Evaluation of the liver function by the liver parenchyma, spleen and portal vein signal intensity during the hepatobiliary phase with Gd-EOB-DTPA-enhanced MRI

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Abstract

Background: Assessment of liver function is essential for the treatment and prognosis of patients with cirrhosis, previous studies used signal intensity of hepatobiliary phase to reflect liver function on gadolinium-ethoxybenzyl-diethylenetriamine penta-acetic acid (Gd-EOB-DTPA)-enhanced MR imaging. But none of these studies has conducted a unified study on liver, spleen and portal vein. Thus, the aim of this study is to conduct a unified study, investigate whether they can be used to evaluate the liver function in patients with cirrhosis and find out which one is better.

Methods: A total of 120 patients with normal (n = 41), Child–Pugh class A (n = 50), B (n = 21) or C (n = 8) who underwent Gd-EOB-DTPA-enhanced 3 Tesla MR imaging were retrospectively reviewed. Comparison of MRI date (signal intensity of liver parenchyma, portal vein, spleen, and liver-to-portal vein [LPC], liver-to-spleen [LSC], portal vein-to-spleen [PSC] contrast ratio) on hepatobiliary phase 15 min among groups, and liver function parameters was quantitatively analyzed as well.

Results: A significantly difference was observed between the SI of liver parenchyma, LPC and LSC among groups ($P < 0.001$). They all decreased gradually from normal to cirrhotic livers with Child–Pugh class C ($P < 0.001$). The SI of portal vein constantly and slightly increased from normal to Child–Pugh class C, but there were not differences among the groups ($P > 0.05$). LPC is more correlated with Child-Pugh score or MELD score than LSC and liver parenchyma SI. A receiver operating characteristic curve analysis revealed that the AUC in order from the large to the small are: LPC, LSC, and the liver parenchyma SI (0.892, 0.889, 0.836), but the differences of AUCs among them were not significant.

Conclusion: Liver parenchyma SI, LSC and LPC might be used as alternatives imaging biomarker for assessing liver function. Furthermore, LPC values can more efficaciously indicate the severity between patients with cirrhosis than SI of liver parenchyma and LSC. Although the portal vein signal shows a certain upward trend, it could not be used to reflect liver function.

Background

The assessment of liver function is one of the most important issues in patients with cirrhosis. Accurate evaluation can guide clinicians to adopt appropriate treatment programs for these patients and obtain the best prognosis. Because of the advent of special contrast agents, MR imaging can also be used to reflect liver function. Gadolinium-ethoxybenzyl-diethylenetriamine penta-acetic acid (Gd-EOB-DTPA) is a kind of hepatocellular contrast agent. It is easily taken up by hepatocytes and secreted into the biliary system without any change in its chemical structure [1]. It has characteristics of both non-specific extracellular space contrast agent and hepatocyte specific contrast agent [2, 3].

Gd-EOB-DTPA-enhanced MR imaging was not only used for the detection and characterization of liver lesions [4–7], but also for the assessment of hepatic function. Previous studies have evaluated liver function through the Gd-EOB-DTPA-enhanced MR imaging, including biliary tract enhancement [8], the signal intensity ratio of liver with or without reference groups [9, 10], T1 or T2* mapping [2], and dynamic

contrast-enhanced MR imaging [11], etc. However, using the signal intensity ratio to assess the liver function is the simplest and the most convenient method, and more practical for clinical work. The relative enhancement ratio of the liver parenchyma and liver-spleen contrast ratio (LSC) have been widely described [12–14]. Recent study have shown that the liver-to-portal vein contrast ratio(LPC) can indicate the severity of liver function in patients with HBV-related cirrhosis [10]. Previous study has suggested that the delayed hyperintense portal vein sign can potentially be used to reflect hepatobiliary function [15]. And Takatsu concluded LPC was more useful instead of LSC in patients, especially in cases of splenectomy and Gamna–Gandy bodies [16]. But none of these studies has conducted a unified study on them (SI of liver parenchyma, portal vein, spleen, and liver-to-portal vein [LPC], liver-to-spleen [LSC], portal vein-to-spleen [PSC] contrast ratio). Thus, the aim of this study is to conduct a unified study, investigate whether they can be used to evaluate the liver function in patients with cirrhosis and find out which one is better.

Materials And Methods

Patient collective

This retrospective study of existing data was approved by the institutional review board, and the written informed consent was waived.

