, and 5%-16% in patients undergoing liver transplantation. PVT often regresses rather than progresses uniformly as thrombus. [5]

Portal vein thrombosis (PVT) is a frequent thrombotic complication in patients with cirrhosis PVT is further associated with hyper dynamic circulation and gastrointestinal bleeding. This study aims to evaluate the clinical features of PVT in cirrhotic patients and identify the clinical and biochemical factors contributing to its development. [6]

It may develop without a liver disease, or it may occur in conjunction with liver cancer or cirrhosis. When choosing a treatment strategy, it's critical to distinguish between benign and malignant PVT. A significant factor in determining tumor stage, prognosis, and therapy choice is the existence of neoplastic thrombus. [7]

There are two main categories of portal vein thrombosis (PVT): acute (symptoms lasting less than 60 days, without portal cavernoma or portal hypertension) and chronic. According to these findings, the development of portal hypertension and the start of symptoms are unrelated to the severity of PVT, which is exclusively linked to the formation of portal cavernoma. [8]

About 1% of people in the general population have portal vein thrombosis, a consequence of decompensated cirrhosis that is more likely to happen in late-stage liver cirrhosis. According to studies, 5–20% of cirrhotic patients develop PVT. The prevalence of PVT that has been recorded varies. Based on the many locations where PVT occurs in the portal vein system, it can be divided into four groups. To ascertain whether there is an anatomic location

where PVT preferentially occurs, more investigation is necessary. [9]

Although liver cirrhosis is frequently the cause of PVT, other conditions such hereditary thrombocytopenia, cancer, intestinal disorders, or stomach infections can also cause it. [10]

Patients frequently arrive with fever, sepsis, lactic acidosis, gastrointestinal hemorrhage, nausea, vomiting, and abdominal pain. A physical examination may reveal splenomegaly and abdominal distension. [11]

The mesenteric, splenic, and portal veins make up the portal venous system, which is unique in that the hepatic sinusoids mediate rather than directly communicate with the systemic circulation. When a portal vessel is blocked, the organs it drains become congested, resulting in elevated venous pressures and the emergence of collateral circulation. [12]

Portal vein thrombosis, the second most common cause of portal hypertension in Western countries, is characterized by a thrombus that partially or completely obstructs the portal vein lumen. PVT may present in two clinical settings: asymptomatic individuals or symptomatic patients. In symptomatic cases, clinicians must perform imaging studies to confirm or rule out the diagnosis. When PVT is detected incidentally, the clinician should focus on identifying the underlying risk factors that may have contributed to its development. [13]

Cirrhosis was a severe liver disease that was common throughout the world and had high rates of morbidity and death. Around 2 million people die from liver disease each year, with cirrhosis, viral hepatitis, and hepatocellular carcinoma accounting for 1 million of these fatalities. Men account for more than 60% of liver disease-related deaths, with cirrhosis being the third most prevalent cause of death for people aged 45 to 64 and the eleventh most common cause of death overall. As liver cirrhosis worsens, serious side effects like ascites, hypersplenism, and portal hypertension develop. [14]

In patients with cirrhosis, portal vein thrombosis unrelated to solid malignancy is common; in people without cirrhosis, it is less common. It is crucial to diagnose and treat acute symptomatic portal vein thrombosis as soon as possible. [15]

Patients with cirrhosis frequently develop portal vein thrombosis, which exacerbates portal hypertension and sets off a chain of serious consequences. The purpose of this study was to create a nomogram for predicting PVT in cirrhotic patients based on an easy-to-use and efficient model. Serum albumin, D-dimer, portal vein diameter, splenectomy, and esophageal and gastric varices were among the predictors of PVT. Cryptogenic and metabolic-associated fatty liver disease-related cirrhosis are risk factors for PVT in cirrhosis. [16]

To assess, using the hepatic venous pressure gradient (HVPG) as a reference standard, the precision of spleen stiffness (SS) and liver stiffness (LS) evaluated by acoustic radiation force impulse imaging in the diagnosis of portal hypertension in patients with liver cirrhosis. [17]

