

Original paper

Association of liver function with hepatic and renal parenchymal signal intensities during the hepatobiliary phase of Gd-EOB-DTPA-enhanced MRI

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Abstract

Purpose: To investigate the association between hepatic function and liver and renal parenchymal signal intensities (SI) during the hepatobiliary phase of gadoxetic acid (Gd-EOB-DTPA)-enhanced magnetic resonance imaging (MRI), and to provide imaging evidence for the combined evaluation of hepatic and renal function in cirrhotic patients.

Material and methods: Seventy-eight patients who underwent Gd-EOB-DTPA-enhanced MRI were retrospectively enrolled and divided into a normal control group ($n = 28$), Child-Pugh grade A group ($n = 25$), grade B group ($n = 17$), and grade C group ($n = 8$). Pre-enhancement SI of the liver and renal cortex/medulla ($SI_{liver-pre}$, $SI_{cortex-pre}$, $SI_{medulla-pre}$) and post-enhancement SI ($SI_{liver-post}$, $SI_{cortex-post}$, $SI_{medulla-post}$) were measured. The signal intensity differences between the renal cortex and medulla ($SI_{[cortex-medulla]pre}$ and $SI_{[cortex-medulla]post}$) were calculated. Relative enhancement rates (RE) were calculated. One-way analysis of variance (ANOVA), Kruskal-Wallis, and Mann-Whitney U tests were used to compare signal intensity parameters among the normal liver function group and different Child-Pugh grade groups (A, B, C).

Results: As the Child-Pugh grade increased, $SI_{liver-pre}$, $SI_{liver-post}$, and RE_{liver} showed statistically significant differences ($p < 0.05$). $SI_{cortex-pre}$ and $SI_{cortex-post}$ exhibited significant differences in subgroup comparisons ($p < 0.05$), but RE_{cortex} showed no statistical significance ($p = 0.859$). Pre- and post-enhancement renal medullary signals and $SI_{[cortex-medulla]}$ differences showed no significant variations across all groups ($p > 0.05$).

Conclusions: Hepatic dysfunction was associated with lower hepatic signal intensity and enhancement in the hepatobiliary phase. Significant differences were observed in the renal cortical signal intensity before and after contrast administration, whereas the renal medullary signals and corticomedullary signal differences showed no significant variation.

Key words: gadoxetic acid disodium (Gd-EOB-DTPA), renal cortex and medulla, signal intensity parameters, hepatic function, hepatobiliary phase.

Introduction

Gadoxetic acid (Gd-EOB-DTPA), a hepatobiliary-specific magnetic resonance imaging (MRI) contrast agent, has been widely applied in the detection of hepatic lesions, assessment of liver function, and diagnosis of biliary disease. It has also demonstrated potential value in the detection and diagnosis of extrahepatic organ diseases such as renal disorders [1-3]. However, hepatocellular uptake and

excretion of Gd-EOB-DTPA are significantly reduced in cases of impaired liver function, leading to decreased biliary excretion [4]. Since the excretion of Gd-EOB-DTPA depends primarily on both biliary and renal pathways, impaired liver function may not only affect hepatic uptake and excretion but may also indirectly influence the renal signal characteristics [5]. However, the specific impact of liver dysfunction on renal parenchymal enhancement, particularly in the cortex and medulla, remains unclear.

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Authors' contribution:

A Study design · B Data collection · C Statistical analysis · D Data interpretation · E Manuscript preparation · F Literature search · G Funds collection

Therefore, the present study focused on renal parenchymal signal alterations during the hepatobiliary phase of Gd-EOB-DTPA-enhanced MRI in patients with liver cirrhosis, aiming to provide new imaging evidence for clinical diagnosis and functional evaluation.

Material and methods

Study population

Patients who underwent Gd-EOB-DTPA-enhanced MRI at our institution between June 2022 and June 2023 were retrospectively reviewed. The inclusion and exclusion process followed predefined eligibility criteria (Figure 1). Patients with cirrhosis were classified according to Child-Pugh grade, whereas the control group comprised individuals evaluated during the same period who demonstrated normal liver function, no documented chronic liver disease, and no biochemical evidence of renal dysfunction.

