Original Article

# CECT Imaging Spectrum of Portal Venous Hypertension: A Case Series in Tertiary Care Centre

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## Abstract

**Background:** Portal hypertension is an altered hemodynamic condition & common complication of chronic liver disease & other portal venous system disorders. Contrast-Enhanced Computed Tomography (CECT) plays a vital role in identifying liver morphology, including cirrhosis, suspected non-cirrhotic etiology, complications & associated collateral pathways. The objective is to evaluate the CECT imaging spectrum of portal venous hypertension & associated complications in patients presenting to the radiology department of Government Mohan Kumarmanagalam Medical College, Salem, Tamil Nadu. **Material and Methods:** This retrospective case series of patients suspected & diagnosed with portal venous hypertension who underwent CECT abdomen from May 2024 to November 2024. Imaging findings, including portal vein, hepatic vein & IVC luminal patency; luminal diameter; collateral pathways; splenomegaly; and complications, were analyzed. **Results:** The most common underlying cause was liver cirrhosis (70%), followed by portal vein thrombosis (20%) and Budd–Chiari Syndrome (10%). Splenomegaly was observed in 90% of patients, ascites in 70%, and portosystemic collateral formation in 80%. Gastroesophageal varices were the most frequently observed collateral pathway. **Conclusion:** CECT demonstrated multiple manifestations of portal hypertension, especially portal, hepatic vein thrombosis, portal gastro-entero-colopathy, splenic infarcts, Porto-systemic collaterals & varices.

**Keywords:** CECT, portal hypertension, portal thrombosis, budd-chiari syndrome.

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## Introduction

Portal hypertension is defined as increased pressure in the portal venous system, typically due to increased resistance to portal blood flow by liver fibrosis / venous congestion secondary to thrombosis or flow obstruction, such as a web. The most common cause is cirrhotic liver disease; other extrahepatic causes are portal vein thrombosis & Budd-Chiari syndrome. Complications include massive ascites, esophageal varices, portal gastro-entero-colopathy, and splenic infarcts. Early detection of these complications is essential for appropriate clinical management. CECT allows assessment of liver morphology, portal and hepatic venous anatomy, identification of portosystemic collaterals, and detection of complications.

AIM: To describe the CECT imaging spectrum of portal venous hypertension in patients presenting to the radiology department in a tertiary care institute located in South India.

## Materials and Methods

**Study design:** Retrospective case series

**Study location:** Department of Radiology, Government Mohan Kumarmanagalam Medical College, Salem, Tamil Nadu.

**Study duration:** May 2024- November 2024

**Inclusion criteria:** Patients with suspicion & diagnosis of portal hypertension on Ultrasonography & underwent CECT abdomen.

**Exclusion criteria:** Incomplete imaging data & Postsurgical portal systemic alterations.

**Imaging protocol:** Abdomen section from lower chest up to the pelvis scanned in a Multidetector 16-slice CT scanner in non-contrast, arterial (10-20seconds), portal venous (40-50seconds) & delayed phase (120-130 seconds). Images were reconstructed in axial, coronal & sagittal planes. CT Images were systematically evaluated for

1. Liver morphology (Hilar peri-portal space widening, size, Surface nodularity, caudate lobe hypertrophy, regenerative nodules)
2. Portal, Splenic, Hepatic vein & IVC (Diameter, lumen patency for presence of thrombosis)
3. Spleen (Size, parenchymal infarcts)
4. Ascites,
5. Porto-systemic collaterals (Esophageal varices at the Gastro-esophageal junction, Para-umbilical vein at the anterior abdominal wall, spleno-renal shunt between the splenic-

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renal vein & gastric varices at the stomach fundus).

**CASE 1**

30-year-old male with complaints of hematochezia and abdominal distension.

CECT revealed Cirrhotic liver morphology with ascites, esophageal and rectal varices. Chronic calcific pancreatitis. Gastric, small bowel & colonic mural edema with water target sign. Diagnosis - Portal hypertension due to liver cirrhosis. Porto-systemic shunts with esophageal & rectal varices, portal gastro-entero-colopathy.

