

Role of Multidetector Computed Tomography (MDCT) in Evaluation of Cirrhosis of Liver

Dr. Ashish Sachdeva¹, Dr. Mona D Shastri², Dr. Nipa Patidar³, Dr. Dhagash Patel⁴, Dr. Avani Bhatt⁵

¹Third year Resident, Department of Radiodiagnosis, SMIMER Hospital, Umarwada, Surat, Gujarat-395101

²Head of Department, Department of Radiodiagnosis, SMIMER Hospital, Umarwada, Surat, Gujarat-395101

³Assistant Professor, Department of Radiodiagnosis, SMIMER Hospital, Umarwada, Surat, Gujarat-395101

⁴Senior Resident, Department of Radiodiagnosis, SMIMER Hospital, Umarwada, Surat, Gujarat-395101

⁵Senior Resident, Department of Radiodiagnosis, SMIMER Hospital, Umarwada, Surat, Gujarat-395101

ABSTRACT : 50 known cases of Cirrhosis were evaluated by Multidetector Computed Tomography (MDCT) to detect various intrahepatic and extrahepatic manifestations of Cirrhosis. Four scans of hepatic parenchyma were obtained after the administration of contrast material. The Late arterial, Portal venous, Nephrogenic and Delayed phases were acquired with a 35-40 sec, 60 sec, 100 sec and 5 min delay time respectively, after contrast material injection. Hepatic surface irregularity, Ascites & Splenomegaly are commonly seen and well detected on MDCT. Collateral formation and portal vein thrombosis, Regenerative and Dysplastic nodules are well diagnosed on MDCT. Hepatocellular Carcinoma is well detected and confirmed on MDCT study. MDCT Scan is highly Sensitive and Specific for diagnosis of complications of cirrhosis like Hepatocellular carcinoma, Portal vein- hepatic vein & splenic vein thrombosis, Collateral formation, Retroperitoneal edema and Bowel wall thickening.

Keywords: Cirrhosis, MDCT, Regenerative nodule, Dysplastic nodule, Hepatocellular carcinoma

1. Introduction

Cirrhosis of Liver is the end stage of a complex process- resulting from hepatocyte injury and the response of liver, that leads to partial regeneration and fibrosis of the liver.

Cirrhosis poses a difficult challenge for management, while the disease's prevention, detection and therapy reduces major health costs. Diagnostic imaging offers diverse modalities for use in the noninvasive evaluation of liver, as well as in interventional techniques; the latter may be used to treat such complications such as portal hypertension and neoplasia.

Regardless of etiology, gross morphological changes of cirrhosis are recognized by a variety of imaging techniques. Enlargement of the left lobe and caudate lobe, believed to be the result of lobar-relative regeneration rather than fibrosis, secondary to an accident of vascular supply, is recognized by a cross-sectional technique, such as computed tomography (CT) scanning.

Computed Tomography

CT scanning is useful for demonstrating the morphologic evidence of cirrhosis within the liver and in showing mesenteric and Gastrointestinal tract abnormalities, as well as the development of collateral vessels in portal hypertension.

2. Aims and Objectives

To evaluate the role of Multidetector Computed Tomography (MDCT) in detecting the intrahepatic and extrahepatic manifestations of Cirrhosis of liver.

- To detect intrahepatic manifestations of Cirrhosis like Regenerative and Dysplastic Nodules.
- To detect changes of portal hypertension and portal vein thrombosis.
- To detect extrahepatic manifestations like splenomegaly, ascites, collateral vessel formation, gall bladder wall thickening and retroperitoneal edema.
- To detect hepatocellular carcinoma.

3. Materials and Methods

- The study was carried out on 50 known cases of Cirrhosis, without any gender bias and included patients of all age groups, referred for CECT Abdomen between January 2018 to June 2018.
- All examinations were performed with a multislice spiral CT scanner. Images were acquired using the following parameters: slice collimation- 2.5 mm; slice thickness - 3.0 mm; table feed, 10.8 mm/sec; mAs- 165; kVp- 120. Four scans of hepatic parenchyma were obtained after the administration of contrast material.
- The Late arterial, Portal venous, Nephrogenic and Delayed phases were acquired with a 35-40 sec, 60 sec, 100 sec and 5 min delay time respectively, after contrast material injection.

Inclusion Criteria

All known cases of Cirrhosis from OPD and Indoor facility of Department of Medicine, referred to the Department of RadioDiagnosis, Tertiary Care Hospital, South Gujarat for CECT Abdomen, during the time period were included in the study.

Exclusion Criteria

- Patient not willing to provide oral/written consent.

Volume 7 Issue 11, November 2018

www.ijsr.net

Licensed Under Creative Commons Attribution CC BY

- Allergy to intravenous contrast agent
- Acute Renal Failure

4. Etiology & Pathology of Cirrhosis of Liver

Hepatic cirrhosis is the clinical and pathologic result of a multifactorial chronic liver injury characterized by extensive fibrosis and nodular regeneration replacing the normal liver parenchyma. It is well known that cirrhosis is associated with a markedly increased risk of hepatocellular carcinoma (HCC), the sixth most common malignancy worldwide and third most common cause of cancer related death. The detection of hepatic malignancy in cirrhotic patients is a diagnostic challenge due to distortion of the hepatic architecture.

