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Research Article

TO STUDY RADIOLOGICAL FINDINGS OF LIVER CIRRHOSIS BY COMPUTED TOMOGRAPHY (CT)

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ABSTRACT

Cirrhosis, the last stage of chronic liver disease, is a major global health problem because it can result in serious side effects such as variceal haemorrhage, ascites, and hepatic encephalopathy brought on by portal hypertension. This study's objective is to evaluate how effectively computed tomography (CT) imaging can identify liver cirrhosis from radiological findings. A 64row CT scanner named the Somatom Sensation 64 (with a slice thickness of 1.2 mm and a pitch of 0.8, produced by Siemens in Erlangen, Germany) was used to scan all 175 patients (93 with cirrhosis, 45 with precirrhotic fibrosis, and 37 control individuals). From portal venous abdominal scans, only the 5mm axial CT slices were utilised. The scans were sequentially and carefully analysed.

Key Words: Computed tomography, Hepatic encephalopathy, Parenchymal fibrosis, Hypertension.

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INTRODUCTION

Liver cirrhosis is the end stage of chronic liver disease. It is caused by diffuse fibrosis and regenerating nodules that result from recurrent necrosis of liver cell and degeneration. It is recognized as an irreversible form of parenchymal fibrosis. Which reduces hepatic function and results in multiple complications such as nodular regeneration and portal hypertension along with ascites, variceal bleeding, and renal failure due to hepatorenal syndrome, hepatic encephalopathy, and

spontaneous bacterial peritonitis. Recently, early liver cirrhosis was shown to be improved by regression of collagen tissue.

There are a variety of causes of liver cirrhosis, with alcohol consumption, viruses, and fatty liver disease making up the majority of factors. These various etiologies induce chronic inflammation. Normal lobular architecture of the liver parenchyma is replaced by a parenchymal nodule surrounded by the fibrous tissue. Portal-central septa, connecting the portal vein and central vein, develop. As the inflammation persists, various form of fibrosis develops. (Massarrah S, *et al.*, 2004). The gross morphologic appearance of a cirrhotic liver is categorized by the size of the parenchymal nodules: micronodular, macronodular, or mixed. Micronodular cirrhosis is characterized by regenerative nodules of relatively uniform and small size. This pattern is seen in chronic alcoholic, hepatitis C, and biliary cirrhosis. In macronodular cirrhosis, the parenchymal nodules are larger, and more variable in size. Chronic hepatitis B is the most common cause of macronodular cirrhosis. Therefore, early diagnosis of liver cirrhosis and quantification of the proportion of

fibrosis in the liver are very important in the management of chronic liver disease. Prognosis and management of chronic liver diseases hinge strongly on the amount and progression of liver fibrosis. (Castera *et al.*, 2011)

Early diagnosis improves the benefit of therapeutic strategies before the development of irreversible and potentially lethal complications such as loss of liver function, oesophageal variceal bleeding, hepatic encephalopathy and hepatocellular carcinoma. (Gentilini *Pet al.*, 2018)

The new sensitive methods using magnetic resonance imaging (MRI) have been described, such as MR-elastography, double contrast-enhanced MRI and diffusion weighted MRI. Computed tomography (CT) is useful for imaging liver cirrhosis complications, such as portosystemic collaterals with bleeding. However, this is not an appropriate method for the primary diagnosis of liver fibrosis, because of the radiation dose and inferior accuracy compared to the fibro scan. (Garcia-Tsao *et al.* 2001) On the other hand, clinically occult liver fibrosis as an auxiliary finding in routine abdominal CT scans is under diagnosed.

Even liver cirrhosis has a mediocre sensitivity (77.1%–84.3%) and specificity (52.9%–67.6%) in CT⁵. However, since CT is an important and frequently used diagnostic tool in modern medicine, an accurate method to detect liver fibrosis in CT scans could bring forward the diagnosis and enable treatment in an early stage of fibrosis before its clinical appearance. The aim of the study Radiological findings of liver cirrhosis by Computed tomography (CT).

MATERIAL AND METHODS

A total of 175 patients (116 male/59 female) were retrospectively included between January 2017 and March 2019 SreeBalaji Medical College and Hospital, Chrompet, Chennai, including 93 patients with histologically proven liver cirrhosis (fibrosis stage 4), 45 with histologically proven precirrhotic liver fibrosis stage 1–3 and a control group of 37 trauma patients without known liver pathology. The mean age of all selected patients was 57.3 years (range: 32–75 years).

