The Eurasian
Journal of Medicine

Original Article

Impaired Visibility of the Hepatic Veins on the Multi-Detector Computed Tomography in Patients with Cirrhosis

Sirozlu Hastalarda ÇKBT'de Hepatik Venlerin Yetersiz Vizualizasyonunu

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Abstract

Objective: To determine the prevalence and associations of the impaired visibility of the hepatic veins (HV) on the multi-detector row computed tomography (MDCT) in cirrhotic patients.

Materials and Methods: Eighty-seven consecutive cirrhotic patients undergoing triphasic liver MDCT were enrolled. The patency of the HV and the direction of the blood flow in the main portal vein, inhomogeneity of the liver, portal vein thrombosis (PVT), a spontaneous splenorenal shunt, splenic indexes (cm³), contour abnormalities, and enlargement of the fissure were evaluated.

Results: The prevalence of the impaired visibility of the HV was 38% in the patients with cirrhosis. No significant associations exist between the impaired visibility of the HV and age (p=0.96), sex (p=0.14), portal vein thrombosis (p=0.29), or splenic indexes (p=0.32). Inhomogeneity of the liver (p=0.0001), marked contour abnormalities, (p=0.0001), splenorenal shunt (p=0.02), enlargement of fissure (p=0.0001), and hepatofugal flow (p=0.01) were significantly associated with the impaired visibility of the HV.

Conclusion: Inhomogeneity of the liver, marked contour abnormalities, and hepatofugal flow are independently associated with the impaired visibility of the HV in cirrhotic patients on hepatic venous phase CT.

Key Words: Liver, hepatic vein, cirrhosis, multi-detector computed tomography, visibility

Özet

Amaç: Sirozlu hastalarda çok kesitli bilgisayarlı tomografide de (ÇKBT) hepatic venlerin yetersiz vizualizasyonunun sıklığını belirlemektir.

Gereç ve Yöntem: ÇKBT ile Trifazik karaciğer incelemesi yapılan ardışık 87 hasta çalışma kapsamına alındı. Hepatik venlerin patensi durumu, ana portal vendeki akım yönü, karaciğerin inhomojenitesi, portal ven trombozu (PVT), spontan splenorenal şant, splenik indeksler (cm³), kontur anormallikleri ve fissurde genişleme değerlendirildi.

Bulgular: Sirozlu hastalarda hepatik venlerin yetersiz visualize olmasının sıklığı %38'dir. Hepatik venlerin yetersiz visualize olması ile yaş (p=0.96), cinsiyet (p=0.14), PVT (p=0.29) ya da splenik indeksler (p=0.32) arasında anlamlı ilişki saptanmadı. Hepatik venlerin yetersiz visualize olması ile karaciğerin inhomojenitesi (p=0.0001), belirgin kontur anormalliği (p=0.0001), splenorenal şant (p=0.02), fissürde genişleme (p=0.0001) ve hepatofugal akım (p=0.01) arasında anlamlı ilişki saptandı.

Sonuç: Sirozlu hastalarda BT de hepatik venöz fazda hepatik venlerin yetersiz visualize olması ile karaciğerin inhomojenitesi, belirgin kontur anormalliği ve hepatofugal akım bağımsız olarak ilişkilidir.

Anahtar Kelimeler: Karaciğer, hepatik ven, siroz, çok kesitli bilgisayarlı tomografi, görünürlük

Introduction

Cirrhosis is characterized by chronic damage of the liver, parenchyma injury leading to extensive fibrosis and nodular regeneration [1]. Altered hepatic morphology occurs accompanying a progressive loss of function [2]. Triphasic liver computed tomography (CT) including arterial, portal, and hepatic venous phase is generally performed in cirrhotic patients for surveillance of hepatocellular carcinoma (HCC). We have frequently observed in the daily practice that the hepatic veins (HV) do not visualize clearly even on the hepatic venous phase CT in cirrhotic patients. Therefore, we aimed to evaluate the prevalence and associations of the impaired visibility of the HV in cirrhotic patients using multi-detector computed tomography (MDCT).