The distribution of underlying etiology will vary regionally, with viral hepatitis being much higher in the developing world, especially Asia. Causes for cirrhosis of liver are as follows⁴:

- Alcohol: 60-70%
- Viral hepatitis: 10%
- Cryptogenic / Nonalcoholic steatohepatitis (NASH): 10-15%
- Biliary disease, e.g. Primary Sclerosing Cholangitis (PSC)
- Metabolic disease, e.g. Hereditary Hemochromatosis, Wilson's disease, alpha-1-antitrypsin deficiency
- Autoimmune hepatitis
- Vascular disease, e.g. congestive hepatopathy (right heart failure), Budd-Chiari syndrome, hepatic veno-occlusive disease
- Cystic fibrosis

Common pathologic features of cirrhosis include hepatic fibrosis, nodular distortion of hepatic architecture, and perfusion abnormalities. The fibrotic changes appear as bridging bands or focal confluent fibrosis. Bridging bands usually have variable thickness and may mimic a tumor capsule due to delayed contrast enhancement. Focal confluent fibrosis is defined as a peripheral, wedge shaped, hypoattenuated area on unenhanced and portal venous phase CT. On delayed phase, enhancement of the lesion may occur. Overlying capsular retraction with volume loss in areas of focal confluent fibrosis is an important feature to differentiate this entity from malignant conditions.

Morphological changes of liver vary with the stage of cirrhosis. More than 60% of patients with early cirrhosis have hepatomegaly. Additional early detectable morphologic changes of the liver include widening of the porta hepatis, enlargement of the interlobar fissure, and expansion of pericholecystic space. During advanced stages shrinkage of the liver is seen, especially in alcohol induced cirrhosis. The medial segment (IV) of the left lobe shrinks with concomitant hypertrophy of the lateral segments (II, III), giving a "tongue-like" appearance. These changes lead to a nodular contour and heterogeneity of the liver, which is classically associated with cirrhosis.

Hepatic steatosis is a nonspecific reversible response of hepatocytes to chronic injury, commonly seen in alcohol induced cirrhosis. A diffuse uniform fatty infiltration

involving the entire liver is the most common pattern. When hepatosteatosis occurs, the average liver attenuation is at least 10 Hounsfield Units (HU) less than the splenic parenchyma on unenhanced CT. The identification of normal course of vascular structures in areas of fatty infiltration is crucial to differentiate this abnormality from hepatic tumors.

Evolving hepatic nodular lesions are another important feature of cirrhosis. In attempt to standardize the terminology, an international working party has suggested terms and definitions of nodular lesions in cirrhotic patients. These are categorized as regenerative nodules, dysplastic nodules and HCC.

A Regenerative Nodule (RN) is a well-defined area of liver parenchyma that has enlarged in response to necrosis and altered circulation. Based on gross morphologic features, the nodular regeneration can be classified as micronodular (<3mm in diameter) or macronodular (>3 mm in diameter). Unless a regenerative nodule contains iron, it is rarely seen on a noncontrast CT. If iron deposition is present (siderotic nodule), the nodule appears hyperdense to the surrounding liver on a non-contrast CT. Micronodular changes are rarely identified on CT, despite being present in all cirrhotic livers. Regenerative nodules do not enhance in the arterial phase and are isodense to the remaining parenchyma on the venous phase, making them indistinguishable from the hepatic background. The accuracy of non-contrast CT in detecting a RN is approximately 25%. A combination of micro- and macronodular regeneration is the most common morphologic presentation seen in cirrhotic patients.

A Dysplastic Nodule (DN) is defined as a nodular region of dysplastic hepatocytes without histologic features of malignancy. DNs commonly measure 5-10 mm and most of them are undetectable by CT since, even after the administration of contrast, the majority is isoattenuating. Dysplastic nodules can be further characterized as low grade or high grade, according to the degree of dysplasia. Tumor angiogenesis appears to be a mandatory step in the evolution of dysplastic nodules to HCC. During this process, there is a progressive increase in the arterial supply and a concomitant decrease in the portal venous supply to these lesions. The major shift in angiogenesis typically occurs during the transition from low-grade DNs to high-grade DNs. New vessels composed of non-triadial arteries become dominant and the absence of portal tracts is noted. The increasingly dedifferentiated nodule appears more markedly enhanced on post-contrast early arterial phase image, occasionally mimicking an HCC. Several reports have described the detectability of dysplastic nodules on dynamic CT scans.

Extrahepatic abnormalities associated with cirrhosis include portal hypertension, ascites, splenomegaly, diffuse intra and retroperitoneal edema, small bowel, and gallbladder wall thickening.