The 93 patients (69 male/24 female) with liver cirrhosis (26 Child A, 35 Child B, 32 Child C) and the 45 patients with precirrhotic stage of liver fibrosis (7 fibrosis grade 1, 19 fibrosis grade 2 and 19 fibrosis grade 3) were included if they had undergone a CT scan with portal venous phase in the radiological information system of our hospital.

Liver fibrosis and cirrhosis was histologically proven by intercostal percutaneous biopsy from the right liver lobe with the “Menghini-technique” with pre- and post-procedural sonographic checks. Patients who had undergone an earlier partial liver resection or liver

transplantation or those who had a transjugular portosystemic shunt (TIPS) were excluded.

The control group consisted of 37 consecutively selected trauma patients (27 male/10 female) with a mean age of 60.5 years (range: 51–70 years) who were examined with a portal venous phase abdomen (Kudo M, *et al.*, 2008). Patients with liver laceration, known liver fibrosis or cancer, and patients receiving potentially hepatotoxic medication were excluded. The clinical records of all patients in the fibrosis/cirrhosis group were surveyed.

CT imaging technique and measurements

Images of all 175 patients (93 cirrhosis, 45 precirrhotic fibrosis and 37 control patients) were acquired by a 64-row-CT-unit Somatom Sensation 64 (24 x 1.2 mm, pitch 0.8, slice 1.5/5 mm, Siemens, Erlangen, Germany). Exclusively, 5-mm axial CT slices of portal-venous abdominal CT-scans were used. The scans were systematically reviewed in consensus by two radiologists with 2 and 10 years of experience in abdominal imaging, who were blinded to any clinical and imaging results or histological fibrosis stage. The diameters of the three main liver veins were measured 1–2 cm before their aperture into the inferior caval vein and added to give a sum (ld-score).

Accessory hepatic vein branches were not measured. The caudate-right lobe ratio (crl-r) was calculated as described (Awaya *et al.* 2002) Distance from the right lateral border of the first bifurcation of the right portal vein to the medial border of the caudate lobe and to the lateral border of the right liver lobe were divided as illustrated in figure 3.

For a combination of both, two scores were calculated: ld/ crl-r (sum of the three main liver diameters divided by the caudate-right lobe ratio) and rhvd/crl-r (right hepatic vein diameter divided by the caudate-right lobe ratio). In addition, the maximum diameter of the portal vein and the maximum splenic diameter in a strictly axial plane were measured. The presence of hepatic surface nodularity, ascites and portosystemic collaterals was also captured.

Boxplots were used to compare the distribution of each sign across Child classifications, fibrotic and control groups. Average diameters and the sum of the veins were calculated using the formulae described above. The ld/crl-r was defined as the sum of the veins divided by the caudate-right lobe ratio. Receiver operating characteristic (ROC) curves for fibrosis and cirrhosis were calculated for each sign, ratio and score. The area under the curve of the ROC analysis was used to rank the signs for fibrosis and cirrhosis individually. Threshold levels for all findings were determined individually for fibrosis and cirrhosis to maximize sensitivity and specificity. ROC-Curves were compared

applying the pairwise comparison of the area under the curve. The standard errors of the areas under the curves provided the significance level, which equalled the probability of the hypothesis that the difference between the two areas under the curve is zero.

Visual correlation between the signs and scores was explored using pairwise scatterplots. Based on this knowledge, we designed an improved prediction rule for cirrhosis using linear discriminant analysis (LDA), which looks for a linear combination of predictors to best separate the disease classification groups. Plots and statistical analysis were performed on the R statistical software platform. Chi-square test was used to find specific CT signs for each aetiology. A p-value <0.05 was considered significant.

RESULTS

CT findings suggesting fibrosis/cirrhosis

Except for the portal vein diameter, univariate boxplots of all quantifiable findings such as liver vein diameters, caudate-right lobe ratio, including the ratio of both, and the splenic diameter, demonstrated a clear discrimination between normal and cirrhotic liver. A differentiation between the three Child-Pugh stages was never apparent using CT findings. Also, there was no clear visual differentiation between fibrosis and normal or cirrhotic liver; the fibrosis group always ranged between the two groups. Pairwise plots of these predictor variables suggested a possible distinction between the groups for most variable pairs: the precirrhotic fibrosis group always ranged between the control group and the cirrhosis group, legitimising a combined prediction. All 95% confidence intervals of areas under the curve (AUCs).