In the period from November 2017 to October 2019, 761 Gd-EOB-DTPA-enhanced MR imaging examinations were performed. Exclusion criteria included the following: liver function tests were not performed within 1 week before and after MR examination (n = 288); excessive motion artifact or incomplete examination (n = 36); the main portal and its right and left branches were not visualized on MR images typically because of thrombosis or tumor thrombosis (n = 33); splenectomy or diffuse Gamna-Gandy bodies (n = 11); presence of diffuse or massive (d > 10 cm) liver tumor, hemangiomas, or cysts and partial hepatectomy (n = 76); liver dysfunction without cirrhosis (n = 88); various diseases of biliary tract such as cholelithiasis or biliary duct dilatation and kidney failure (n = 109). In total, 120 patients were included in our retrospective study, HBV-related cirrhosis (n = 52), HCV-related cirrhosis (n = 13), alcoholic cirrhosis (n = 5), and schistosoma cirrhosis (n = 9).

Clinical material

Two radiologists separately recorded clinical data of patients, including records of the age, gender, biochemical tests associated with liver function (total bilirubin [TB], aspartate aminotransferase [AST], alanine aminotransferase [ALT], total protein, albumin, creatinine, platelet count, prothrombin time [PT], international normalized ratio [INR]), clinical manifestations (ascites, hepatic encephalopathy), and also got the Child-Pugh score and MELD score. Review the results of the two records and reach an agreement. Note: to ensure the MELD score was positive, we wrote TB and INR values as 1.00 when their values were less than 1.00.

MR imaging technique

All examinations were performed on a 3.0 T magnetic resonance system (Ingenia 3.0 T; Philips), using a 32-channel phased-array coil. Enhanced scanning use a modified Dixon (mDixon) sequence, parameters for mDixon were as follows: repetition time, 3.79 ms; echo time, 1.33 ms; sense factor, 2.0; flip angle, 18°; field of view, 352 × 400 mm; matrix, 268 × 236; reconstruction matrix, 400 × 400; bandwidth, 1260.6 Hz per pixel; scan time, 15 s; thickness, 5 mm. The HBP images were obtained 15 min after Gd-EOB-DTPA administration. Gd-EOB-DTPA (Primovist; Bayer Schering Pharma AG, Berlin, Germany) was used as a hepatocellular contrast agent. All patients received the contrast agent at a rate of 1.0 mL/s (dose = 0.025 mmol/kg body weight). The contrast agent was intravenously administered via a power injector followed by a 25.0 mL saline flush.

Imaging analysis

All examinations were reviewed by two radiologists (not the two above) with 3- and 10-years' experience in abdominal MR imaging, who were blinded to the patient clinical, laboratory, and radiological information. The region-of-interest (ROI) measurement was applied to liver and spleen parenchyma, main, right, and left portal veins on HBP 15 min at a picture archiving and communication system (PACS; Neusoft, Shenyang, China). Each ROI was either a circle. The SI of liver parenchyma was measured at four sections (left lateral, left medial, right anterior, and right posterior). The SI of spleen parenchyma was measured at three evenly distributed sections. In each section of liver and spleen, the ROI (ROI size: 200 mm^2) was manually located by the observers, avoiding visible vessels and biliary ducts, focal lesions, and imaging artifacts. The portal vein ROIs were separately located in the center of the main portal and its right and left branches by contrast to the location of the vessels in the portal phase. LPC was calculated by dividing liver parenchyma SI by portal vein, LSC was calculated by dividing liver parenchyma SI by spleen, and PSC was calculated by dividing portal vein SI by spleen at HBP 15 min SI as follows:

$LPC = [SI_{liver}] / [SI_{Portal\ vein}]$

$LSC = [SI_{liver}] / [SI_{spleen}]$

$PSC = [SI_{Portal\ vein}] / [SI_{spleen}]$

Statistical analysis

Statistical analysis was performed with IBM SPSS Statistics (version 25.0, Chicago, IL). Kolmogorov-Smirnov was used to test the normality of measurement data. The normal distribution data is presented as mean ± standard deviation and the non-normal distribution data is presented as median (interquartile range). One-way ANOVA or the nonparametric Kruskal–Wallis test was used to compare the differences between normal group and Child–Pugh class A, B, and C. One-way ANOVA with LSD or rank sum test with Mann-Whitney U was performed for analysis among groups. Spearman rank correlation coefficients were used to analyze the correlation between hepatic function laboratory markers and MRI date at HBP 15 min. The ROC analysis was used to discriminate between group 1 (normal group and Child–Pugh

class A) and group 2 (Child–Pugh class B and C) at HBP 15 min. All tests were two-sided, and difference of $p < 0.05$ indicated statistically significant.