A variety of morphologic alterations are seen in cirrhotic patients due to portal hypertension. Portal hypertension is defined as a portal venous pressure greater than 5—100mm Hg. Portosystemic collaterals develop spontaneously, as blood flow is shunted away from the liver to low pressure

systemic vessels (hepatofugal flow). Gastrointestinal variceal bleeding is the most common clinical presentation in patients with altered flow dynamics.

Varices appear as well-defined tubular or serpentine homogeneous structures. On unenhanced CT, varices may mimic adenopathy, masses, or nonopacified bowel loops. The administration of intravenous contrast is vital to delineate dilated venous structures.

Portal vein thrombosis may occur in patients with cirrhosis and portal hypertension. After administration of contrast, the portal vein shows a central hypodensity, corresponding to the intraluminal thrombus. In this situation, the hepatic arterial flow to the liver is increased, developing scattered peripheral transient high attenuation areas known as transient hepatic attenuation differences.

In subacute and chronic portal thrombosis, a cavernous transformation of the portal vein may manifest as multiple tubular collaterals in the porta hepatis. When the portal vein is occupied by tumor thrombus, intraluminal enhancement may be seen.

Portal hypertension is considered the most common cause of splenomegaly. Foci of hemosiderin deposition in the spleen are seen in about 9%-12% of patients with portal hypertension. These foci are called Gamma-Gandy bodies, and their CT imaging pattern varies from hypo- to hyperdense spots, depending on the presence of secondary calcium deposition.

Mesenteric edema is defined as increased attenuation of the adipose tissue that surrounds the mesenteric vessels or their branches. Mesenteric edema in patients with cirrhosis has a multifactorial pathogenesis. Inflammation, hemorrhage, neoplastic infiltration, and hypoproteinemia due to hepatic insufficiency are the most frequent conditions identified. The frequency of mesenteric edema in patients with cirrhosis is 86%, and it is usually associated with omental and retroperitoneal edema. Most of the patients with mesenteric, omental, or retroperitoneal edema demonstrate a patchy, infiltrative pattern of fat stranding. The presence of retroperitoneal edema without mesenteric edema is uncommon. In some instances, focal edema may simulate a soft tissue mass. The severity of mesenteric edema parallels other manifestations of fluid overload in patients with cirrhosis such as subcutaneous edema, pleural effusion, and ascites.

Gastrointestinal wall thickening occurs in cirrhotic patients, usually as a result of submucosal edema. The jejunum and the ascending colon are the most common sites of involvement. In almost all cases, the pattern of wall thickening is concentric with homogeneous enhancement after administration of intravenous contrast. Thickening of the colonic haustra have been described in patients with cirrhosis.

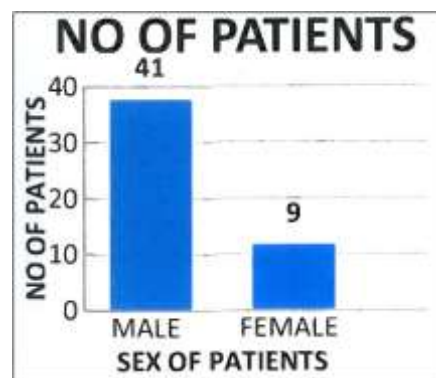
Hepatic cirrhosis may cause diffuse gallbladder wall thickening. The exact pathophysiologic mechanism leading to edema of the gallbladder wall is uncertain, but it is likely due to elevated portal venous pressure, decreased

intravascular osmotic pressure, hypoproteinemia, or a combination of these factors. Recognition of this abnormality is essential to avoid erroneous interpretations and unnecessary cholecystectomy. Ascites is defined as the pathologic accumulation of fluid in the peritoneal cavity. It is the most common complication of cirrhosis. Within 10 years of diagnosis of compensated cirrhosis, about 50% of patients will have developed ascites. The development of ascites is the final consequence of anatomic and pathophysiologic abnormalities occurring in patients with cirrhosis.

5. Observation & Discussion

Table 1: Sex distribution of cases of Cirrhosis of liver

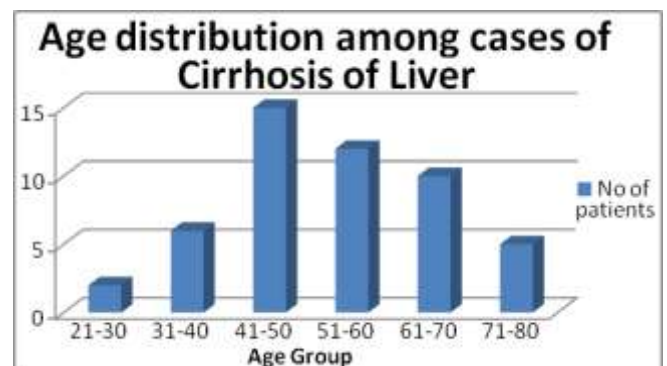
Sex	No. of Patients
Male	41(82%)
Female	9(18%)



Cirrhosis is more common in Males.