In late liver cirrhosis, portal vein thrombosis is a potentially fatal consequence. Developing a diagnostic prediction model to enhance the detection of acute symptomatic portal vein thrombosis was the goal of this investigation. Acute and chronic PVT are the two types of PVT. There isn't a clear definition as of yet, though. In most cases, pictures of acute PVT revealed few compensatory esophageal varices, no spongy morphosis or collateral vasculature, and little splenomegaly. [18]

A typical side effect of liver cirrhosis is hypersplenism, which results in a reduction of peripheral blood cells. Splenomegaly, a variable combination of anemia, leukopenia, and/or thrombocytopenia, is a clinical syndrome known as hypersplenism. Due to splenic congestion brought on by portal hypertension, splenomegaly with pancytopenia develops. Thrombosis in the portal vein system, which includes the portal, splenic, and mesenteric veins, is known as PVST. [19]

Splenomegaly is the term used to describe the expansion of the spleen as determined by size or weight. Immunosurveillance and hematopoiesis are two important functions of spleen. Dead and irregular erythrocytes and their byproducts, opsonized platelets and white blood cells, and the elimination of microbes and antigens are among the spleen's primary duties. The maturation and storage of T and B lymphocytes take place in the spleen, which is also a secondary lymphoid organ. It is crucial for mature B-lymphocytes to produce immunoglobulin G (IgG) after interacting with T-lymphocytes. About one-third of the platelets in blood are kept in reserve in the

spleen. The spleen is normally located next to ribs 9 through 12 in the left upper quadrant of the abdominal cavity. [20]

One of the most frequent and dangerous side effects after splenectomy was PVT. PVT typically involves thrombosis of the spleen, superior mesenteric, inferior mesenteric, and left and right branches of the portal vein. Depending on the position and degree of the thrombus, PVT can produce fever, stomach pain, nausea, vomiting, and ileus, albeit it seldom affects the prognosis of young patients. Furthermore, ascites, decreased hepatic blood flow, and significant liver function impairment might result from severe PVT. [21]

Computed Tomography consists of a tube that travels around the patient, emits a narrow, fan-shaped x-ray beam, and several tiny electronic detectors count the x-rays that pass through the patient. The signals from the detectors are then sent to a computer, which reassembles the data into a two-dimensional image that shows a cross-section of the patient. As the patient passes through the scanner's aperture, the x-ray tube typically rotates 360 degrees around the body constantly. The patient can be moved continuously (helical scan mode) or in a series of tiny increments (axial scan mode). The radiographer can choose exactly how big the area to be scanned should be and how thick the tissue each image represents. [22]

Rationale of Study

Portal vein thrombosis (PVT) is a serious complication in cirrhotic patients, often worsening portal hypertension and clinical outcomes. CT is a reliable tool for detecting PVT and also demonstrates features like splenomegaly and ascites. However, limited studies have explored the correlation between PVT and the severity of these findings. This study aims to fill that gap, providing useful insights for better risk assessment and management of cirrhotic patients.

OBJECTIVES

To detect the presence of portal vein thrombosis in cirrhotic patients using computed tomography scans and to assess its correlation with the severity of splenomegaly and ascites.

METHODOLOGY

This study was designed as a cross-sectional analytical study conducted at Social Security Hospital, Multan Road, Lahore. The sample size of 60 patients was calculated using the formula $n = Z^2P(1-P)/d^2$, based on a prevalence rate of 4% [23], margin of error of 0.05, and a 95% confidence level ($Z = 1.96$). A convenient sampling technique was used. The total study duration was 4 months following synopsis approval.Inclusion criteria consisted of adults >18 years with radiologically confirmed cirrhosis, those undergoing contrast-enhanced CT abdomen, with adequate image quality, and complete imaging records. Exclusion criteria included patients with prior liver transplant or portal vein surgery, non-cirrhotic PVT, poor or incomplete CT images, and pregnancy or iodinated contrast allergy. A 128-slice MDCT scanner with a dual-head injector (3–4 mL/s) and 18–20G cannula was used. Non-ionic contrast (1.5–2.0 mL/kg) was administered, and scans were obtained in the arterial (25–30 s), portal venous (60–70 s), and delayed phases (3–5 min). Images were acquired in ≤1 mm slices and reconstructed in axial, coronal, and sagittal planes. Each scan was assessed for PVT, splenomegaly (>13 cm), ascites severity, and related cirrhotic findings. Data were analyzed using Chi-square and correlation tests, with $p < 0.05$ as significant.