MRI protocol

All patients underwent MRI examinations on a GE Signa Premier 3.0 T scanner, with coverage extending from the dome of the diaphragm to the inferior pole of the kidney. Gd-EOB-DTPA was administered via a peripheral intravenous bolus at a dose of 0.025 mmol/kg. The imaging protocol included T2-weighted imaging (T2WI), T1-weighted imaging with fat suppression (T1WI-FS), and contrast-enhanced T1WI-FS. Post-contrast images were acquired at 15 s (arterial phase), 45 s (portal venous phase), 50 s (transitional phase), and 20 min (hepatobiliary phase) after Gd-EOB-DTPA injection.

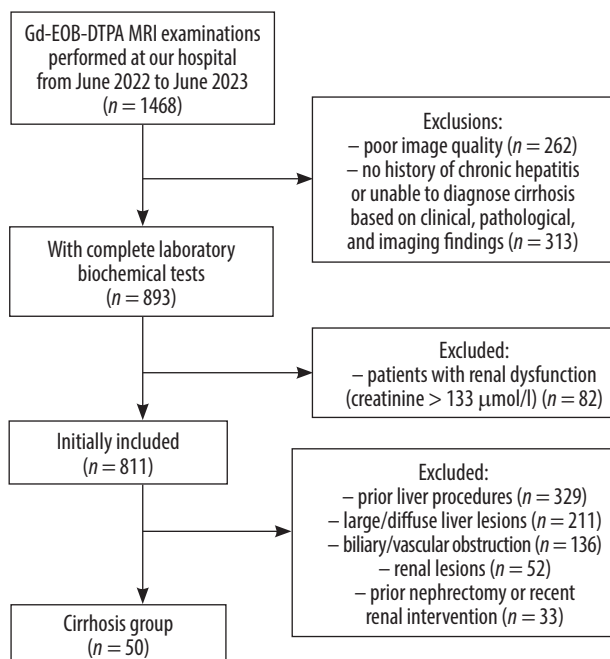


Figure 1. Flowchart of the enrollment of patients with liver cirrhosis in the study cohort

Signal intensity measurement

The largest cross-section of the liver was selected on pre- and post-contrast hepatobiliary phase axial T1-weighted images (T1WI). Regions of interest (ROIs) with an area of approximately 1 cm² were placed in the left lateral lobe, left medial lobe, right anterior lobe, and right posterior lobe of the liver, and the mean signal intensity of these ROIs was recorded as the pre-contrast and post-contrast liver signal intensities (SI).

Similarly, the SI parameters of the renal cortex and medulla were measured on pre- and post-contrast hepatobiliary phase images. ROIs with an area of approximately 20–40 mm² were placed in both renal cortices and medullae (Figure 2), with positioning guided by unenhanced and corticomedullary phase images. The mean signal intensity of each ROI was calculated.

The pre-contrast signal intensities of the liver, renal cortex, and renal medulla were denoted as SI_{liver-pre}, SI_{cortex-pre}, and SI_{medulla-pre}, respectively, while post-contrast values were denoted as SI_{liver-post}, SI_{cortex-post}, and SI_{medulla-post}. The relative enhancement (RE) of the liver was calculated as $RE_{liver} = (SI_{liver-post} - SI_{liver-pre}) / SI_{liver-pre}$. Similarly, the relative enhancement of the renal cortex and medulla was calculated as RE_{cortex} and $RE_{medulla}$. In addition, the difference in signal intensity between the renal cortex and medulla was calculated as $SI_{(cortex-medulla)pre} = SI_{cortex-pre} - SI_{medulla-pre}$ and $SI_{(cortex-medulla)post} = SI_{cortex-post} - SI_{medulla-post}$. $RE_{(cortex-medulla)} = [SI_{(cortex-medulla)post} - SI_{(cortex-medulla)pre}] / SI_{(cortex-medulla)pre}$.