Table 2: Age distribution of cases of Cirrhosis of Liver

Age	No. of patients
21-30	2
31-40	6
41-50	15
51-60	12
61-70	10
71-80	5



Cirrhosis is most common in 41-50 year age group.

Table 3: Clinical complaints among cases of Cirrhosis of liver

Clinical Complaints	No. of patients
Abdominal distension	29(58%)
Abdominal pain	13(26%)
Weight loss and Anorexia	4(8%)
Fatigue and Weakness	2(4%)
Bleeding varices	2(4%)

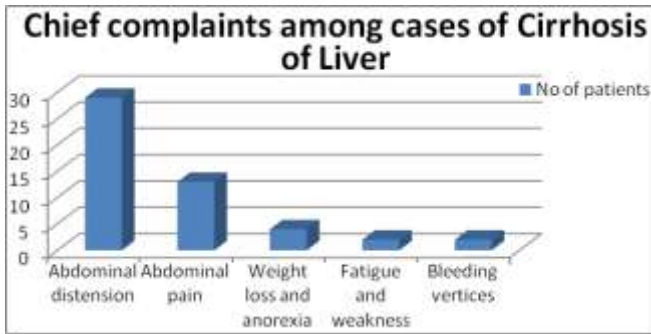
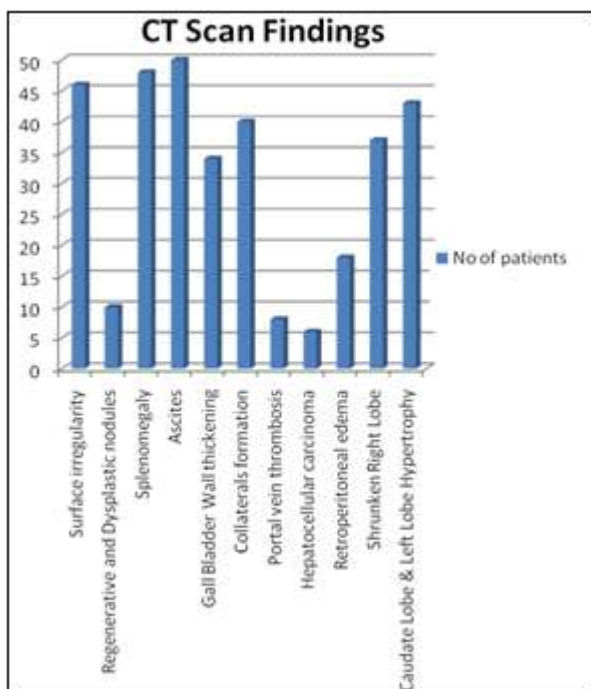


Table 4: MDCT Findings in Cirrhosis of Liver

No	MDCT findings	No. of patients
1	Surface irregularity	46(92%)
2	Regenerative and Dysplastic Nodules	10(20%)
3	Splenomegaly	48(96%)
4	Ascites	50(100%)
5	Gall bladder wall thickening	34(68%)
6	Collateral formation	40(80%)
7	Portal vein thrombosis	8(16%)
8	Hepatocellular carcinoma	6(12%)
9	Retroperitoneal edema	18(36%)
10	Shrunken Right Lobe	37(74%)
11	Caudate & Left Lobe Hypertrophy	43(86%)



- Surface irregularity, ascites & splenomegaly are commonly seen and well detected on MDCT.
- Collaterals formations and portal vein thrombosis, regenerative and dysplastic nodules are also well diagnosed on CT.
- Hepatocellular Carcinoma is well detected and confirmed on CT study.

Table 5: Comparative evaluation of MDCT in detection of Dysplastic nodules in patients with Cirrhosis of liver

Patients	Study of JH LIM et al	Observation of My Study
Total no of patients	76	50
Patients with Dysplastic nodules	8(10%)	10(20%)

In our study, Regenerative and Dysplastic nodules are detected in 10 patients.

Table 6: Comparative evaluation of MDCT in detection of Hepatocellular Carcinoma in patients with Cirrhosis of liver

Patients	Study of Department of Medicine, Indiana University School[4]	Observation of Our Study
Total no of patients	287	50
Patients detected with HCC	27(9%)	6 (10%)

In our study, 10% (6) patients are detected with hepatocellular carcinoma

Table 7: Comparative evaluation of MDCT in detection of Collateral formation in patients with Cirrhosis of liver

Patients	Study of QIN WU [12] et al	Observation of our study
Total no of patients	700	50
No of patients detected with Collateral formation	118(16%)	40(80%)

In our study, 40 patients are detected with collateral formation.

Table 8: Comparative evaluation of MDCT in detection of Extrahepatic manifestations in patients with Cirrhosis of liver

Patients	Study of SAN ANTONIO[13]	Observation of our study
Total no of patients	80	50
No of patients with Bowel wall thickening	39 (48%)	22 (44%)
No of patients with GB wall thickening	52 (65%)	34 (68%)
No of patients with Retroperitoneal edema	40 (50%)	18 (36%)

In our study, 22 patients are detected with bowel wall thickening, 34 patients with GB wall thickening and 18 patients with retroperitoneal edema.