Data is analyzed with statistical package for social sciences (SPSS) 27.0. A descriptive analysis will be performed to investigate the distribution of data. Collected data will be stored in Microsoft office. Data Analysis is done by using Chi-Square test and Spearman correlation.

RESULTS

This study consists of 60 patients having splenomegaly, Ascites and Portal vein thrombosis. The main purpose of this study was to correlate the relationship of PVT with splenomegaly and ascites. As a result, this study showed a significant relationship between splenomegaly and portal Vein thrombosis in cirrhotic patients. By using Chi-square testing ($p = 0.001$), Spearman correlation (rho = 0.267, $p = 0.039$), and independent samples t-test ($p = 0.03$), all showed consistent relationship that PVT increases with severity of splenomegaly on Computed Tomography in cirrhotic patients. On other hand there is no

significant relationship detected between ascites and portal vein thrombosis. Chi-square analysis (p = 0.573) and t-test results (p = 0.137) are not according to their significant value. Overall, in patients with cirrhosis, splenomegaly generally seems to be a major radiographic indicator associated with PVT, although ascites severity does not show a significant prognostic value.

Table 4.1 Table of Gender Distribution

		Frequency	Percent
Valid	Female	25	41.7
	Male	35	58.3
	Total	60	100.0

This table shows the gender distribution of the sample population, with females (25) accounting for 41.7% and males (35) for 58.3%, for a total of 60 participants. The cumulative percentage of both genders is 100%.

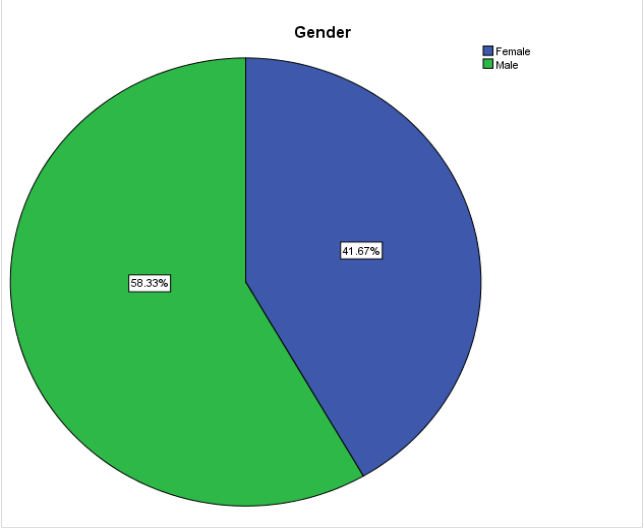
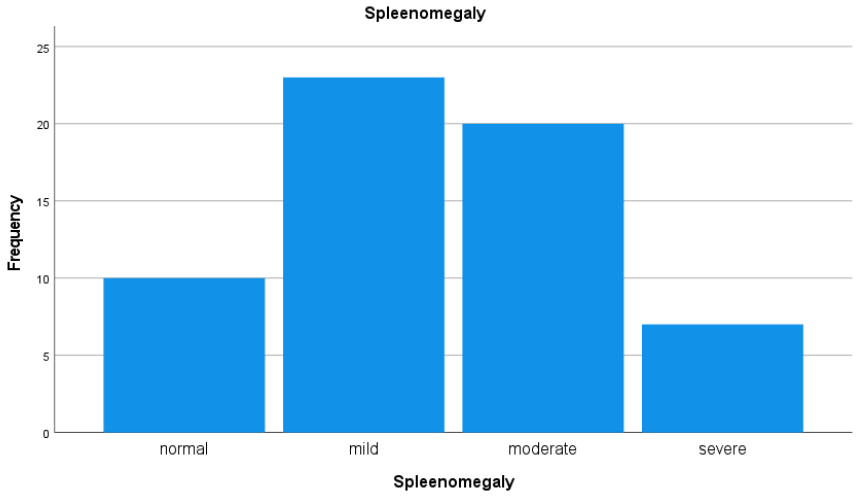


