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CT ANGIOGRAPHY PARAMETERS OF PORTAL HYPERTENSION IN LIVER CIRRHOSIS – CASE CONTROL STUDY

Radiology

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ABSTRACT

Background : 9/*9 Cirrhosis is defined as the histological development of regenerative nodules surrounded by fibrous bands in response to chronic liver injury, that leads to portal hypertension and end stage liver disease [1]. Almost all patients develop feature of portal hypertension related to cirrhosis. This study was conducted to evaluate the MDCT angiographic feature of portal hypertension for its early recognition & better diagnosis. **Materials And Methods:** Multiphasic MDCT angiographic was performed in 21 cases of liver cirrhosis and various vascular parameter were calculated for evaluation of portal hypertension. Diameter of portal vein, splenic vein, superior mesenteric vein, left renal vein, splenic artery, common hepatic artery and right hepatic artery were measured. Portosystemic collaterals and concomitant pathology were also noted. Study also includes the 20 subjects without any feature of liver disease. **Results:** All the parameters (Diameter of portal vein, splenic vein, superior mesenteric vein, left renal vein, splenic artery, common hepatic artery and right hepatic artery) showed higher mean value in cases as compared to that of control. The difference between two groups was also found to be significant ($p<0.001$). Presence of collaterals and ascites were also significantly higher in cases as compared to controls ($p<0.001$). Cases with ascites as compared to those without ascites, did not show significant difference statistically for any of the parameters ($p>0.05$). **Conclusion:** Preoperative evaluation of the hepatic vasculature and parenchymatous lesions is important in patients with chronic liver disease and portal hypertension where surgical intervention is planned. Several reports have described findings at CT in portal hypertension. To our knowledge, no report has described CT angiography in identifying detailed vascular abnormalities in portal hypertension.

KEYWORDS

INTRODUCTION:

Portal hypertension is a significant and potentially life-threatening complication of liver cirrhosis, characterized by elevated pressure within the portal venous system. This condition arises primarily due to architectural distortion and increased resistance to portal blood flow caused by hepatic fibrosis and regenerative nodules. Accurate and early diagnosis is crucial for timely intervention, reducing morbidity, and improving clinical outcomes.(1)

Among various imaging modalities, computed tomography (CT) angiography has emerged as a valuable non-invasive tool for assessing the hemodynamic changes associated with portal hypertension. It offers high-resolution visualization of both hepatic parenchyma and the vascular anatomy, enabling detailed evaluation of the portal venous system, collateral circulation, and associated complications such as varices, splenomegaly, and ascites. (2)

CT angiography provides a range of quantitative and qualitative parameters that aid in the diagnosis and grading of portal hypertension. These include measurements such as portal vein diameter, splenic vein diameter, portosystemic collateral formation, hepatic and splenic volume changes, and the presence of thrombosis or variceal bleeding. Understanding and interpreting these parameters can significantly contribute to the clinical management of patients with cirrhosis and guide decisions regarding endoscopic surveillance, pharmacological therapy, or surgical intervention. (3)

This section explores the key CT angiographic findings relevant to portal hypertension in liver cirrhosis, highlighting their diagnostic value and correlation with disease severity.

Materials And Methods:

A case control study was undertaken in the department of radiodiagnosis, Dr. Ram Manohar Lohia Institute of Medical Sciences (RMLIMS) Lucknow from September 2016 to January 2017. Our study comprised 21 patients (1 3 females, 8 males, aged 20-82 years, and mean 48 years) with chronic liver disease with portal hypertension. Twenty patients, with no evidence of liver disease or splenic vein thrombosis, served as controls (10 women, 10 men, aged 20-88 years, mean 48 years).