Table 9: Comparative evaluation of MDCT in detection of Extrahepatic manifestations in patients with Cirrhosis of liver

Patients	Study of THI-QAR[14] Medical Journal	Observation of our study
Total no of patients	85	50
No of patients with ascites	85(100%)	50 (100%)
No of patients with splenomegaly	85(100%)	48(99%)

In our study, ascites is detected in 50 patients (100%) and splenomegaly is detected in 48 patients (99%).

Multidetector Computed Tomography (MDCT) is highly sensitive to detect cirrhosis . It's specificity is high when an obvious cause is present and imaging reveals an inhomogeneous hepatic texture or surface, rarefied hepatic central vein, an enlarged caudate lobe, splenomegaly or collateral veins. However, other etiologies such as portal vein thrombosis, parasitic diseases or hematological malignancies need to be excluded, and normal radiographic findings do not exclude compensated cirrhosis.

MDCT with contrast is the modality of choice when HCC or vascular lesions are suspected.

CT scan provides important information on hepatic architecture. Nodularity and surface irregularity is well delineated on CT scan. Atrophy of the right lobe and hypertrophy of the left and especially caudate lobes are well appreciated on CT scan. However, the width of the caudate relative to the right lobe is a poor predictor of cirrhosis. Portal vein thrombosis and collateral formation in cirrhosis of liver are well diagnosed by CT scan. Ultrasonography is the first imaging modality for suspected HCC, but its sensitivity and specificity to detect HCC is below that of CT, and nodular lesions should be confirmed by MDCT.

6. Images & Discussion



Figure 1: Axial images of CECT Abdomen show Regenerative and Dysplastic Nodules

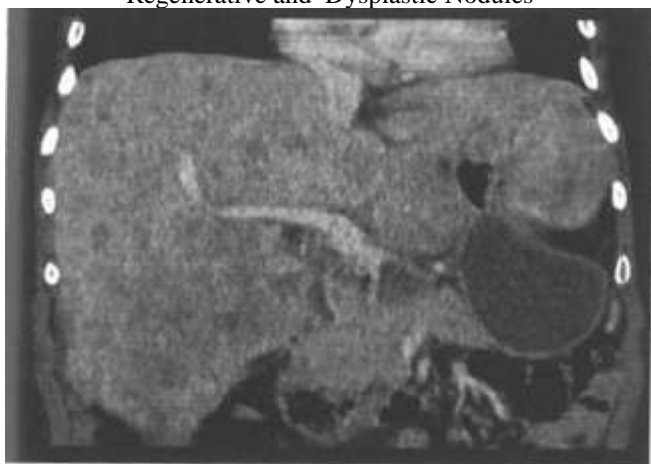


Figure 2: Coronal Image of CT Abdomen shows Regenerative and Dysplastic Nodules with Portal venous thrombosis

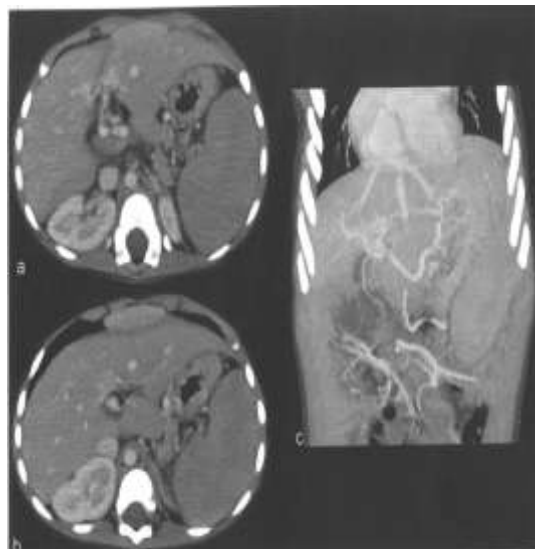


Figure 3: Axial and Coronal images of CT Abdomen shows hypertrophy of Caudate and Left lobe of liver, multiple Collateral formation and Splenomegaly

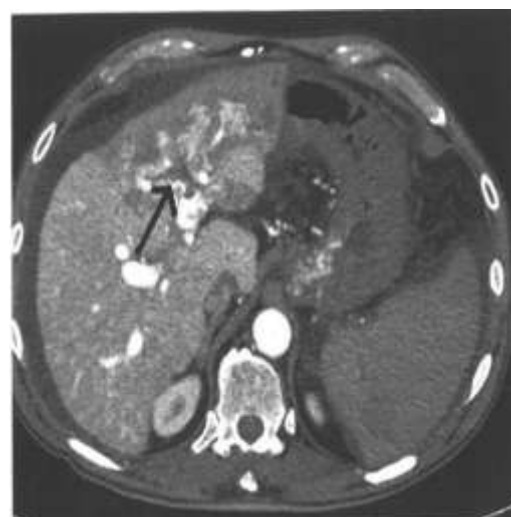


Figure 4: Axial Image of CT Abdomen shows portal cavernoma formation , thrombosis in left branch of portal vein (arrow) , caudate lobe hypertrophy , perigastric collateral formation and Ascites