Figure # 4.1 this bar chart describes the gender breakdown of a sample population, indicating 41.67% females and 58.33% males. The male group has a slightly higher frequency than the Male group.

Table 4.2 Frequency Table of Splenomegaly

Splenomegaly	Frequency	Percent
normal	10	16.7
mild	23	38.3
moderate	20	33.3
severe	7	11.7

According to Table 4.2, among the 60 patients assessed, 10 exhibited normal spleen size, while 23 patients showed mild splenomegaly, 20 showed moderate splenomegaly, and 7 demonstrated severe splenomegaly.

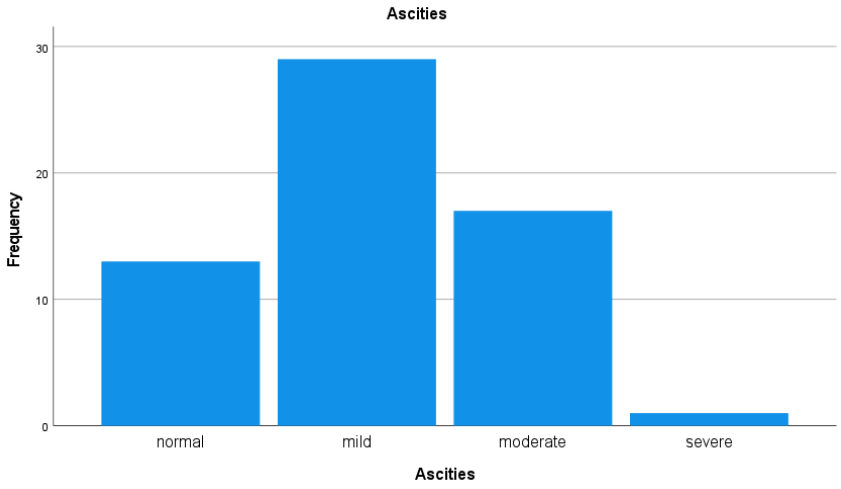


According to Figure 4.2, among the 60 patients assessed, 10 exhibited normal spleen size, while 23 patients showed mild splenomegaly, 20 showed moderate splenomegaly, and 7 demonstrated severe splenomegaly.

Table 4.3 Frequency Distribution of Ascites

Ascites	Frequency	Percent
normal	13	21.7
mild	29	48.3
moderate	17	28.3
severe	1	1.7

Regarding table 4.3, there were 60 patients noticed, 13 showed normal ascites, while 29 presented with mild ascites, 17 exhibited moderate ascites and only 1 showed severe ascites.



Regarding Figure 4.3, there were 60 patients noticed, 13 showed normal ascites, while 29 presented with mild ascites, 17 exhibited moderate ascites and only 1 showed severe ascites.

Table 4.4 Frequency Distribution of PVT

PVT	Frequency	Percent
absent	12	20.0
right	19	31.7
main	22	36.7
left	4	6.7
main and left	3	5.0

Table 4.4 illustrates the prevalence of portal vein thrombosis (PVT) in the study population. Out of a total of 60 patients, 12 had no evidence of PVT, 19 had thrombosis in the right portal vein, 22 had thrombosis in the main portal vein, 4 had thrombosis in the left portal vein, and 3 had involvement of both main and left portal veins.