Statistical analysis

All statistical analyses were performed using SPSS version 26.0. The normality of the continuous data was assessed using the Shapiro-Wilk test. Continuous variables are presented as mean ± standard deviation (SD), and categorical variables are expressed as frequency (n) and percentage (%). For normally distributed data, comparisons between groups were performed using independent-

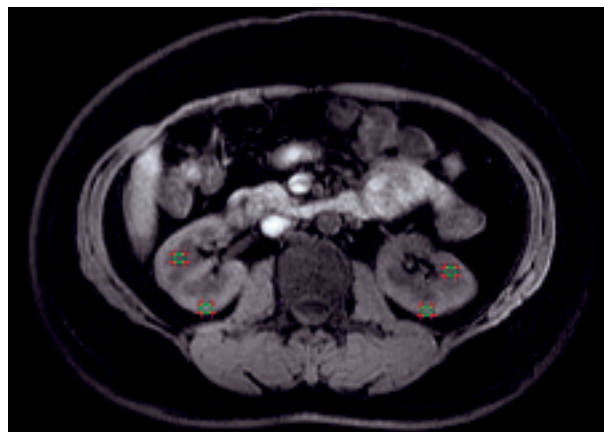


Figure 2. Measurement of renal cortical and medullary signal intensities on unenhanced images. Measurements during the hepatobiliary phase were performed in the same manner as for the unenhanced images

samples *t*-tests or one-way analysis of variance (ANOVA). For non-normally distributed data, the Kruskal-Wallis test or Mann-Whitney *U* test was applied, as appropriate. A two-sided *p*-value of < 0.05 was considered statistically significant.

Results

Comparison of baseline characteristics

A total of 78 participants were included in this study, comprising 28 in the normal control group, 25 in the Child-Pugh grade A group, 17 in the Child-Pugh grade B group, and 8 in the Child-Pugh grade C group. A comparison of the baseline characteristics among the groups showed that, except for age, sex, serum creatinine, and blood urea nitrogen, all other parameters differed significantly between the groups (Table 1).

Pre- and post-contrast liver signal intensities

Significant differences were observed in $SI_{liver-pre}$, $SI_{liver-post}$, and RE_{liver} among the normal control group and the different Child-Pugh grades. Pairwise comparisons showed that $SI_{liver-pre}$ differed significantly between the normal control and Child-Pugh grade C groups. For $SI_{liver-post}$ and RE_{liver} , significant differences were observed between the normal control and grade B groups, the normal control and grade C groups, the grade A and grade

B groups, and the grade A and grade C groups (Table 2, Figures 3-4).

Renal signal intensities before and after contrast enhancement

Renal cortical signal intensities

Significant differences in $SI_{cortex-pre}$ and $SI_{cortex-post}$ were observed among the normal control group and the different Child-Pugh grades. Pairwise comparisons showed that $SI_{cortex-pre}$ did not differ significantly between any groups, whereas $SI_{cortex-post}$ differed significantly between the Grade A and B groups. No significant differences were found in RE_{cortex} among the normal control group and the different Child-Pugh grades (Table 3 and Figure 5).

Renal medullary signal intensities

No significant differences were observed in $SI_{medulla-pre}$, $SI_{medulla-post}$, or $RE_{medulla}$ among the normal control group and the different Child-Pugh grades (Table 4 and Figure 6).

Corticomedullary signal differences before and after contrast enhancement

Analysis showed that the three signal parameters did not differ significantly between the normal control group and the different Child-Pugh grades (Table 5).

Table 1. Baseline clinical characteristics of participants in each group

Parameter	Normal	Child-Pugh grade A	Child-Pugh grade B	Child-Pugh grade C	Test statistic	<i>p</i> -value
Age (years)	46.90 ± 16.16	53.05 ± 11.26	55.50 ± 10.09	57.89 ± 13.14	5.09	0.065
Sex (M/F)	15/13	10/15	8/9	7/1	3.68	0.298
ALB (g/l)	43.21 ± 3.50	37.85 ± 6.70	30.66 ± 4.37	30.13 ± 2.34	49.03	0.000*
TBIL (μmol/l)	21.03 ± 18.60	20.41 ± 10.51	74.74 ± 105.58	70.92 ± 47.98	38.62	0.000*
PT (s)	11.62 ± 1.14	13.78 ± 1.85	15.46 ± 1.65	17.6 ± 2.6	50.90	0.000*
INR	0.96 ± 0.08	1.13 ± 0.18	1.28 ± 0.14	1.51 ± 0.26	52.20	0.000*
Cr (μmol/l)	74.24 ± 24.75	69.05 ± 16.13	60.58 ± 12.58	66 ± 13.86	6.80	0.079
BUN (mmol/l)	4.86 ± 1.51	5.26 ± 1.67	4.81 ± 1.53	3.97 ± 0.85	5.20	0.158