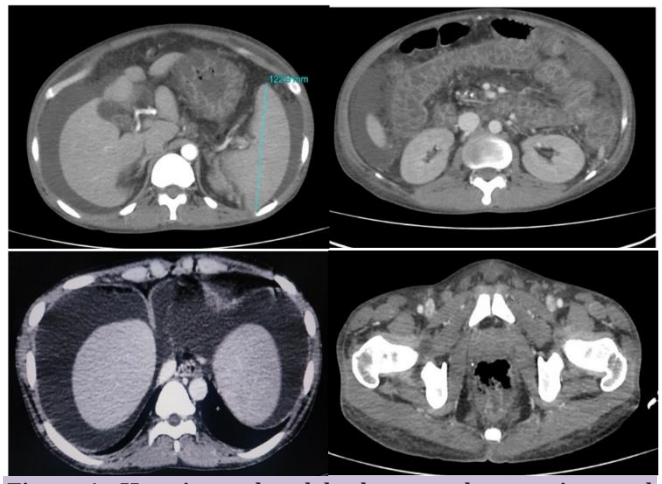

**Figure 1: Hepatic caudate lobe hypertrophy, gastric mural edema.**
**Figure 2: Pancreatic calcifications, small bowel & colonic mural edema.**
**Figure 3: Esophageal varices with ascites**
**Figure 4: Rectal varices**

**CASE 2**

75-year-old female with abdominal pain, no history of alcohol abuse

CECT findings were dilated portal vein with luminal filling defects in portal phase images & multiple hilar collaterals, splenomegaly & mild ascites.

Diagnosis – Portal hypertension due to portal & splenic thrombosis with multiple hilar collaterals.

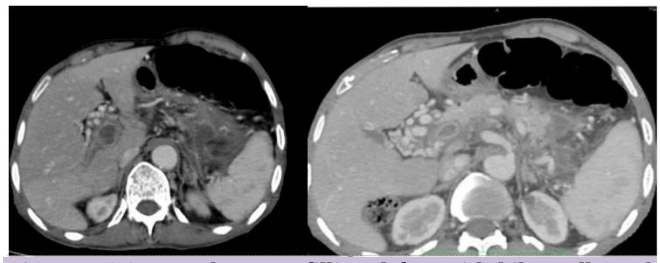

**Figure 5 &6: Portal venous filling defect with hilar collateral formation.**

**CASE 3**

27-year-old female with sudden onset of abdominal pain & yellowish discoloration of urine. Lab investigation revealed elevated liver enzymes.

CECT findings: Hepatomegaly, flip-flop pattern parenchymal enhancement with thrombosis of hepatic veins & IVC, Splenic infarct.

Diagnosis- Budd-Chiari syndrome with secondary portal hypertension.

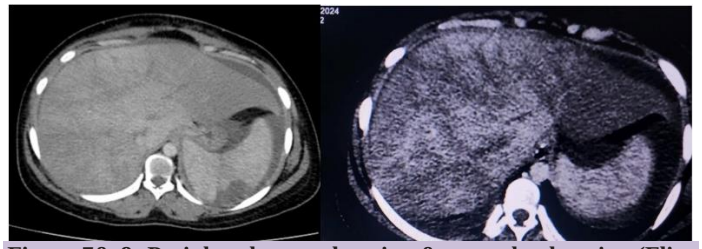

**Figure 7& 8: Peripheral non enhancing & central enhancing (Flip-flop) pattern of liver parenchyma. Non enhancing wedge shaped infarct in spleen. Nonopacified IVC in portal venous phase**

## Results

After exclusion, 13 cases remained; 10 were included.

| | |
|---|---|
| Total cases | 10 |
| Mean age | 49 |
| Male | 7 |
| Female | 3 |

Most cases presented with abdominal distension, hematemesis & weakness. Less frequently, sudden onset of abdominal pain & hematochezia.

Etiology: Cirrhotic liver disease was the most common etiology.

| Etiology | Number of patients |
|---|---|
| Cirrhosis | 7 |
| Budd Chiari syndrome | 1 |
| Portal vein thrombosis | 2 |

Imaging findings: Splenomegaly was observed in 9 of the 10 patients. Ascites was detected in 7 patients, with moderate volume in all. Portal vein dilation (>13 mm) was noted in the majority of patients. Two patients demonstrated features consistent with portal vein thrombosis. Porto-systemic collateral pathways were observed in eight patients. The most common collateral pathways identified were the Gastro-Esophageal collaterals. These were the most frequently observed collaterals and are associated with the development of esophageal varices. Lieno-renal shunts were seen in several patients with advanced portal hypertension. Paraumbilical vein recanalization was observed in a few patients.