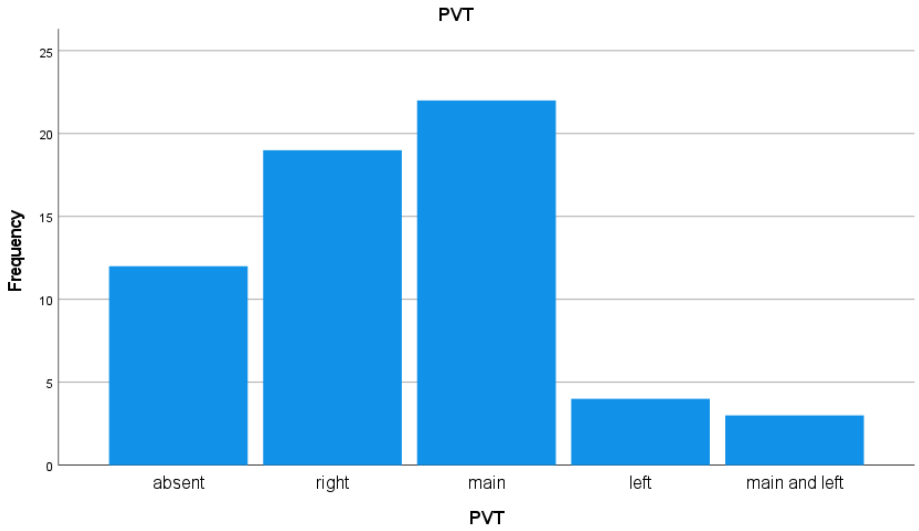


Figure 4.4 illustrates the prevalence of portal vein thrombosis (PVT) in the study population. Out of a total of 60 patients, 12 had no evidence of PVT, 19 had thrombosis in the right portal vein, 22 had thrombosis in the main portal vein, 4 had thrombosis in the left portal vein, and 3 had involvement of both main and left portal veins.

Table 4.5 PVT * Splenomegaly Crosstabulation

		Splenomegaly				Total
		normal	mild	moderate	severe	
PVT	absent	7	4	1	0	12
	right	0	9	6	4	19
	main	1	8	10	3	22
	left	0	2	2	0	4
	main and left	2	0	1	0	3
Total		10	23	20	7	60

Table 4.5 shows that out of the 12 patients without PVT, 7 had normal spleen size, 4 had mild splenomegaly, 1 had moderate splenomegaly, and none had severe splenomegaly. None of the 19

patients with Right PVT had a normal spleen size; nine had mild splenomegaly, six had moderate splenomegaly, and four had severe splenomegaly. One of the 22 patients with Main PVT had a normal spleen size, eight showed mild splenomegaly, ten showed moderate splenomegaly, and three showed severe splenomegaly. None of the four Left PVT patients had a normal spleen size; two had mild splenomegaly, two had moderate splenomegaly, and none had severe splenomegaly. Among the 3 patients with Left and Main PVT, 2 had normal spleen size, none exhibited mild splenomegaly, one showed moderate splenomegaly, and none presented with severe splenomegaly.

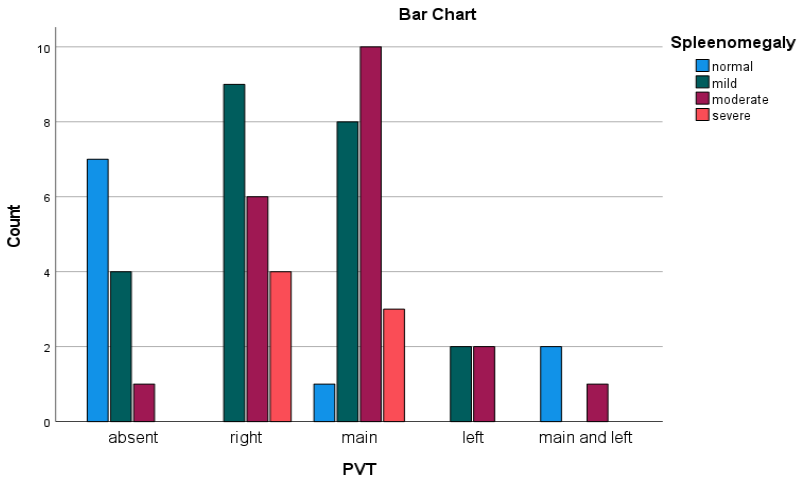


Figure 4.5: Crosstab of Splenomegaly and PVT

Table 4.6 Chi-Square Tests of Splenomegaly and PVT

Chi-Square Tests			
	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	31.865[a]	12	.001
Likelihood Ratio	33.457	12	.001
Linear-by-Linear Association	2.358	1	.125
N of Valid Cases	60		