Cirrhotic liver

Area under the ROC curve for most predictors ranged between 0.82 and 0.88, except for measurement of the portal vein diameter, which showed a much lower AUC for cirrhosis of 0.62. Liver vein diameters were better predictors (AUC: 0.82–0.88) than the caudate-right lobe ratio (0.82). The combination of both (ld/crl-r) scored the highest AUC (0.89) and is, therefore, considered the best quantifiable radiological sign to distinguish between cirrhotic and normal liver. The second best predictor for cirrhosis was the sum of all vein diameters (AUC = 0.88) and splenic diameter (AUC = 0.88), followed by the right (AUC = 0.87), the left vein diameter (AUC = 0.86) and the caudate-right-lobe ratio (AUC = 0.82). Qualitative signs such as liver surface nodularity, ascites and collateral vessels demonstrated high specificity (100%, 82% and 100%, respectively) and lower sensitivity (58%, 91% and 72%, respectively). When the predictors were compared with the ld/crl-r, some variables tested significantly inferior: portal

diameter, ascites, caudate-right-lobe ratio and middle hepatic vein diameter showed a p-value below 0.05.

Precirrhotic Fibrotic Liver

The AUC for fibrosis was always lower than the AUC for cirrhosis and ranged from 0.81 to 0.92, except for portal vein diameter (0.56). The best sign for liver fibrosis was also the ld/crl-r score (AUC = 0.82), followed by the sum of vein diameters (AUC = 0.79) and the left hepatic vein diameter (AUC = 0.76). Splenic diameter (AUC = 0.76) was better than caudate-right lobe ratio (AUC = 0.72). A significant difference compared with the ld/crl-r could be shown for portal vein diameter, collaterals, ascites and middle hepatic vein. Qualitative signs such as liver surface nodularity, ascites and collateral vessels demonstrated high specificity (100%, 82% and 100%, respectively) and rather low sensitivity (58%, 34% and 38%, respectively).

Threshold-based sensitivity and specificity

The threshold values showed the greatest accuracy for a combination of the sum of the three main liver vein diameters and the caudate-right lobe ratio. An ld/crl-r score ≤ 18.6 identified cirrhosis with a sensitivity of 88% and a specificity of 82%. An ld/crl-r score ≤ 25.9 was the threshold for fibrosis with a sensitivity of 84% and a specificity of 77%. A combined variable of the right hepatic vein diameter and the crl-r (rhvd/crl-r) was slightly inferior, especially for the fibrosis group. The sum of the three main liver vein diameters (ld score) demonstrated a higher sensitivity but a lower specificity than the crl-r. If only one hepatic vein diameter was measured, the diameter of the right hepatic vein showed the highest accuracy for cirrhosis and the left hepatic vein demonstrated the highest accuracy for fibrosis.

Fibrotic versus cirrhotic liver

To differentiate between cirrhotic and fibrotic liver, qualitative variables are superior to quantifiable variables. The highest AUC of ROC was found for collaterals (0.73), followed by nodular liver surface (AUC = 0.68) and ascites (AUC = 0.66). The quantifiable variables and scores demonstrated inferior AUC between 0.59 and 0.69 compared with the predictor collaterals (p-value: 0.059–0.58).

Aetiology of liver fibrosis/cirrhosis and specific CT findings

There was no significant difference between the cirrhosis aetiologies and the imaging findings, except for ascites, which was more frequently found in the alcoholic cirrhosis group (p-value = 0.045).

Combined prediction

The weighted sum of the best individual predictor variables for cirrhosis was used to analyse the combined prediction: $\text{combvar} = -0.015 * \text{ld/crl-r} + 0.215 * \text{crl-r} - 0.045 * \text{ldscore} + 0.66 * \text{ascites} (1 = y/0 = n) + 0.819 * \text{nodular liver surface} (1 = y/0 = n) + 1.905 * \text{collaterals} (1 = y/0 = n)$. This combined variable reached a sensitivity of 94% and aspecificity of 100% for cirrhosis. This means that a mathematical combination of all evaluated imaging findings could identify liver cirrhosis in abdominal CT scans in 94 out of 100 cases with almost no false positive results.