Results

Clinical data and laboratory examination

All 120 subjects included normal patients (n = 41), and Child–Pugh class A (n = 50), B (n = 21) and C (n = 8). Laboratory parameters and clinical date of patients are shown in Table 1. There were no significant differences in the age, gender, creatinine and mean interval (between MRI and laboratory testing) among the groups. However, distinct differences were identified in all analyzed groups regarding TB, albumin, ALT, AST, PLT, PT, INR, Child–Pugh score, and MELD score ($P < 0.001$).

Table 1

Laboratory texts and clinical information of the patients.

Characteristic	Total	Normal	C–P A	C–P B	C–P C	P value
Sample size	n = 120	n = 41	n = 50	n = 21	n = 8	-
Child–Pugh score	6.0 (5.0–7.0)	-	5.0 (5.0–6.0)	7.0 (7.0–8.0)	12.0 (11.3–12.0)	< 0.001
MELD score [a]	7.41 (6.43–8.52)	6.43 (6.43–6.43)	7.50 (7.12–7.97)	10.04 (8.55–10.50)	16.33 (15.34–16.69)	< 0.001
Mean interval(days) [a]	2.0 (0.3-4)	2.0 (1.0–4.0)	1.0 (0.0-3.3)	2.0 (0.5-3.0)	3.0 (1.3-5.0)	0.177
Age (years) [a]	56.0 (52.0-64.8)	54.0 (49.5–58.5)	60.0 (52.0-64.3)	60.0 (52.5–66.5)	55.0 (45.3–71.5)	0.208
Gender(male/female)	73 / 47	25 / 16	32 / 18	13 / 8	3 / 5	0.566
Standard hepatic function test						
TB (mg/dL) [a]	0.85 (0.60–1.18)	0.54 (0.40–0.65)	0.92 (0.80–1.09)	1.35 (1.13–1.88)	3.45 (3.13–3.95)	< 0.001
Albumin (g/dL) [a]	4.28 (3.90–4.57)	4.60 (4.41–4.71)	4.20 (4.10–4.40)	3.50 (2.96–3.82)	2.61 (2.39–2.64)	< 0.001
INR [a]	1.07 (0.99–1.15)	0.96 (0.95-1.00)	1.09 (1.05–1.12)	1.19 (1.15–1.24)	1.53 (1.47–1.67)	< 0.001
PT [a]	13.9 (12.8–14.6)	12.5 (12.3–12.8)	14.1 (13.8–14.5)	14.9 (14.4–15.8)	18.6 (18.1–19.2)	< 0.001
Serum hepatic enzyme levels						
AST (U/L) [a]	26.0 (18.0–38.0)	17.0 (14.0–21.0)	29.0 (21.0-37.3)	40.0 (34.5–61.0)	87.0 (70.3–92.8)	< 0.001
ALT (U/L) [a]	25.0 (17.0-35.8)	17.0 (11.5–21.0)	27.0 (21.0-37.3)	39.0 (34.0-42.5)	71.0 (63.3–84.3)	< 0.001

MELD, model for end-stage liver disease; TB, total bilirubin; INR, international normalized ratio; PT, Prothrombin time; AST, aspartate transaminase; ALT, alanine transaminase.

[a] For the quantitative analysis, the non-normal distribution data is presented as median (interquartile range).

Characteristic	Total	Normal	C–P A	C–P B	C–P C	P value
Serum renal function levels						
Creatinine (mg/dL) [a]	0.73 (0.67–0.82)	0.71 (0.65–0.77)	0.76 (0.69–0.90)	0.72 (0.64–0.78)	0.76 (0.70–0.95)	0.132
Platelet count [a]	135 (81.0-209.8)	230.0 (182.0-262.0)	124.5 (84.8–143.0)	78.0 (68.5–90.0)	48.5 (36.8–66.8)	< 0.001

MELD, model for end-stage liver disease; TB, total bilirubin; INR, international normalized ratio; PT, Prothrombin time; AST, aspartate transaminase; ALT, alanine transaminase.

[a] For the quantitative analysis, the non-normal distribution data is presented as median (interquartile range).

Differences of MRI data among groups at HBP 15 min

Liver parenchyma SI, LPC and LSC values were significantly different in all groups at HBP 15 min ($p < 0.001$), and they gradually decreased from normal to Child–Pugh C cirrhotic livers. Spleen parenchyma SI, portal vein SI and PSC values were not significantly different in all groups, and portal vein SI constantly and slightly increased from normal to Child–Pugh class C ($p > 0.05$), as shown in Table 2. Hepatobiliary phase images among the groups are shown in Fig. 1.