*Indicates a statistically significant difference.

Table 2. Liver signal intensities before and after contrast enhancement in each group

Signal intensities	Normal	Child-Pugh grade A	Child-Pugh grade B	Child-Pugh grade C	Test statistic	<i>p</i> -value
$S_{liver-pre}$	697.33 ± 183.30	670.84 ± 297.68	558.60 ± 269.97	467.22 ± 221.68	14.14	0.003*
$SI_{liver-post}$	2194.16 ± 604.99	1793.47 ± 463.54	1060.94 ± 361.35	703.97 ± 370.13	43.63	0.000*
RE_{liver}	2.30 ± 1.02	2.09 ± 1.32	1.01 ± 0.61	0.45 ± 0.43	25.94	0.000*

*Indicates a statistically significant difference.

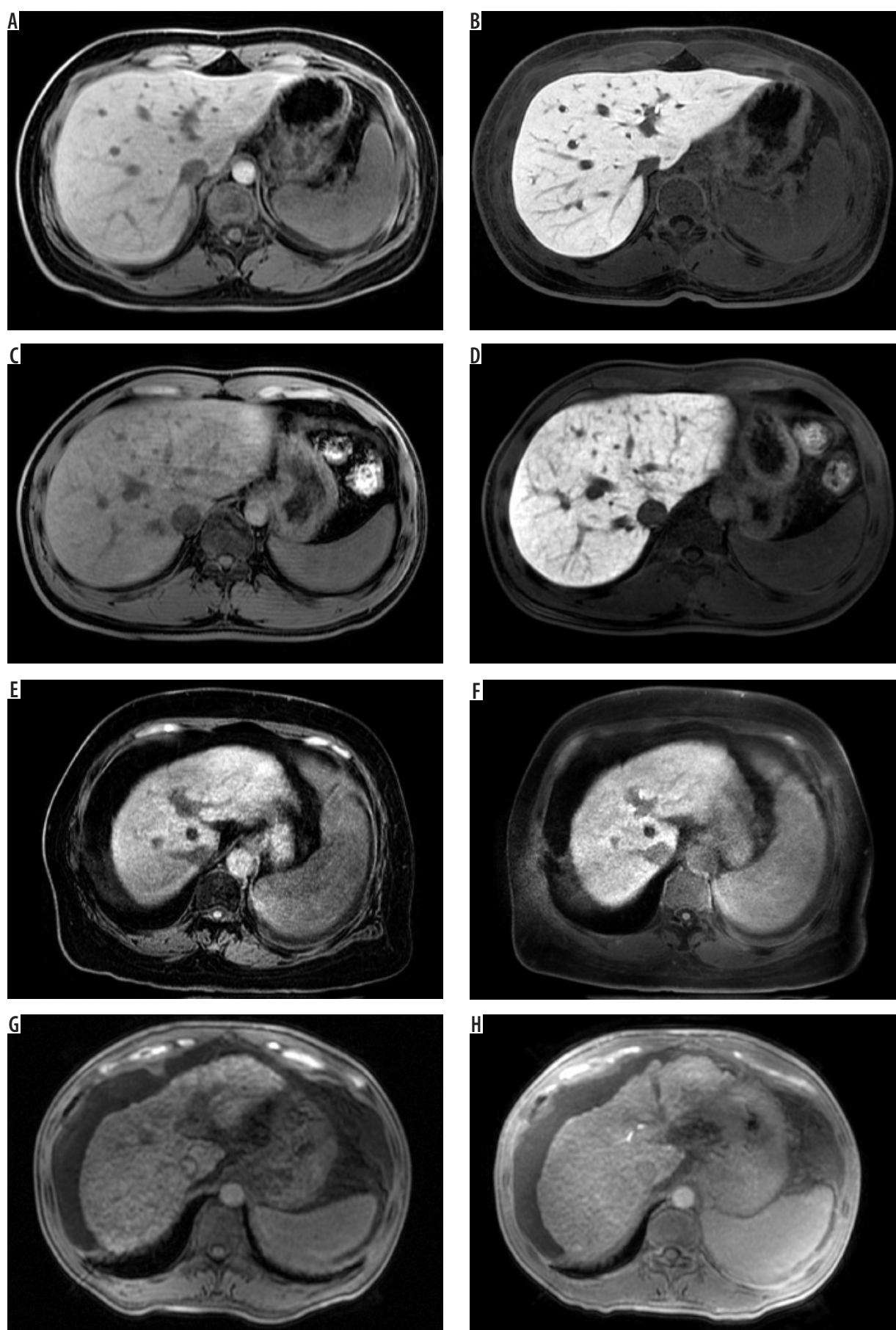


Figure 3. Liver images of the normal control, Child-Pugh grade A, B, and C groups under identical window settings. Unenhanced images are shown in panels A, C, E, and G; hepatobiliary phase images are shown in panels B, D, F and H

