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MDCT EVALUATION OF CHRONIC LIVER PARENCHYMAL DISEASE: A CROSS SECTIONAL STUDY

Radiology

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ABSTRACT

Chronic liver disease is an increasingly common and important disorder, now afflicting more than 25% of the world's population. Regardless of its underlying cause, chronic liver disease can progress to cirrhosis, liver cancer, and liver-related death. Cirrhosis is a leading cause of mortality and morbidity across the world. It is the 11th leading cause of death and 15th leading cause of morbidity, accounting for 2.2% of deaths and 1.5% of disability-adjusted life years worldwide. **Aim:** To provide a comprehensive overview of imaging findings in patients with chronic liver disease and cirrhosis. **Material and methods:** MDCT plain and contrast study was done in 50 patients with clinical suspicion of liver disease. CECT is the investigation of choice for detection of chronic liver parenchymal disease and cirrhosis. It has higher sensitivity and specificity detection rate as compared to ultrasound. **Results:** CECT is useful in determining different spectrum of findings of chronic liver disease (CLD).

KEYWORDS

CLD, Cirrhosis, Portal hypertension, HCC, NAFLD

INTRODUCTION

Chronic liver disease represents a spectrum of inflammation, injury, fibrosis, and eventually, cirrhosis (Fig1). The absolute number of CLD cases (inclusive of any stage of disease severity) is estimated at 1.5 billion worldwide. The most common causes of prevalent disease are NAFLD (59%), followed by HBV (29%), HCV (9%), and ALD (2%). Other liver diseases, including primary biliary cholangitis, primary sclerosing cholangitis, alpha-1-antitrypsin deficiency, Wilson's disease, and autoimmune hepatitis, account for 1% of cases. An important caveat to these data is that alcohol most likely accounts for a larger proportion of liver disease prevalence and mortality but is underreported by persons and countries because of cultural concerns and is often a secondary (and unreported) liver disease etiology that coexists with viral hepatitis or NAFLD.

Hepatic cirrhosis is the pathological sequelae of all chronic liver diseases.

Although, USG remains the initial modality for anatomical imaging but several new techniques such as elastography and quantitative US parameters for assessment of liver tissue properties other than echogenicity are introduced. MRI is insensitive in early cirrhosis but has a significant role in screening cirrhotic livers for small hepatocellular carcinomas. MR elastography (MRE) appears to be the most reliable of these new MRI-based methods.

CT represents a mainstay for liver assessment, with the vast majority of acquisitions performed with single energy CT scans [5].

Recent advances in imaging knowledge and technology have elevated the role of radiology in the diagnosis and management of patients with or at risk for chronic liver disease across its entire spectrum and course.

ETIOPATHOGENESIS

Chronic liver disease represents a continuous and progressive process of hepatic fibrosis, liver tissue architectural distortion, and regeneration nodule formation. In chronic liver disease, if not treated, the endpoint is usually irreversible fibrosis, regeneration nodule formation, and development of cirrhosis liver. Hepatic fibrosis is the deposition of extracellular matrix (ECM) in response to chronic liver injury by any etiology [13].

The common pathway is initiated by hepatic stellate cells (HSC), which usually are vitamin A storing dormant cells found in space between sinusoids and hepatocytes. In response to chronic liver injury, HSC gets activated into proliferative fibrogenic myofibroblast and upregulates the expression of inflammatory receptors such as chemokine receptors, ICAM-1, and other inflammatory mediators by releasing chemokines and other leukocyte chemoattractants. This pro-inflammatory phase or initiation phase also changes gene and phenotypic expression of the liver cells, making them more responsive to these inflammatory cytokines, and perpetuation of activated HSC cells results in the accumulation of ECM and progressive fibrosis [14].

Furthermore, 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 (<3 mm) or macronodular (>3 mm).

Cirrhosis is a final stage of chronic liver disease that results in disruption of liver architecture, the formation of widespread nodules, vascular reorganization, neo-angiogenesis, and deposition of an extracellular matrix.

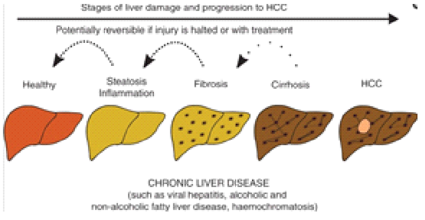

Figure 1: Stages of CLD

Cirrhosis may also present due to one of its complications:

- liver failur
- ascites
- portal hypertension
- hepatocellular carcinoma (HCC)

ROLE OF MDCT

The great benefit of MDCT is in identifying a reliable method to quantify hepatic steatosis or fibrosis as compared to MRI for many clinical purposes [6].