Table 2

Differences of MRI data among groups at HBP 15 min

	Total	Normal	C–P A	C–P B	C–P C	P value
SI_{liver} [a]	459.2 ± 112.5	528.1 ± 100.3	458.9 ± 98.1	383.7 ± 71.2	312.6 ± 64.5	< 0.001[b]
SI_{spleen} [a]	284.7 ± 70.6	279.1 ± 75.1	279.3 ± 73.6	305.8 ± 58.3	296.3 ± 55.7	0.794
$SI_{portal\ vein}$ [a]	268.3 ± 80.9	254.1 ± 80.9	258.3 ± 79.6	275.5 ± 76.7	298.7 ± 79.3	0.626
LPC [a]	1.80 ± 0.58	2.17 ± 0.50	1.86 ± 0.63	1.34 ± 0.47	0.96 ± 0.13	< 0.001[c]
LSC [a]	1.67 ± 0.47	1.98 ± 0.43	1.69 ± 0.34	1.29 ± 0.32	0.93 ± 0.16	< 0.001[d]
PSC [a]	0.93 ± 0.14	0,93 ± 0.13	0.92 ± 0.12	0.95 ± 0.15	0.99 ± 0.19	0.213

[a] The normal distribution data is presented as mean ± standard deviation.

[b] The statistical difference was found in SI_{liver} at HBP 15 min among all groups ($p < 0.001$) except C–P B and C–P C ($p = 0.023$).

[c] The statistical difference was found in LPC at HBP 15 min among all groups ($p < 0.001$) except C–P B and C–P C ($p = 0.047$).

[d] The statistical difference was found in LSC at HBP 15 min among all groups ($p < 0.001$) except C–P B and C–P C ($p = 0.020$).

The correlation between laboratory markers and MRI date at hepatobiliary phase 15 min

The correlations between laboratory markers and MRI date at HBP 15 min are summarized in Table 3. TB, albumin, PT, INR, PLT, ALT, AST, Child–Pugh score, and MELD score were significantly correlated with liver parenchyma SI, LPC and LSC. And there a strong correlation was observed between LPC and LSC with respect to all groups, as you can see in Fig. 2.

Table 3

The correlation among clinical parameters, LPC or LSC in cirrhosis groups at hepatobiliary phase 15 min.

Laboratory indexes	Correlation coefficient			P value
	LPC	LSC	SI_{liver}	
TB	-0.577	-0.613	-0.522	< 0.001
Albumin	0.565	0.623	0.479	< 0.001
ALT	-0.426	-0.455	-0.492	< 0.001
AST	-0.477	-0.512	-0.506	< 0.001
INR	-0.641	-0.646	-0.553	< 0.001
PT	-0.579	-0.576	-0.524	< 0.001
Creatinine	-0.090	-0.139	0.024	> 0.050
Platelet count	0.464	0.467	0.518	< 0.001
Child-Pugh score	-0.576	-0.569	-0.562	< 0.001
MELD score	-0.632	-0.580	-0.526	< 0.001

TB, total bilirubin; ALT, alanine transaminase; AST, aspartate transaminase; INR, international normalized ratio; PT, Prothrombin time; MELD, model for end-stage liver disease.

ROC analysis

The ROC curve analysis revealed the optimal cutoff value for LPC to distinguish group 1 (normal group and Child–Pugh class A) from group 2 (Child–Pugh class B and C) was 1.20 (AUC 0.892) with a sensitivity of 98.9% and a specificity of 69.0%. The optimal cutoff value for LSC to distinguish group 1 from group 2 was 1.27 (AUC 0.889) with a sensitivity of 95.6% and a specificity of 72.4%. The optimal cutoff value for liver parenchyma SI to distinguish group 1 from group 2 was 405.4 (AUC 0.836) with a sensitivity of 81.3% and a specificity of 75.9%. (Fig. 3). The differences of AUCs among LPC, LSC and the liver parenchyma SI were not significant ($p > 0.05$). The reason for this grouping was due to the cirrhosis patients with Child–Pugh class B or C contraindications for operation studies [17, 18].

Discussion

As we all know, cirrhosis can cause damage to liver cells, increased spleen volume, and portal hypertension and so on, so we mainly involved the liver, spleen and portal vein. Our research was conducted at the HBP 15 min, we believed that this period could meet the needs for diagnosis of liver diseases and shorten the examination time of patients.