Materials and Methods

Patient population

This study has been approved by the institutional review board. From January 2009 to February 2010, the images of the patients with cirrhosis who underwent triphasic CT protocol (arterial, portal phase, hepatic venous phase) were prospectively reviewed.

A total of 107 consecutive patients underwent triphasic liver CT scan enrolled in this study. Patients were excluded

from the study if they had known or suspected HCC with an elevated tumour marker (n=10); they had cirrhosis secondary to the hepatic venooclusive disease (n=5); they had splenectomy (n=2); the presence of the intrahepatic portacaval shunt (n=1), detailed clinical data and laboratory findings and complete triphasic CT exam according to our routine protocol were unavailable for review (n=2). None of the patients included in the study had clinical evidence of the failure of the right side of the heart.

A total of 87 patients met these criteria and were included in the study group. There were 24 females and 63 males aged 37-83 years (mean 57.8±11.3 years). The clinical severity of the cirrhosis was determined according to the criteria of Child-Pugh classification. Forty-four patients had Child-Pugh class A, 35 had Child-Pugh class B, and 8 had Child-Pugh class C. The cirrhosis was caused by viral infection (hepatitis B [n=54] or hepatitis C [n=15]), cryptogenic (n=9), and alcoholic (n=9). The diagnosis of cirrhosis was established by a percutaneous liver biopsy in 22 patients and by clinical and imaging findings in 65 patients.

CT scanning protocol

At our institution, triphasic liver CT is performed for the surveillance of HCC in cirrhotic patients. All helical CT scans were obtained by using a 64-slice CT scanner (Aquillion 64, Toshiba Medical Systems, Tokyo, Japan). Scanning parameters were as follows: 120 kV, 37-180 mAs, 64 x 0.5 mm detector configuration, rotation time 350 msec. The scanning protocol consists of an initial unenhanced study to identify the liver location with 5-mm collimation. After then, intravenous injection of contrast material (110 mL non-ionic contrast material, 350 mgI/mL, at a rate of 5 mL/s) through 18 G cannula placed into the right median antecubital vein using a double-head power injector was performed and was followed by 40 mL of normal saline at 5 mL/s. Arterial-phase imaging was initiated within 5 sec after the enhancement of the descending aorta to 150 HU, as measured by an automated bolus-tracking technique (SureStart, Toshiba Medical Systems, Tokyo, Japan) with 0.5-mm collimation. The portal venous phase was initiated with a 75-sec delay time after the initiation of the contrast material injection. The hepatic venous phase was obtained with a 120-sec delay time after the initiation of the contrast material injection. Arterial and portal venous phase covered only the liver while the hepatic venous phase covered the entire abdomen. For diagnostic reading, 5-mm slice thickness and 5-mm reconstruction interval were obtained and then images transferred to the workstation (Vitrea, version 3.2, Vital Images, Minneapolis, USA).

Doppler examination

The patency of the HV and the flow direction in the main portal vein were determined based on the results of colour Doppler US. Doppler US examinations were performed with 3-4 MHz convex probe (GE Medical systems, Milwaukee, WI, USA) by using a LOGIQ 900 scanner (GE Medical systems, Milwaukee, WI, USA). All patients included, the mean interval between CT and Doppler US were 6 days (ranged 1-13 days). The results of the Doppler US were considered as the gold standard for the patency of HV.