## Discussion

Portal Hypertension is a clinically significant hemodynamic condition resulting from increased resistance to blood flow within the portal venous system. Cirrhosis causes architectural distortion of hepatic parenchyma, formation of regenerative nodules, and fibrosis, all of which contribute to increased intrahepatic vascular resistance. Previous studies have demonstrated that cirrhosis accounts for nearly 80–90% of portal hypertension cases in clinical practice.[1,2] In our case series, cirrhosis was also the most frequently identified

underlying cause, accounting for the majority of patients included in the study.

In addition to cirrhosis, extrahepatic causes such as Portal vein thrombosis and Budd–Chiari syndrome can also lead to portal hypertension. Portal vein thrombosis results in obstruction of the portal venous flow, leading to increased portal pressure and the development of collateral circulation. Similarly, Budd–Chiari syndrome results from obstruction of hepatic venous outflow, causing hepatic congestion and secondary portal hypertension.[3] In the present case series, a small proportion of patients demonstrated imaging findings consistent with these extrahepatic causes.

Imaging plays a vital role in the diagnosis and evaluation of portal hypertension. Ultrasonography with Doppler is commonly used as an initial screening modality; however, contrast-enhanced computed tomography (CECT) provides superior anatomical detail and allows comprehensive evaluation of the portal venous system and surrounding structures. CECT enables accurate visualization of the liver parenchyma, portal vein, hepatic veins, and collateral pathways, making it an important imaging modality in patients with suspected portal hypertension.[4]

Several characteristic imaging findings of portal hypertension can be identified on CECT. One of the most common findings is splenic enlargement.[5] Splenomegaly is considered one of the earliest and most consistent imaging features of portal hypertension and was observed in the majority of patients in our study.

Ascites develops due to a combination of increased hydrostatic pressure within the portal venous system and alterations in renal sodium and water retention mechanisms. Imaging plays an important role in detecting and quantifying ascites and in identifying associated complications such as spontaneous bacterial peritonitis or hepatic hydrothorax.[6] In the present study, ascites was detected in a significant proportion of patients, consistent with previous reports.

The development of portosystemic collateral circulation is a hallmark of portal hypertension. One of the most clinically significant collateral pathways involves the Gastro-Esophageal region, which may result in the formation of esophageal varices. These varices are prone to rupture and can lead to life-threatening upper gastrointestinal bleeding.[7] In our study, gastroesophageal collaterals represented the most common collateral pathway observed on CT imaging.

Other collateral pathways include splenorenal shunts, paraumbilical veins, and retroperitoneal collaterals. Splenorenal shunts occur between the splenic vein and the left renal vein and are frequently seen in advanced portal hypertension. Recanalization of the paraumbilical vein may lead to dilated abdominal wall veins, clinically manifesting as caput medusae.[8]

CECT is also highly useful for identifying vascular complications associated with portal hypertension. For example, in patients with Portal Vein Thrombosis, CT imaging may demonstrate filling defects within the portal vein and cavernous transformation in chronic cases. Similarly, in Budd–Chiari Syndrome, CT findings may include hepatic vein occlusion, caudate lobe hypertrophy, and heterogeneous hepatic enhancement patterns.[9]

The findings observed in our case series are consistent with previously published studies that highlight the importance of CT imaging in evaluating portal hypertension and its complications. By providing detailed anatomical information, CECT allows clinicians to identify the underlying etiology, evaluate disease severity, and plan appropriate therapeutic interventions.[10]

Despite the valuable insights obtained from this series, certain limitations should be acknowledged. The sample size was relatively small, and the study was conducted retrospectively at a single institution.

## Conclusion

Contrast-enhanced computed tomography plays a pivotal role in evaluating portal venous hypertension. CECT is particularly useful in identifying the underlying cause of portal hypertension and detecting complications such as splenic infarcts, esophageal, rectal varices & portal gastro-entero-colopathy. Early detection through imaging enables timely clinical management and may help reduce morbidity associated with portal hypertension.

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### Conflicts of interest

There are no conflicts of interest.

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