The signal intensity of liver parenchyma, spleen and portal vein

Liver parenchyma SI can be used to estimate liver function has been widely described, the way it works: the hepatobiliary phase of Gd-EOB-DTPA was made by the selective uptake of membrane-bound organic anion transporters (OATP1 B1/B3) [19–21]. Normal hepatocytes could use these transporters to ingest Gd-EOB-DTPA and the amount of Gd-EOB-DTPA reached the peak on the HBP 20 min; the impaired amount and functional capacity of these transporters could reduce the uptake of Gd-EOB-DTPA into hepatocytes [22], subsequently affected the liver signal. Our data shown that liver parenchyma SI gradually decreases with the increase of liver function damage, Previous studies [22–24] have also proved that the severity of cirrhosis can significantly affect the absorption of gadolinium, and then affect the degree of liver enhancement, which was consistent with our research.

The spleen does not contain hepatocytes, Gd-EOB-DTPA only shows the characteristics of non-specific extracellular space contrast agent. Our research indicates that the SI of spleen cannot reflect liver function and the mean value of spleen signals are equally likely in each group. In addition, we found most of the cases in all our groups, the spleen signal increased gradually from inside to outside for MR images whether pro-enhanced or HBP 15 min (Fig. 4), leading to an increase in the mean signal value of the spleen. We don't know how to explain this phenomenon, it may be related to the uneven magnetic field or the hemodynamics of the spleen.

In our study, portal vein SI constantly and slightly increased from normal to Child–Pugh class C, but there was no difference among groups, Zhang reported that LPC can efficaciously indicate the severity of liver function [10], their data on portal vein SI is similar to our research. Previous one has suggested that the delayed hyperintense portal vein sign can potentially be used to reflect hepatobiliary function [15], their subjects are mostly patients with extrahepatic cholestasis, we think that's the main reason for the difference. Study proved hepatic uptake and biliary elimination of bilirubin compete against Gd-EOB-DTPA, hyperbilirubinemia will lead to decreased absorption and clearance of Gd-EOB-DTPA, it also leads to delayed the contrast agent to stay in the blood for longer [25]. However, we hold that the bilirubin level in patients with cirrhosis may not be as high as that in patients with extrahepatic cholestasis, and hepatocytes may be able to meet this competition in patients with cirrhosis.

LPC, LSC and PSC

Different from enhanced CT, the signal intensity of MRI enhancement has a nonlinear relationship with the concentration of contrast agent, most studies used reference tissue (spleen) to correct the liver signal [12–14]. As we have seen, only one literature has studied the relationship between LPC and LSC [16], their results showed that LPC was strongly correlated with LSC, and LPC of each group was lower than LSC, They owed it to the portal vein SI, which can represent a more consistent reflection of the blood pool than that of the spleen. Our research also shows a strong correlation between LPC and LSC among groups (Fig. 2), but LPC was greater than LSC. The reasons for this difference may be as follow: (1) The different

causes might lead to the different patterns of uptake and excretion of Gd-EOB-DTPA. Our patients are mainly hepatitis B cirrhosis, their patients are mainly chronic liver disease; (2) The MRI device and imaging sequence are different.

As we all known, no one has studied PSC yet, our research suggests that PSC cannot reflect liver function in patients with cirrhosis. As discussed before, portal vein SI constantly and slightly increased from normal to Child–Pugh class C, but there was no difference among groups, and the mean value of spleen signals are equally likely in each group. It's possible that there's no difference in portal vein-to-spleen (PSC) among groups.

The correlation between laboratory markers and MRI date

Some studies used ICG to reflect liver function, because there was a direct correlation between ICG clearance and hepatocytes, which can provide more complete information on liver uptake and excretion function [26–28]. We did not carry out this test because of operational difficulties. we quantitatively analyzed the correlations between MRI date and the liver function parameters. In this study, the liver parenchyma SI, LPC and LSC were weak to moderate correlated with laboratory markers. Zhang also demonstrated that weak to moderate correlation was observed between LPC and laboratory markers [10], which was consistent with our research. We also found that the liver parenchyma SI, LPC and LSC were negatively correlated with hepatic function scoring systems (Child–Pugh score and MELD score), the correlation coefficient in order from the large to the small both are: LPC, LSC, and the liver parenchyma SI, the reason may be that the changing trend of portal vein signal makes LPC have a higher correlation with liver function.

Receiver operating characteristic analysis showed that the AUC in order from the large to the small are: LPC, LSC, and the liver parenchyma SI (0.892, 0.889, 0.836), but the difference of AUCs among LPC, LSC and the liver parenchyma SI were not significant. This illustrates that they have the same ability to identify group 1 and group 2.