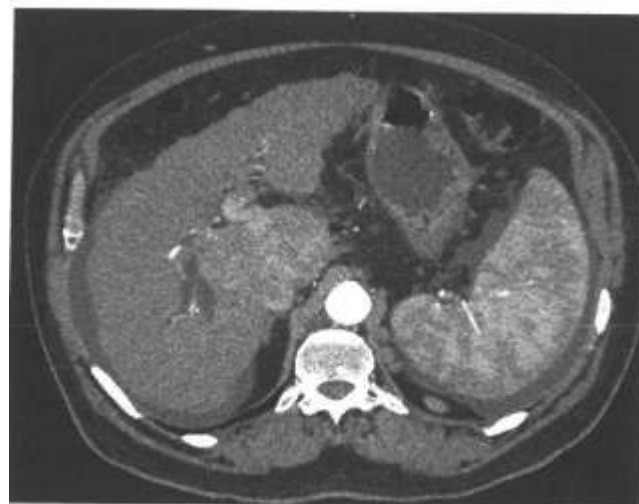


Figure 5: Axial images of CT Abdomen, shows surface irregularity, shrunken right lobe of liver, caudate lobe hypertrophy with regenerative & dysplastic nodules, splenomegaly and ascites

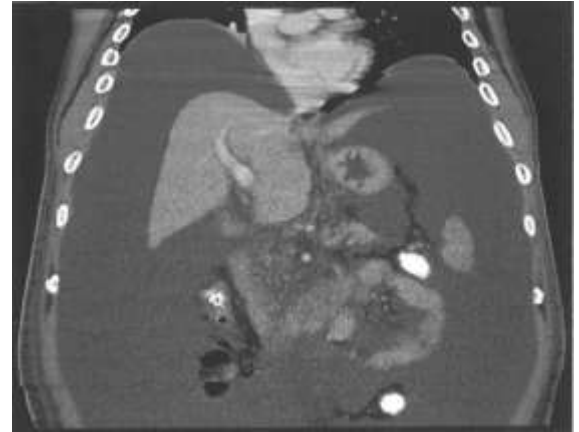
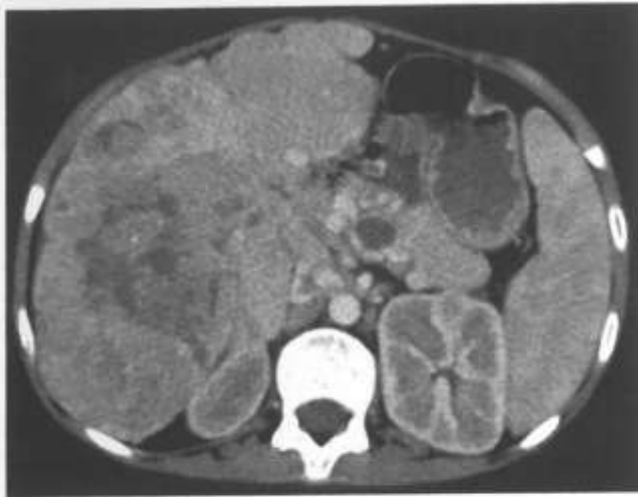
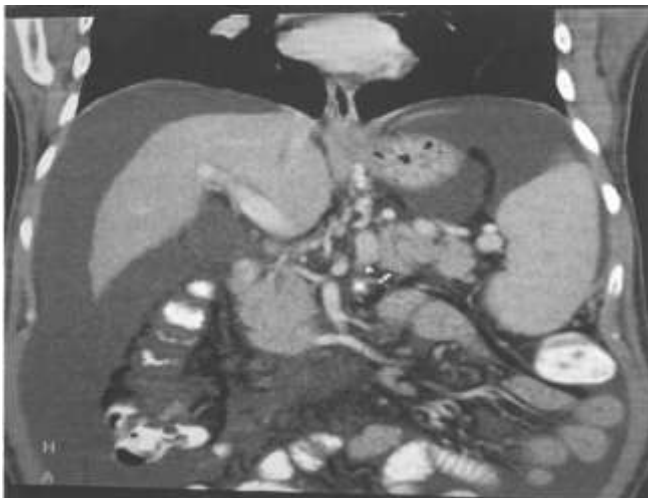
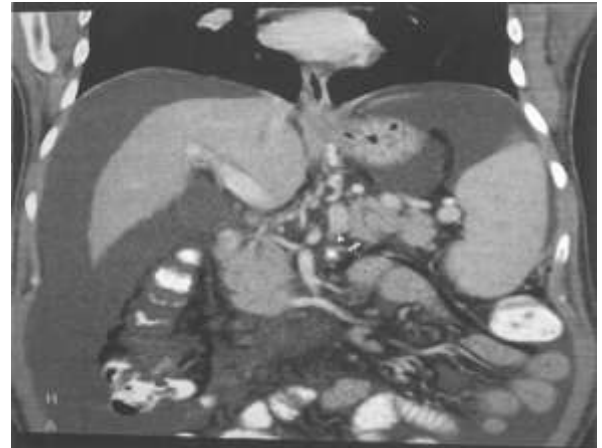