Image evaluation

Two radiologists blinded for the results of the Doppler examination analysed all triphasic CT images. In cases of inter-observer disagreement, final decisions were reached by means of consensus. Helical CT images were obtained with the standard settings of window level and width used in our department for the evaluation of liver parenchyma (WL 50 HU, WW 350 HU); and the same values were used to assess the images. Each radiologist evaluated the images for the presence or absence of liver inhomogeneity, portal vein thrombosis (PVT), spontaneous splenorenal shunt, contour abnormalities, and enlargement of the fissure. Hepatic venous phase CT images were used to score the visibility of the HV. The portal venous phase CT images were chosen for the evaluation of the liver inhomogeneity, PVT, spontaneous splenorenal shunt, contour abnormalities, and enlargement of the fissure. Splenic indexes (cm^3) were measured by each radiologist. The mean splenic index was also calculated on portal venous phase CT images.

The inhomogeneity of the liver was defined as patchy areas or mottled regions of low attenuation or lacelike or thick bands of low attenuation on portal phase CT images due to the fibrosis or regeneration nodules [3]. PVT was defined as the presence of the occlusive thrombus within the lumen of the portal vein or cordlike structure with no patent lumen of the portal vein [4]. A spontaneous splenorenal shunt was defined as the anastomosis of the splenic vein or perisplenic varices to an enlarged left renal vein. The reason that we choose splenorenal shunts among portosystemic collaterals was significantly dropping in liver perfusion if present [5]. Long-axis, short-axis, and craniocaudal measurements (cm) of the spleen were obtained at the level of splenic hilum. These measurements were used to calculate the splenic indexes (cm^3). The contour abnormalities were considered absent if no visible or minimal contour abnormalities are seen and present when marked lobulation or irregularity are detected (Figure 1). The enlargement of the liver fissure scored as minimal (<or=1.5 cm) or marked (1.5 cm<). Assessment of the HV visibility was categorized as not visible or faint and well-visualized (Figure 2).

Statistical analysis

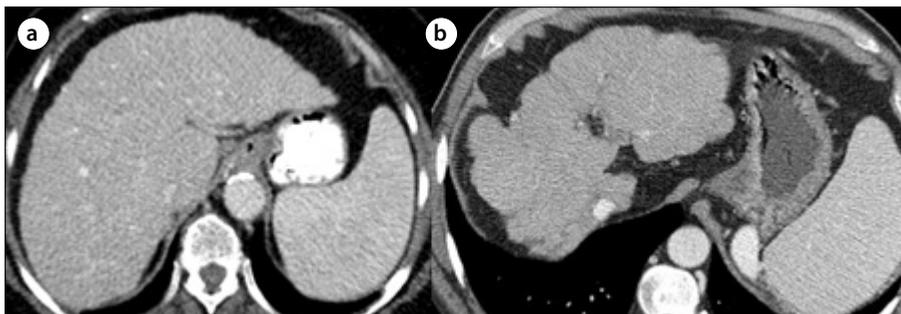Statistical analyses were performed with commercially available statistical software (SPSS, version 11.5 for

Figure 1. a, b. *Minimally contour abnormalities of the liver (a), marked contour abnormalities of the liver on axial contrast-enhanced portal venous phase CT images (b).*
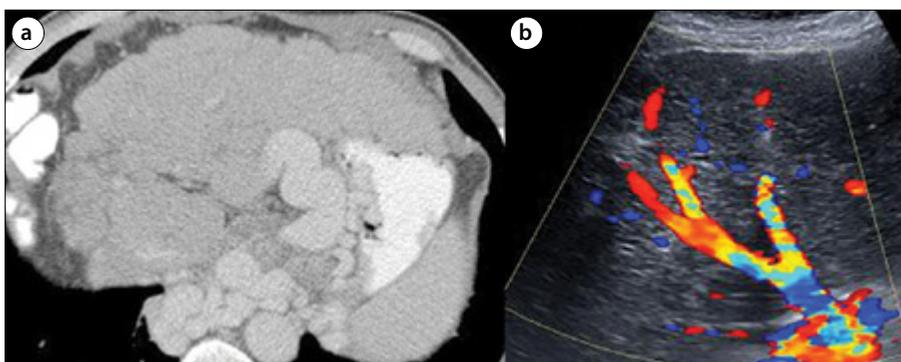