These results suggested that the LPC might be a more useful alternative imaging biomarker for the evaluation of liver function than LSC and the liver parenchyma SI when the liver function test and hepatic function scoring systems is not readily accessible at the time of reviewing images. Takatsu found that LPC could be used as a substitute for LSC for a simple assessment of degree of hepatic contrast enhancement [16], this is consistent with our research. In addition, they also believed that LPC can be especially useful in cases of splenectomy and Gamna–Gandy bodies [16]. And yet, we thought this conclusion needed further verification because of few patients with splenectomy (n = 6), Gamna–Gandy bodies (n = 7), and these patients were not graded for liver function.

However, our study had several limitations. Firstly, the severity of cirrhosis was not grouped by liver biopsy. Secondly, we did not classify the causes of cirrhosis such as viral hepatitis, alcohol abuse, and so on. The different causes might lead to the different patterns of uptake and excretion of Gd-EOB-DTPA.

Last, it was difficult to avoid selection bias because of the retrospective nature of this study. Taken together, further prospective and multi-center studies are needed.

Conclusion

The liver function can be assessed and classified using LPC, LSC and the liver parenchyma SI obtained in the hepatobiliary phase with Gd-EOB-DTPA-enhanced MR imaging, the LPC might be a more useful imaging biomarker for the evaluation of liver function compared with LSC and the liver parenchyma SI. In addition, the portal vein SI in each group showed a certain increasing trend, it was unbale to be used to reflect the liver function of patients with cirrhosis. But it is precisely because of the changing trend of portal vein signal that LPC can better reflect the liver function of patients with cirrhosis.

Abbreviations

Gd-EOB-DTPA:gadolinium-ethoxybenzyl-diethylenetriamine penta-acetic acid; MRI:magnetic resonance imaging; LPC:liver-to-portal vein contrast ratio; LSC:liver-to-spleen contrast ratio; PSC:portal vein-to-spleen contrast ratio; SI:signal intensity; AUC:area under curve; ROI:region-of-interest; HBP:hepatobiliary phase; MELD:model for end-stage liver disease; TB:total bilirubin; INR:international normalized ratio; PT:Prothrombin time; AST:aspartate transaminase; ALT:alanine transaminase.

Declarations

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Authors’contributions

MY and YZ proposed the method, analyzed data and wrote the manuscript. WZ and SG manually plotted all the ROIs, and participated in manuscript revisions, and provided critical review that helped in improving the manuscript. WC and HW collected clinical data and laboratory tests from all patients. All authors read and approved the final manuscript.

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Availability of data and materials

Data related to the current study are available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests.

Ethics approval and consent to participate

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards. For this type of study formal consent is not required.

Consent for publication

Not applicable.