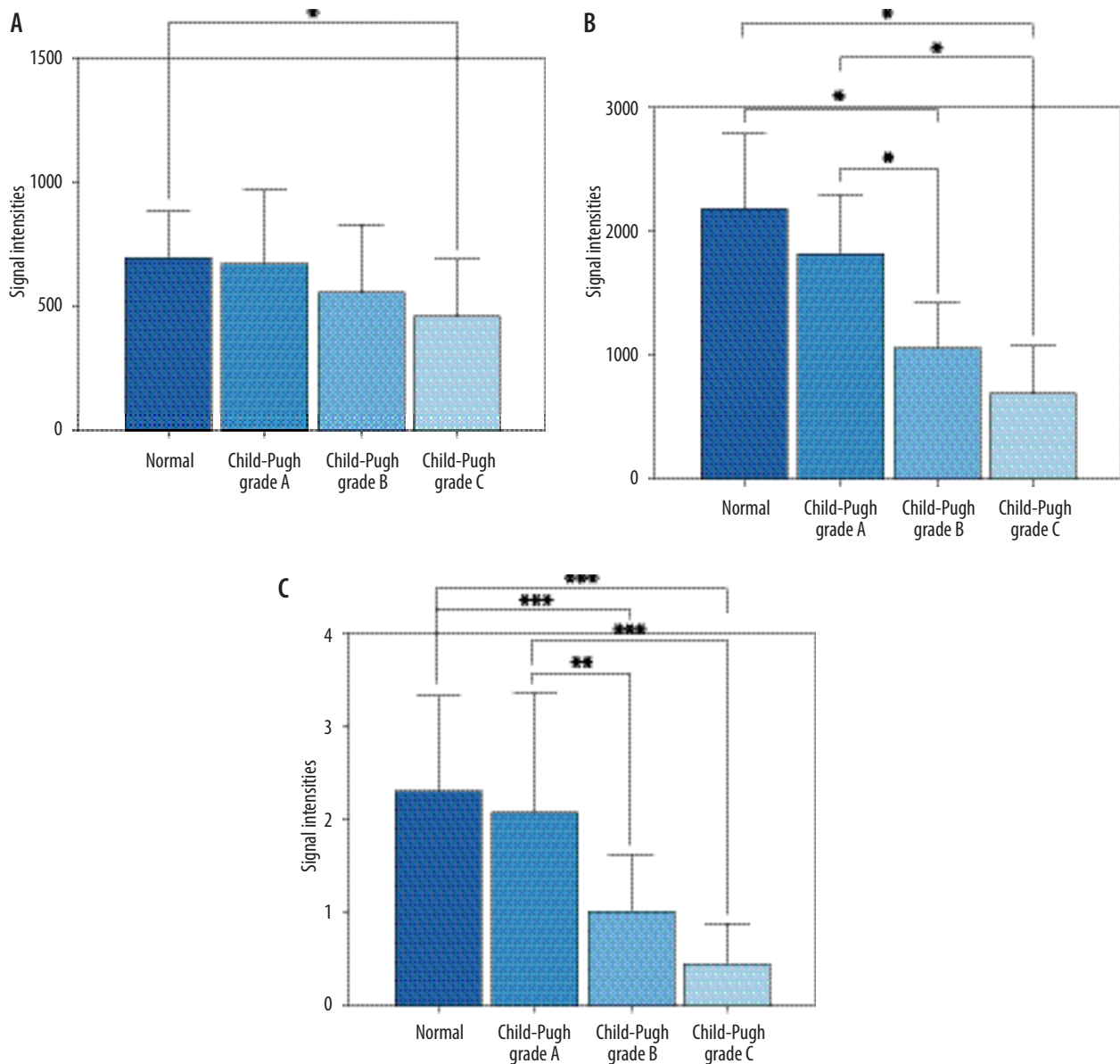


Figure 4. Comparisons of hepatic signal intensity parameters before and after contrast enhancement among groups (* $p < 0.05$, **~*** $p < 0.01$).

A) $SI_{liver-pre}$; B) $SI_{liver-post}$; C) RE_{liver}

Discussion

Effects of liver function on hepatic signal intensities

Studies have shown that as liver function deteriorates from Child-Pugh grade A to C, hepatic signal intensity on T1-weighted imaging ($SI_{liver-pre}$) gradually decreases. This reduction is closely associated with the pathological changes of cirrhosis, where normal hepatocytes are progressively replaced by fibrous tissue and regenerative nodules [6]. Both fibrous proliferation, which prolongs the T1 relaxation time, and the formation of iron-containing nodules contribute to the decreased T1WI signal. Similarly, $SI_{liver-post}$ also showed a decreasing trend, consistent with previous studies [7], primarily due to the impaired function of organic anion-transporting polypeptides

(OATPs) on hepatocyte membranes in cirrhosis [8,9], resulting in reduced Gd-EOB-DTPA uptake [10]. Moreover, fibrotic tissue lacks the capacity to metabolize contrast agents, further contributing to the decline in $SI_{liver-post}$. Beyond transporter dysfunction, additional structural and hemodynamic factors may also contribute. Reduced hepatobiliary phase signal in cirrhosis may not only reflect impaired transporter activity but also decreased viable hepatocyte mass, microstructural alterations, and intrahepatic shunting, all of which may reduce the effective uptake of Gd-EOB-DTPA [11]. In addition, RE_{liver} gradually decreased with worsening cirrhosis, in agreement with the findings of Haimerl *et al.* [2] This is attributed to the combined effects of reduced $SI_{liver-pre}$ and impaired Gd-EOB-DTPA uptake and excretion, leading to diminished enhancement in $SI_{liver-post}$.