DISCUSSION

The satisfactory interpreter to discover liver fibrosis turned into an aggregate of the liver vein diameters and the caudate-right-lobe ratio (ld/crl-r). This ratio is calculated by means of including the diameter of the three essential liver veins 1–2 cm away from the inferior caval vein and dividing this sum by means of the caudate-proper lobe ratio.

An ld/crl-r of <20 showed a sensitivity of 88% and a specificity of 82% for liver cirrhosis, which is higher than that of Kudo M, Zheng RQ, et al study CT symptoms for liver cirrhosis. CT findings for liver fibrosis in a precirrhotic stage (f1–f3) have not been properly investigated till now. With an ld/crl-r ratio of <24 it's miles viable to hit upon precirrhotic liver fibrosis with a sensitivity of 83% and a specificity of 76%. If the ld/crl-r ratio become calculated in every habitual CT, liver fibrosis as a tentative prognosis might justify laboratory trying out and a fibroscan in these patients and produce ahead analysis. (Kudo *Met al.*, 2008)

To optimise this fibrosis assessment, a likely paintings-up system is shown in determine 5, which would take approximately 2 mins with a calculator to hand. Another, more pragmatic possibility could be to calculate the ld/crl-r most effective if the liver veins appear small or if the caudate lobe appears nearly as broad as the proper hepatic lobe on a subjective visible affect. Measurement of the ld/crl-r is likewise viable with MRI and b-mode sonography, even if specific reproduction of the axial plane on the level of the proper portal vein bifurcation is hard in sonography.

Not all the tested imaging findings were statistically significantly not as good as the ld/crl-r. While the crl-r become notably inferior for cirrhosis ($p = \text{zero}.028$), it become now not for fibrosis ($p = 0.081$). The sum of the liver vein diameters (ld) was additionally a good predictor by myself and not considerably not so good as the mixed ld/crl-r radio for cirrhosis ($p = 0.291$) and fibrosis ($p = \text{zero}.548$).

In this study the high specificity of the splenic diameter for liver fibrosis is probably a bias, due to

splenomegaly. However, the sensitivity of 62% of the splenic diameter for fibrosis is rather low compared with the ld/crl-r (83%). The portal vein diameter is a poor predictor because of assess liver fibrosis, as previously reported (Lafortune M, *et al* 1984). All of the measured parameters for precirrhotic liver fibrosis were between the healthy control group and the liver cirrhosis group. This is not surprising, since liver fibrosis is a continuously ongoing condition ending in irreversible end-stage liver cirrhosis.

As already reported for other diagnostic tools such as sonographic elastograph, differentiating normal liver from early fibrosis (f1–2) is more challenging than differentiating normal liver from f3 stages and cirrhosis (f4). The patient population was not big enough in this study to differentiate significantly between the histological grades. However, there was a sufficient differentiation between normal liver and fibrosis (f1–3) as well as normal liver and cirrhosis (f4).

Qualitative signs of liver conversion, such as hepatic surface nodularity, the right posterior notch sign, blunt edge of the liver, ascites and portosystemic collateral vessels, showed a high specificity if positive but a small sensitivity in precirrhotic fibrosis. In present study ascites more significantly frequently in alcohol-induced liver fibrosis than in other cirrhosis aetiologies. A correlation of enlargement of the caudate lobe in alcohol induced cirrhosis compared to viral-induced cirrhosis as described by Okazaki *etal.* was not reproducible but our study is the retrospective single centre design. (Okazaki H, *et al.* 2000)

This could also rule out other possible circumstances leading to a low hepatic vein diameter, for example acute hepatitis without fibrosis⁹. However, a coexisting active hepatitis was not detected by the pathologist in any fibrosis specimens used for present study. In addition, the probability of hepatic remodelling and thus alteration of the caudate right-lobe ratio as a result of acute hepatitis is low. Another limitation is the disregard of a possible impact of cardiovascular factors such as blood pressure and hydration of the patients on the hepatic vein diameters, which have been considered in this study. However, it is the control group with the trauma patients who seem to be more prone to false low hepatic vein diameters due to possible hypotension and low hydration.

CONCLUSION

The most effective method for detecting liver fibrosis in CT images is combining hepatic vein diameters and caudate-right-lobe ratio. Laboratory testing and fibroscans are justified for abdominal CTs with ld/crl-r ratios below 24, potentially enabling early treatment.

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