Figure 2. a, b. *Axial contrast-enhanced hepatic venous phase CT image showed inhomogeneity and marked contour abnormality of the liver. No visible hepatic veins on CT image. Note large paraesophageal and fundal varices (a), Colour Doppler US revealed the patency of the hepatic veins (b).*

Windows; SPSS Inc., Chicago, USA). Quantitative variables were expressed as mean values and standard deviations. Univariate analyses were performed to determine whether an association exists between the explanatory variable and the impaired visibility of the HV. Chi-square test and Fisher exact test were used for categorical variables and Student's t tests were used for continuous variables. Logistic regression analysis was used for multivariate analysis. Individual variables with a univariate p value less than or equal to 0.10 was retained in a logistic regression model. Statistical significance was considered to exist when $p < 0.05$.

Results

The HV was patent in all patients on Doppler US. The prevalence rate of the impaired visibility of the HV on CT was 38% in the patients with cirrhosis. Table 1 summarizes the results of the univariate analysis. No significant associations exists between the impaired visibility of the HV and age (p=0.96), sex (p=0.14), PVT (p=0.29) or splenic indexes (p=0.32). Inhomogeneity of the liver (p=0.0001), marked contour abnormalities (p=0.0001), splenorenal shunt (p=0.02),

and hepatofugal flow (p=0.01) were significantly associated with the impaired visibility of the HV. Latter variables deserve further analysis with a logistic regression model. Inhomogeneity of the liver, marked contour abnormalities, and hepatofugal flow were all independently associated with the impaired visualization of the HV (Table 2).

Discussion

Cirrhosis causes some architectural and hemodynamic changes in the liver. Cirrhotic process distorts the hepatic parenchyma as nodularity of the liver surface at different grades. Nodularity of the liver corresponds to the variable sizes of the regenerative nodules. Atrophy and hypertrophy of the liver segments also contribute to the contour lobulation of the liver [3, 6]. Additionally, diffuse heterogeneity is observed in approximately 25% of the cirrhotic patients due to iron, fat deposition, and fibrosis [7]. Enlarged hilar periportal space and gallbladder fossa secondary to the atrophy of the liver segments and replaced by fat are other classical findings of advanced cirrhosis [6, 8]. Increased intrahepatic portal resistance causes portal hypertension. Portosystemic

Table 1. Variables effecting the visibility of the hepatic veins (HV) on computed tomography

Variables	HV invisible (n=33)	HV visible (n=54)	p
Age (years)[b]	56.4±11.2	58.6±11.4	0.96[a]
Female	6 (18)	18 (33)	0.14[a]
Inhomogeneity of the liver	25 (75)	3 (0.5)	0.0001
Portal vein thrombosis	5 (15)	4 (0.7)	0.29[a]
Splenorenal shunt	11 (33)	6 (11)	0.02
Splenic index (cm^3)[b]	893±552	968±866	0.32[a]
Marked contour abnormalities	23 (69)	2 (0.3)	0.0001
Marked enlargement of fissure	27 (50)	12 (22)	0.0001
Hepatofugal flow	10 (30)	50 (92)	0.01

Note–[b]: Values are given as mean±standard deviation, [a]: not significant, Numbers in parenthesis represent percentage, HV: hepatic veins

Table 2. Multivariate analysis

Variables	OR	Lower 95% CL	Upper 95% CL	p
Inhomogeneity of the liver	39.2	5.7	266.8	0.0002
Marked contour abnormalities	45.7	5.5	373.8	0.0004
Hepatofugal flow	19.6	2.56	150.7	0.004

OR: odds ratio; CL: confidence limits

collaterals are developed to alter the blood flow from the high-pressure portal system to the low-pressure systemic circulation [1, 4-6, 9].