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Figures

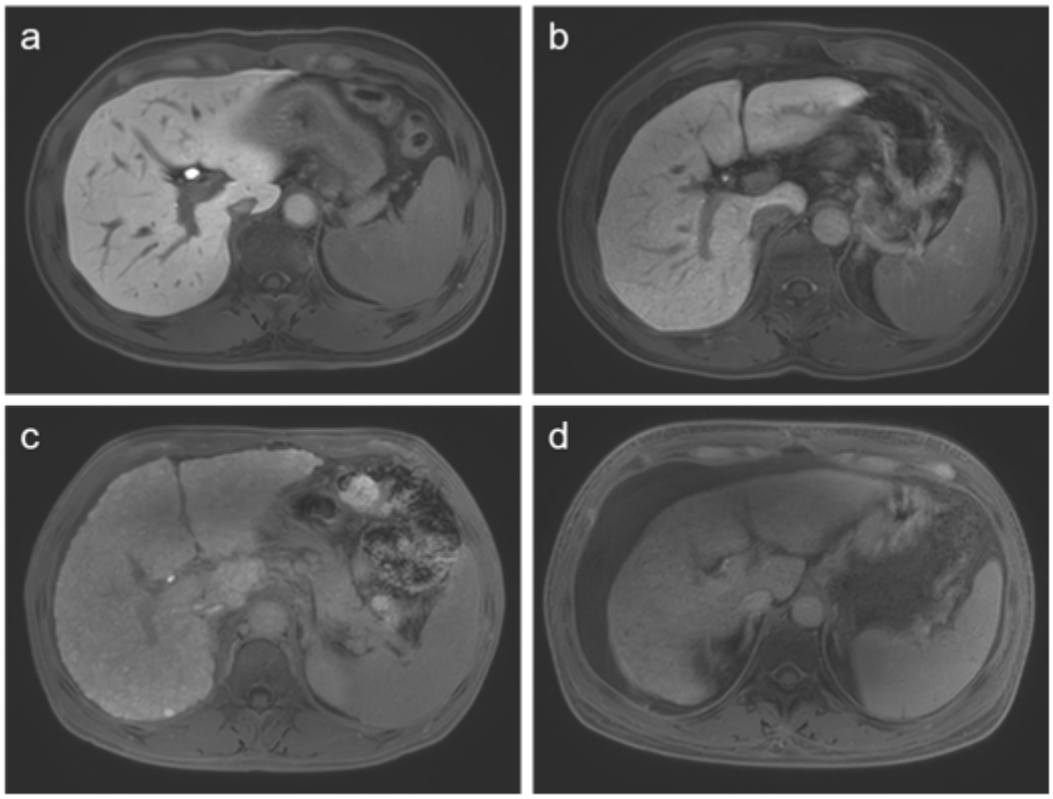

Figure 1

Hepatobiliary phase at 15 min images among the groups: (a) normal, (b) Child–Pugh A, (c) Child–Pugh B, (d) Child–Pugh C.