Figure 6: Axial images of CT Abdomen show hepatocellular carcinoma, portal vein thrombosis, surface irregularity, shrunken right lobe, hypertrophy of caudate and left lobe of liver and splenomegaly



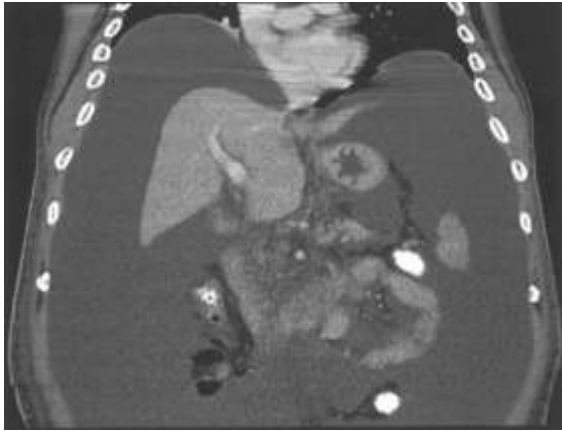


Figure 7: Axial and Coronal view of CT Abdomen shows shrunken right lobe of liver, hypertrophy of caudate lobe , multiple collaterals formation , splenomegaly and ascites

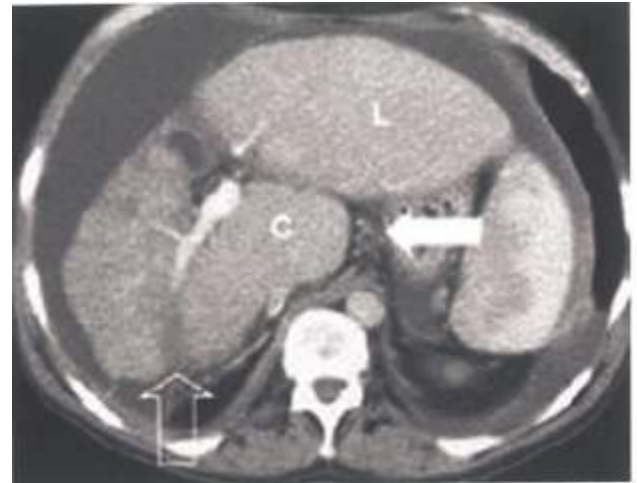


Figure 9: Axial image of CT Abdomen shows Caudate lobe hypertrophy (C) and Left Lobe of Liver (L) with shrunken right lobe of liver (plain arrow) and collateral formation (white arrow) , thickened wall of gall bladder

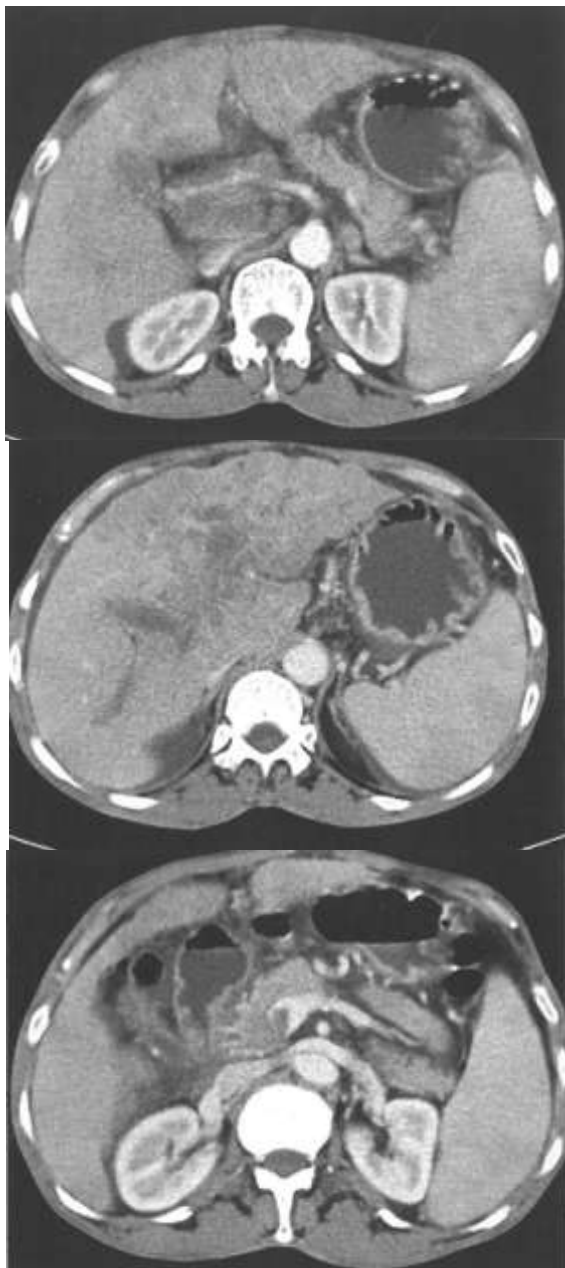


Figure 8: Axial images of CT Scan shows hepatocellular carcinoma, portal vein thrombosis and collateral formation and splenomegaly