CECT Protocol:

After unenhanced scanning, CT images were obtained during the hepatic arterial dominant using a 30 seconds delay and the portal venous dominant phase using a 65 second delay after the initiation of IV injection of ~90 ml of non-ionic contrast media (iohexo /omnipaque 370) at the rate of 3ml/s using a power injector. A delayed phase was taken after minutes. During all phases CT images were obtained with a 7-mm collimation, pitch of 1.5 and 7mm reconstruction levels. Images were obtained from dome of diaphragm up to lower pole of right kidney during a single breath hold. All patients received oral contrast material (800 – 1000ml of diluted meglumine and diatrizoate sodium over 45-60 minutes before examination.)

All images obtained were independently analysed using a workstation (Extended Brilliance workspace, Phillips Medical Systems). All the measurements were taken in axial planes as follows:

Splenic artery and splenic vein at splenic hila. Portal vein anterior to IVC. Superior mesenteric vein within 5mm on confluence and left renal vein at its mid-point. Common hepatic artery and right hepatic artery within 1mm of their origins.

RESULTS:

Data was analysed using Statistical Package for Social Sciences (SPSS) version 21.0. Chi-square test was used to compare categorical outcomes. Parametric data was compared using independent samples 't'-test. The confidence level of the study was kept at 95%, hence a 'p' value less than 0.05 indicated a statistically significant association.

1. All the parameters (Diameter of portal vein, splenic vein, superior mesenteric vein, left renal vein, splenic artery, common hepatic artery and right hepatic artery) showed higher mean value in cases as compared to that of control. The difference between two groups was also found to be significant ($p<0.001$). [GRAPH 1 and TABLE 1]
2. No. of patients showing collaterals and ascites was significantly higher in cases as compared to controls ($p<0.001$). [GRAPH 2 AND TABLE 2]
3. Cases with ascites as compared to those without ascites, did not show significant difference statistically for any of the parameters ($p>0.05$). [GRAPH 3 AND TABLE 3]

Table 1: Between Group Comparison Of Different Parameters Being Studied

Parameters	Cases (n=21)		Controls (n=20)		t'	p'
	Mean	SD	Mean	SD		
Portal Vein	16.57	1.78	10.81	1.19	12.10	<0.001
Splenic Vein	10.57	1.62	6.55	1.24	8.92	<0.001

Superior Mesenteric Vein	13.89	2.16	9.84	1.42	7.07	<0.001
Splenic Artery	4.95	0.74	3.24	0.66	7.81	<0.001
Common Hepatic Artery	5.10	0.70	3.95	0.93	4.48	<0.001
Right Hepatic Artery	3.35	0.29	6.30	0.87	-14.62	<0.001
Left Renal Vein	9.45	1.91	2.48	0.63	15.54	<0.001

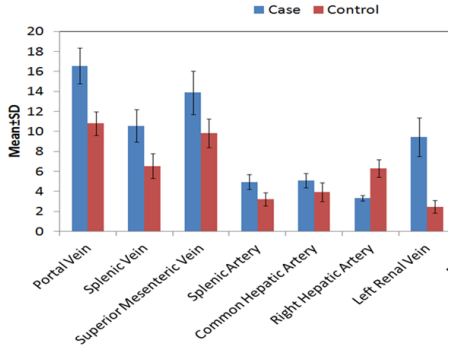
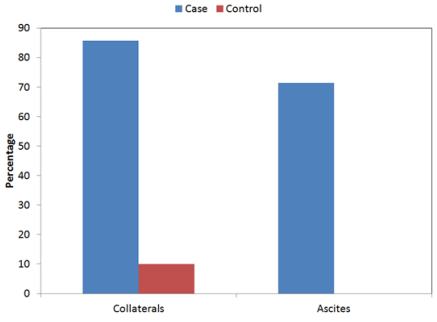