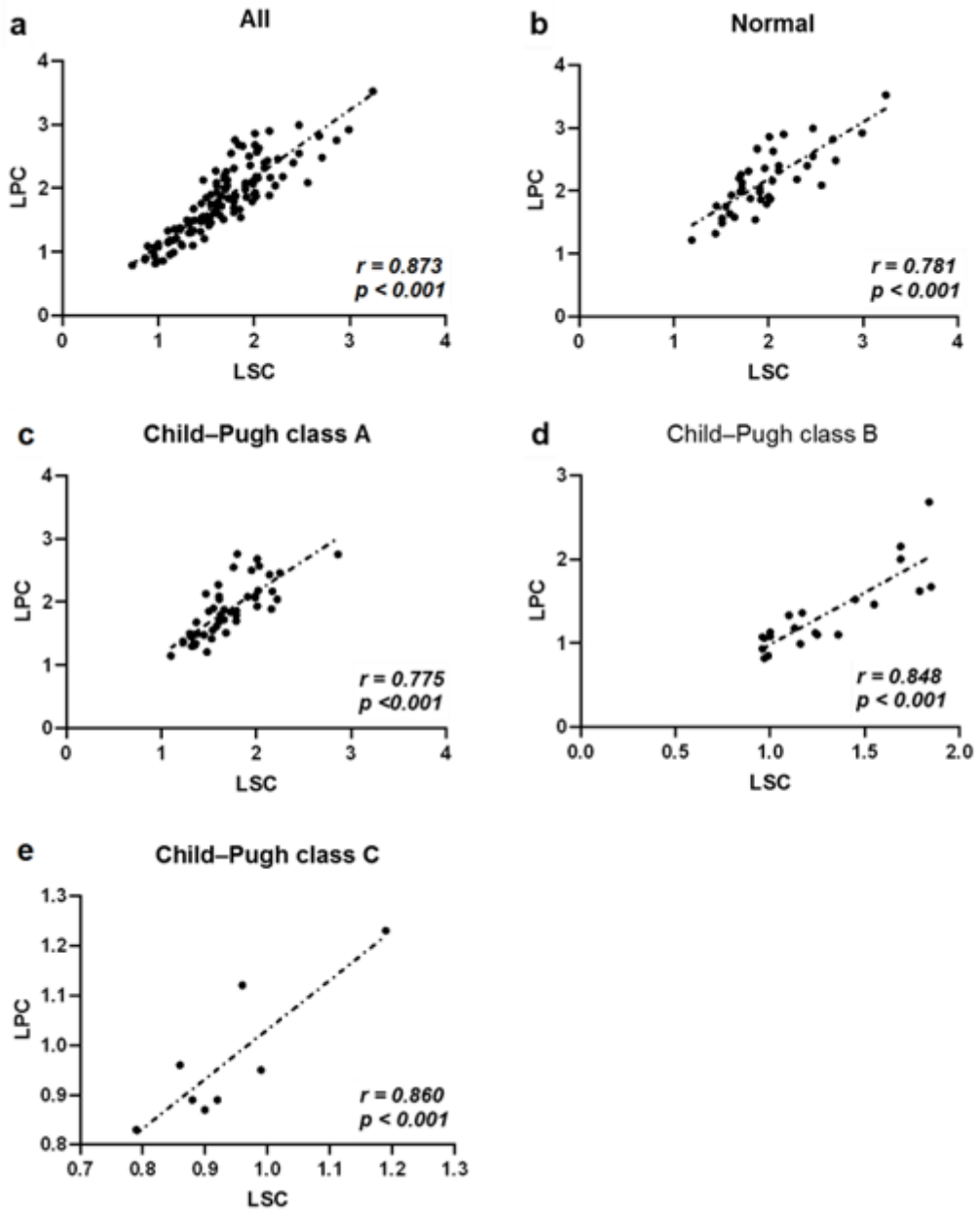

Figure 2

Correlations between LSC and LPC among the groups: (a) overall, (b) normal, (c) C–P A, (d) C–P B, and (e) C–P C. These correlations were strongly positive.

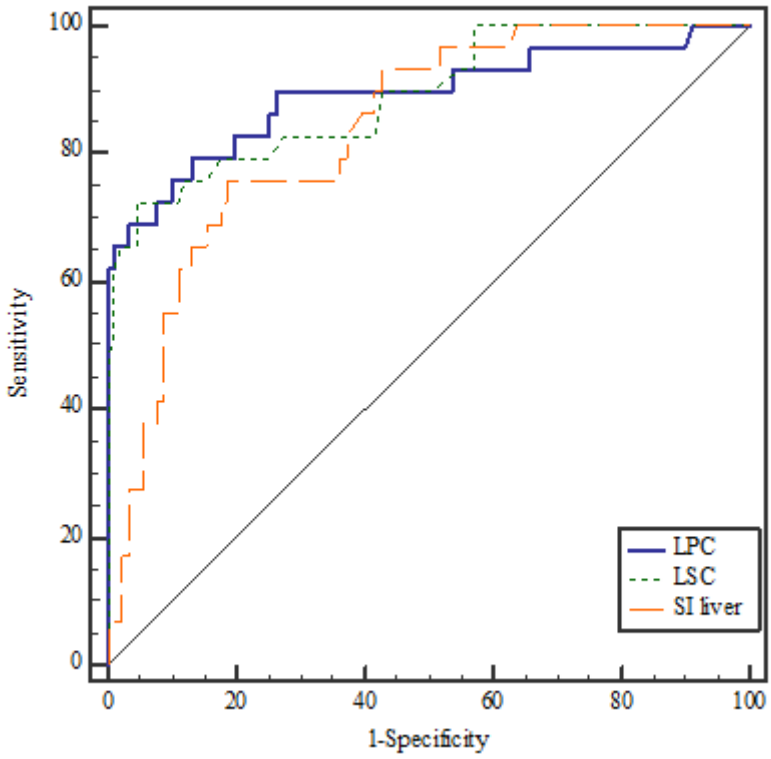

Figure 3

The ROC curve showed that the AUC value of LPC was 0.892 (95%CI 0.822 – 0.941), the AUC value of LSC was 0.889 (95%CI 0.818 – 0.939), and the AUC value of the liver parenchyma SI was 0.836 (95%CI 0.758 – 0.898).

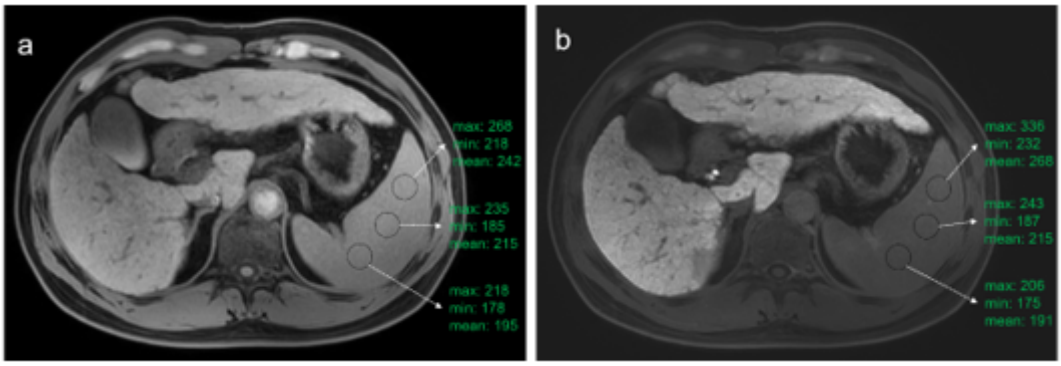

Figure 4

A case of liver cirrhosis with uneven spleen signal: (a) pro-enhanced image (b) hepatobiliary phase at 15 min image. ROI size: 200 mm × mm.