16 cells (80.0%) have expected count less than 5.

The minimum expected count is .35.
Table 4.6 shows that Splenomegaly severity has a high and statistically significant connection with portal vein thrombosis (p = 0.001). This means that the severity of splenomegaly is determined by whether or not PVT is present. Although the link is not precisely linear, there is a strong overall association.

Table 4.7 Crosstab of Ascites and PVT

		Ascites				Total
		normal	mild	moderate	severe	
PVT	absent	1	6	5	0	12
	right	5	10	4	0	19
	main	7	8	6	1	22
	left	0	2	2	0	4
	main and left	0	3	0	0	3
Total		13	29	17	1	60

Table 4.7 shows that out of the 12 patients without PVT, only one had no ascites, 6 had mild ascites, 5 had moderate ascites, and none had severe ascites. Out of the 19 patients with Right PVT ,5 had a no ascites; ten had mild ascites, 4 had moderate ascites, and none with severe ascites. Seven of the 22 patients with Main PVT had a no ascites, eight showed mild ascites, six showed moderate ascites, and one showed severe ascites. None of the four Left PVT patients had a no ascites; two had mild ascites, two had moderate ascites, and none had severe ascites. Among the 3 patients with Left and Main PVT, none with no ascites, three exhibited mild ascites, none showed moderate ascites, and none presented with severe ascites.

Chi-Square Tests			
	Value	df	Asymptotic Significance (2-sided)
Pearson Chi-Square	10.485[a]	12	.573
Likelihood Ratio	12.770	12	.386
Linear-by-Linear Association	.079	1	.779
N of Valid Cases	60		

Table 4.8: Chi-Square Tests of Ascites and PVT

Table 4.8 shows that Ascites severity and portal vein thrombosis (PVT) did not significantly correlate, according to the Chi-square test (p = 0.573). Since a p-value must be less than 0.05 to establish statistical significance, this finding confirms that ascites severity does not substantially correlate with the existence of PVT. Furthermore, many cells had lower predicted numbers, thus the results should be evaluated cautiously.

Table 4.9 Spearman's Correlation Results
Correlations

			Splenomegaly	PVT
Spearman's rho	Splenomegaly	Correlation Coefficient	1.000	.267*
		Sig. (2-tailed)	.001	.039
		N	60	60

PVT	Correlation Coefficient	.267*	1.000
	Sig. (2-tailed)	.039	.001
	N	60	60

*. Correlation is significant at the 0.05 level (2-tailed).

Table 4.9 indicates the interpretation of Spearman's correlation results. This result shows that there is significant relationship between splenomegaly and PVT.As the Value of P is .003 which is less than .05. Patients with more splenomegaly are more likely to have a PVT.

DISCUSSION

This study examined portal vein thrombosis (PVT) on CT in cirrhotic patients and explored its relationship with splenomegaly and ascites. The results show that PVT is strongly associated with splenomegaly, suggesting a link with portal hypertension. However, no significant association was found between PVT splenomegaly than those without PVT. High portal vein pressure frequently causes splenomegaly, which results in splenic congestion and enlargement. Portal vein thrombosis (PVT) further increases resistance to portal blood flow, increasing portal hypertension and promoting more splenic enlargement. Therefore, finding splenomegaly on CT may serve as a valuable indirect diagnostic of PVT. The degree of ascites and PVT did not significantly correlate, according to this study. Ascites in cirrhosis is also influenced by variables other than portal vein obstruction, including low albumin levels, kidney sodium retention, systemic vasodilation, and fluid management.