Table 2: Between group comparison of different categorical findings

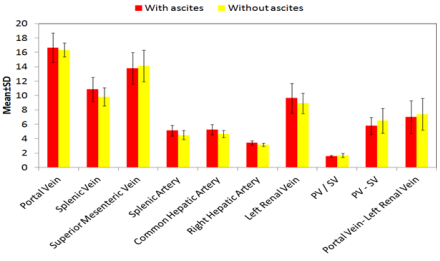
Variables	Cases (n=21)		Controls (n=20)		x^2	p'
	No.	%	No.	%		
Collaterals	18	85.7%	2	10.0	23.50	<0.001
Ascites	15	71.4	0	0	22.53	<0.001



Graph 2

Table 3: Comparison Of Different Parameters Between Cases Having Ascites And Those Not Having Ascites

Variable	Cases with ascites (n=16)		Cases without ascites (n=5)		Statistical significance	
	Mean	SD	Mean	SD	t'	p'
Portal Vein	16.66	2.05	16.35	0.95	0.35	0.729
Splenic Vein	10.87	1.68	9.83	1.26	1.35	0.193
Superior Mesenteric Vein	13.79	2.21	14.13	2.19	-0.33	0.749
Splenic Artery	5.13	0.72	4.52	0.65	1.79	0.089
Common Hepatic Artery	5.27	0.71	4.67	0.48	1.90	0.072
Right Hepatic Artery	3.43	0.29	3.17	0.23	1.97	0.064
Left Renal Vein	9.65	2.08	8.93	1.39	0.77	0.448



DISCUSSION:

Computed Tomography (CT) angiography has emerged as a non-invasive and reliable imaging modality for assessing portal hypertension, particularly in the context of liver cirrhosis.(4) This technique provides valuable insights into vascular morphology, hemodynamic alterations, and the development of portosystemic collateral pathways. In cirrhotic patients, portal hypertension typically results from both increased intrahepatic vascular resistance and augmented portal venous inflow, leading to significant structural and functional changes that can be effectively captured on CT angiography. (5)

Several CT angiographic parameters have been identified as indicative of portal hypertension. These include dilation of the portal vein (usually >13 mm), splenomegaly, enlargement of the splenic vein, and the presence of portosystemic collaterals such as oesophageal and gastric varices, paraumbilical veins, and splenorenal shunts. Moreover, reversal of portal vein flow or delayed contrast enhancement in the portal venous phase can further substantiate the diagnosis. (6) particularly important finding is the detection of varices and shunts, which are crucial for evaluating bleeding risk and for planning therapeutic interventions, including Trans jugular Intrahepatic Portosystemic Shunt (TIPS) placement or endoscopic management.

Quantitative assessment through CT angiography, such as the measurement of portal vein diameter and splenic index, has also been proposed to correlate with the severity of portal hypertension and liver dysfunction. Some studies suggest a strong correlation between these parameters and the hepatic venous pressure gradient (HVPG), the current gold standard for assessing portal hypertension. Although CT angiography does not provide a direct pressure measurement, its ability to visualize anatomical and pathological alterations compensates for this limitation in many clinical scenarios. (7)

Another significant advantage of CT angiography is its role in detecting complications associated with cirrhosis and portal hypertension, such as hepatocellular carcinoma, portal vein thrombosis, and mesenteric varices. This comprehensive diagnostic capability enhances clinical decision-making and long-term management strategies for cirrhotic patients.

However, there are limitations to consider. CT angiography involves exposure to ionizing radiation and iodinated contrast agents, which may not be suitable for patients with impaired renal function or allergy to contrast media. Additionally, while it offers excellent anatomical detail, functional assessment of liver perfusion and pressure gradients still necessitate complementary modalities like Doppler ultrasonography or invasive measurements.

CONCLUSION:

CT angiography serves as a powerful diagnostic tool in the evaluation of portal hypertension in cirrhotic patients. Its ability to delineate vascular abnormalities, detect collateral formation, and assess hepatic and splenic morphologies renders it indispensable in the diagnostic algorithm. Further studies integrating CT parameters with clinical scoring systems may enhance its predictive value for patient outcomes and therapeutic guidance.

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