In our study, we found that inhomogeneity of the liver and marked nodularity were associated with the impaired visualization of the HV on CT. Marked contour nodularity and the structural changes in the liver parenchyma corresponding to the inhomogeneity of the liver can cause the disruption of the course as well as compression of the HV. Non-uniform HV wall and altered straightness has been shown by Vessal et al. [10] on sonography in cirrhotic patients.

Changes that occur in cirrhotic liver cause the diminished portal perfusion of the liver. As a consequence, blood flow into the HV will decrease. Finally, decreased blood flow toward the HV may contribute to the impaired visualization of the HV. Our study showed that there is a significant association between the hepatofugal flow in the portal vein and the impaired visualization of the HV. Splenorenal shunts are generally thought to drop the perfusion of the liver substantially [5]. However, the association between splenorenal shunts as independent variable and the impaired visibility of the HV could not reach the significant level. Since we only consider these shunts as present or absent, quantification of these shunts as well as other portosystemic collateral sites was not made, as a consequence significant association might not be detected. Bryce et al showed in their study that if spontaneous splenorenal shunts is present, main portal vein diameter is reduced which may be an indicator of hepatofugal flow in cirrhotic liver [5]. In our study, we paid attention to splenorenal shunts because of causing decreased outflow to the HV with regard to diminished portal perfusion.

Scan time should be emphasized as another possible factor leading to the impaired visualization of the HV. In our institution, we routinely perform triphasic liver CT including arterial, portal and hepatic venous phase with high injection rate of non-ionic iodinated contrast media for the surveillance of cirrhosis secondary to the different etiologies. Triphasic liver CT is performed for the pre-operative detailed visualization of the vascular structures if transplantation is planned, the detection of hepatocellular carcinoma, or the evaluation of the cirrhotic patients with chronic Budd-Chiari syndrome in our hospital. Delayed phase images are usually included in the liver CT since tumoural wash-out can be seen in HCC. Also, we can evaluate the HV for invasion by HCC or occlusion in Budd-Chiari syndrome in delayed phase images. Probably related to the alteration of the hemodynamic features of the cirrhotic liver, the scan time might not be set in a proper way. As a consequence, the scan timing as well as

the hemodynamic and the architectural changes could be responsible for the impaired visualization of the HV.

A substantial number of the cirrhotic patients showed the impaired visibility of the HV without any occlusion or obstruction of the HV on CT. In such patients, it would better to evaluate the patency of the HV by another imaging method such as Doppler US.

This study had several limitations. First, conventional venography was not performed in our study population because the confirmation of the HV patency was based on the results of colour Doppler US. Second limitation is about the retrospective study design which could lead to the possibility of bias when simultaneously assessing the hepatic vein visibility and the associated findings. Lastly, we used fixed dosage of contrast material and fixed delay time for the portal and delayed phase CT in each patient regardless of body weight or circulatory status.

In conclusion, since the prevalence of the impaired visibility of the HV was 38% in the patients with cirrhosis, the evaluation of the HV patency by CT may not be reliable in cirrhotic patients. Inhomogeneity of the liver, marked contour abnormalities and hepatofugal flow in the portal vein are independently associated with the impaired visibility of the HV in cirrhotic patients on hepatic venous phase CT.

Ethics Committee Approval: Ethics committee approval was received for this study from the ethics committee of Turkiye Yuksek İhtisas Hospital, Ankara, Turkey.

Informed Consent: Written informed consent was not obtained due to the retrospective nature of this study.

Peer-review: Externally peer-reviewed.

Author Contributions: Concept - N.I.O., M.Y.; Design - N.I.O., M.Y., M.T.; Supervision - N.I.O., M.Y., M.T.; Funding - N.I.O.; Materials - N.I.O., M.Y.; Data Collection and/or Processing - N.I.O., M.Y.; Analysis and/or Interpretation - N.I.O., M.Y., M.T.; Literature Review - N.I.O.; Writing - N.I.O., M.Y.; Critical Review - N.I.O., M.Y., M.T.

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