Chen et al 2014 described that PVT was found in 24.7% of patients, mostly those with post-hepatitis B liver cirrhosis. The superior mesenteric vein and portal vein trunk were the primary sites of PVT. Different PVT locations may account for the different clinical manifestations. PVT was linked to reduced haemoglobin and BPC levels as well as splenic thickening. One possible risk factor for PVT is splenic thickness. [24]

Li X et al 2021 A post-operative CT scan on post-operative day (POD) was used to diagnose twenty-nine cases of PVT (19.5%). 5. Univariate analysis indicates three main risk factors linked with post-operative PVT: estimated splenic weight over 500 g with an OR of 8.72 95% CI (3.3–22.9), splenic vein diameter over 10 mm with an OR of 4.92 95% CI (2.1–11.8) and lymphoma with an OR of 7.39 (2.7–20.1).[25]

and the severity of ascites, indicating that different features of portal hypertension relate differently to venous thrombosis. Chi-square and Spearman correlation yielded the same conclusion: cirrhotic individuals with portal vein thrombosis have considerably

In conclusion, the study reveals that splenomegaly is highly linked with the existence of PVT in cirrhotic patients, strengthening its utility as a valid radiographic indication of portal hypertension and venous blockage. Ascites severity, on the other hand, did not significantly correlate with PVT, most likely because of its intricate and multiple etiology. These results shed light on the differential diagnostic utility of imaging characteristics linked to portal hypertension and emphasize the significance of spleen assessment during CT examination of cirrhotic patients. Therefore, according to previous research, all the studies showed that there is correlation between splenomegaly and portal vein thrombosis but there is independent relationship between ascites and splenomegaly. So, it is concluded that splenomegaly caused portal vein thrombosis.

CONCLUSION

This study utilized computed tomography to detect portal vein thrombosis in patients with cirrhosis and to evaluate its connection with splenomegaly and ascites severity. To assess these relationships, statistical tests such as the Chi-Square test, independent t-test, and Spearman correlation were used. The results demonstrated a substantial association between splenomegaly and PVT, suggesting that an enlarged spleen may represent hemodynamic abnormalities in portal hypertension that enhance the likelihood of thrombus formation. Conversely, there was no significant correlation found between the severity of ascites and PVT, suggesting that ascites by itself is not a good indicator of thrombotic risk. These results highlight the clinical significance of evaluating splenomegaly on CT scans in patients with cirrhosis, offering useful data for risk assessment, early PVT identification, and treatment planning.

Limitation

This study has significant drawbacks. The single-centre approach and rather small sample size may restrict how far the results can be applied. The cross-sectional design of the study hinders establishing a causal association between splenomegaly and portal vein thrombosis (PVT). Ascites severity was measured qualitatively rather than objectively, and any confounding factors, such as anticoagulant use or comorbidities, were not thoroughly controlled. While CT is useful for identifying PVT, readings may be impacted by inter-observer variability and tiny thrombi may have gone unnoticed. Lastly, the lack of longitudinal follow-up made it difficult to assess how PVT or splenomegaly changed over time.

Recommendations

Based on the findings of this study, several recommendations are proposed to improve the detection and management of portal vein thrombosis (PVT) in cirrhotic patients. Routine evaluation of splenomegaly on CT scans should be implemented in clinical practice, as it may serve as an early indicator of PVT, enabling timely intervention. Patients with significant splenic enlargement should be considered for closer monitoring and preventive strategies, such as anticoagulant therapy or scheduled follow-up imaging. Future studies should focus on larger, multicenter, and longitudinal designs to validate these associations and observe changes in PVT and splenomegaly over time. Incorporating quantitative assessment of ascites and standardizing CT measurement protocols will enhance diagnostic accuracy. Additionally, careful consideration of confounding factors, including anticoagulant use, coagulopathies, and other comorbidities, is recommended to improve patient risk stratification and guide individualized management plans.

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