Diffuse uniform fatty infiltration involving the entire liver is the most common pattern of hepatic steatosis. When hepatosteatosis occurs, the average liver attenuation is at least 10 Hounsfield Units (HU) less than

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the splenic parenchyma on unenhanced CT[1] (Fig 2).

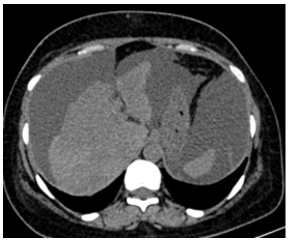

Fig2

Fig 2: 46 year old alcoholic male, CT shows small liver, surface nodular irregularities, ascites with reduced attenuation on unenhanced CT.

The identification of normal course vascular structures in areas of fatty infiltration is crucial to differentiate this abnormality from hepatic tumors.

Evolving hepatic nodular lesions (regenerative nodule, dysplastic nodule or HCC) are another important feature of cirrhosis.

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

Fig 3

Fig 3: A 55 year old male, shows dilated, tortous portal & splenic veins with few perisplenic collaterals and marked splenomegaly.

Portosystemic collaterals develop spontaneously, as blood flow is shunted away from the liver to low pressure systemic vessels 9 (Fig 3). Paraumbilical venous collaterals vessels are found in 43% of patients with portal hypertension. 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 (Fig 4).

Portal hypertension is considered the most common cause of splenomegaly. Mesenteric edema in patients with cirrhosis has a multifactorial pathogenesis. Gastrointestinal wall thickening occurs in 64% of cirrhotic patients, usually as a result of submucosal edema. Hepatic cirrhosis may cause diffuse gallbladder wall thickening[2,3].

Fig 4

Fig 4: A 39 year old female, portal venous phase shows dilated portal vein and central hypodense filling defect suggests thrombosis. Irregular hepatic borders, ascites and pericholecystic edema also seen.

Ascites is the most common complication of cirrhosis. The development of ascites is the final consequence of anatomic and pathophysiologic abnormalities occurring in patients with cirrhosis 4.

Contrast-enhanced CTs in portal venous phase are commonly performed in oncologic patients while multiphase CTs with late arterial, portal venous, and delayed phases are performed for characterization of focal liver lesions 7 (Fig 5).

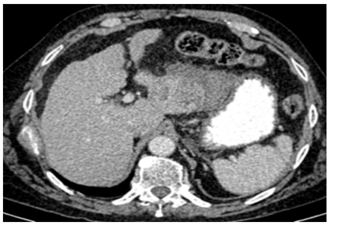

Fig 5

Fig 5: A 35 year old male, CT shows ill defined heterogeneous lesion in noted in segment III of left lobe of liver on venous phases.

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. Focal confluent fibrosis is defined as a peripheral wedge-shaped hypoattenuated area on unenhanced and venous phase CT. On delayed phase, enhancement of the lesion may occur.

Morphologic changes of the 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 8. 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). These changes lead to a nodular contour and heterogeneity of the liver, which is classically associated with cirrhosis.

TABLE – 1 GENERAL DETAILS

FINDINGS IN CIRRHOTIC PATIENTS	NO. OF PATIENTS	FREQUENCY
Surface irregularities	43	86 %
Nodular lesions	19	38 %
Portal vein changes	36	72%
Ascites	28	56%
Splenomegaly	21	42%
Portosystemic collaterals	20	40%
Caudate lobe hypertrophy	15	30%
GB wall edema	08	16%
GI submucosal edema	03	06%

Table 1: Various CT imaging findings in cirrhosis

CASE STUDY

In the present study, MDCT imaging findings of Chronic liver disease and cirrhosis were studied on 50 patients. We found that maximum number of patients which is around 86 % exhibited surface irregularities, followed by 72 % showing portal vein changes. Significant proportion of patients showed ascites (56%), splenomegaly (42%), nodular lesions (38%) including regenerative, dysplastic nodules and HCC . Portosystemic collaterals were found in 40 % and caudate lobe hypertrophy in 30 % of the patients. Around 16 % of patients showed GB wall edema and approx. 6% of patients showed GI submucosal edema.

CONCLUSIONS

Chronic liver parenchymal disease and hepatic cirrhosis is a multifactorial condition with increasing incidence worldwide. Liver fibrosis and cirrhosis are increasingly common in patient populations with a growing incidence of obesity, physical inactivity and alcohol consumption 10, 11. Patients with known risk factors, abnormal clinical and laboratory liver findings or altered imaging results can be screened for liver fibrosis.

HCC, its most lethal complication, seen in 10% of cases after 5 years of hepatitis related cirrhosis. A triple phase evaluation of the liver with CT is essential to detect small HCCs. The recognition of the extrahepatic abdominal complications is vital for adequate clinical assessment and treatment.

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