Figure 10: Axial image of CT shows surface irregularity, splenomegaly , few peripancreatic collateral formation, shrunken right lobe of liver and hypertrophy of caudate lobe of liver

7. Conclusion & Summary

CT scan is a better diagnostic modality for diagnosis of regenerative and dysplastic nodules, hepatocellular carcinoma, collateral formations and portal vein thrombosis. However surface irregularity is well appreciated on USG.

Although USG remains the screening modality for cirrhosis of liver, MDCT scan plays a superior and very important role in diagnosis of following complications of cirrhosis like:

- Hepatocellular carcinoma
- Portal vein, hepatic vein and splenic vein thrombosis
- Collateral formations.
- Retroperitoneal edema.
- Thickened wall of small bowels.

References

- [1] Grainger R.G. Allison D.J., diagnostic radiology, 5th edition.

- [2] Zakim, Boyer, Reviewed by D H Adams (2003), *Hepatology: a Textbook of Liver Disease*, 4th edition, Saunders, 2003.
- [3] J. J. Brown, M. J. Naylor, and N. Yagan, "Imaging of hepatic cirrhosis," *Radiology*, vol. 202, no. 1, pp. 1-16, 1997.
- [4] G. Brancatelli, R. L. Baron, M. S. Peterson, and W. Marsh, "Helical CT screening for hepatocellular carcinoma in patients with cirrhosis: frequency and causes of false-positive interpretation," *American Journal of Roentgenology*, vol. 180, no. 4, pp. 1007-1014, 2003.
- [5] K. Ohtomo, R. L. Baron, G. D. Dodd III et al., "Confluent hepatic fibrosis in advanced cirrhosis: appearance at CT," *Radiology*, vol. 188, no. 1, pp. 31-35, 1993.
- [6] A. A. Gupta, D. C. Kim, G. A. Krinsky, and V. S. Lee, "CT and MRI of cirrhosis and its mimics," *American Journal of Roentgenology*, vol. 183, no. 6, pp. 1595-1601, 2004.
- [7] I. R. Wanless, F. Callea, J. R. Craig et al., "Terminology of nodular hepatocellular lesions," *Hepatology*, vol. 22, no. 3, pp. 983-993, 1995.
- [8] G. D. Dodd III, R. L. Baron, J. H. Oliver III, and M. P. Federle, "Spectrum of imaging findings of the liver in end-stage cirrhosis: part II, focal abnormalities," *American Journal of Roentgenology*, vol. 173, no. 5, pp. 1185-1192, 1999.
- [9] M. Roncalli, E. Roz, G. Coggi et al., "The vascular profile of regenerative and dysplastic nodules of the cirrhotic liver: implications for diagnosis and classification," *Hepatology*, vol. 30, no. 5, pp. 1174-1178, 1999.
- [10] J. H. Lim, C. K. Kim, W. J. Lee et al., "Detection of hepatocellular carcinomas and dysplastic nodules in cirrhotic livers: accuracy of helical CT in transplant patients," *American Journal of Roentgenology*, vol. 175, no. 3, pp. 693-698, 2000.
- [11] R. L. Baron and M. S. Peterson, "From the RSNA refresher courses: screening the cirrhotic liver for hepatocellular carcinoma with CT and MR imaging: opportunities and pitfalls," *Radiographics*, vol. 21, pp. S117—S132, 2001.
- [12] Wu, Qin, et al. "Characterization of uncommon portosystemic collateral circulations in patients with hepatic cirrhosis." *Oncology letters* 9.1 (2015): 347-350.
- [13] Karahan, Okkes I., et al. "Gastrointestinal wall thickening in patients with cirrhosis: frequency and patterns at contrast-enhanced CT." *Radiology* 215.1 (2000): 103-107.
- [14] Abdulazeez, Riyadh Adel, and Zaid Khedher Ahmad "ABDOMINAL CT FINDINGS IN PATIENT WITH ASCITES." *Thi-Qar Medical Journal* 6.1 (2012): 63-78.



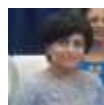
Dr. Mona D Shastri – Head of Department , Department of Radiology, SMIMER Hospital , Umarwada, Surat



Dr. Nipa Patidar – Assistant Professor , Department of Radiology, SMIMER Hospital , Umarwada, Surat



Dr. Dhagash Patel – Senior Resident , Department of Radiology, SMIMER Hospital , Umarwada, Surat



Dr. Avani Bhatt – Senior Resident , Department of Radiology, SMIMER Hospital , Umarwada, Surat

Author Profile



Dr. Ashish Sachdeva – 3rd year Resident Doctor in Radiology, SMIMER Hospital, Umarwada, Surat