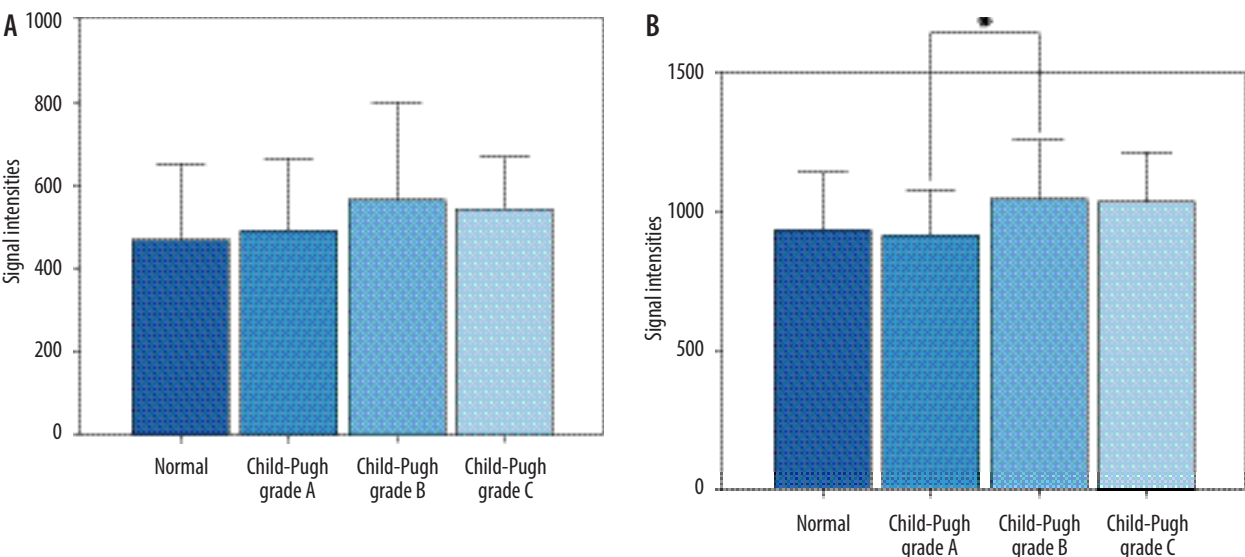


Figure 5. Comparison of renal cortical signal intensities before and after contrast enhancement among groups ($p < 0.05$)

Table 3. Renal cortical signal intensities before and after contrast enhancement in each group

Signal intensities	Normal	Child-Pugh grade A	Child-Pugh grade B	Child-Pugh grade C	Test statistic	<i>p</i> -value
SI _{cortex-pre}	472.83 ± 175.40	494.76 ± 173.08	569.86 ± 230.30	545.43 ± 123.72	8.45	0.038*
SI _{cortex-post}	943.63 ± 200.66	920.76 ± 166.51	1057.39 ± 200.43	1039.50 ± 177.63	8.28	0.041*
RE _{cortex}	1.18 ± 0.778	1.01 ± 0.60	1.04 ± 0.68	0.98 ± 0.51	0.76	0.859

*Indicates a statistically significant difference.

Table 4. Renal medullary signal intensities before and after contrast enhancement in each group

Signal intensities	Normal	Child-Pugh grade A	Child-Pugh grade B	Child-Pugh grade C	Test statistic	<i>p</i> -value
SI _{medulla-pre}	425.35 ± 141.80	407.71 ± 159.92	463.18 ± 168.45	433.76 ± 106.82	3.49	0.323
SI _{medulla-post}	995.24 ± 237.48	954.54 ± 189.59	1056.72 ± 193.75	1067.25 ± 186.30	6.65	0.084
RE _{medulla}	1.53 ± 0.81	1.59 ± 0.89	1.48 ± 0.81	1.57 ± 0.71	0.11	0.990

Note: * indicates a statistically significant difference.

Table 5. Comparison of corticomedullary signal differences before and after contrast enhancement among different liver function groups

Signal intensities	Normal	Child-Pugh grade A	Child-Pugh grade B	Child-Pugh grade C	Test statistic	<i>p</i> -value
SI _{(cortex-medulla)pre}	47.48 ± 113.99	89.36 ± 42.60	106.67 ± 75.90	111.67 ± 58.05	7.76	0.051
SI _{(cortex-medulla)post}	-51.61 ± 133.69	-31.98 ± 81.16	0.68 ± 91.73	-27.75 ± 96.17	1.93	0.586
RE _(cortex-medulla)	-0.771 ± 4.802	-1.352 ± 1.482	-0.214 ± 3.501	-1.823 ± 2.121	0.77	0.855

*Indicates a statistically significant difference.

Renal signal findings and mechanistic interpretation

The renal elimination of Gd-EOB-DTPA differs from hepatocellular uptake. Hepatic enhancement depends on active transporter mechanisms, such as OATPs and MRP2, whereas renal excretion primarily occurs through glomerular filtration. Therefore, renal and hepatic enhancement kinetics are not directly compar-

able, and the 20-minute hepatobiliary phase may not represent the peak renal enhancement. The present study did not aim to evaluate dynamic renal excretion but to determine whether progressive hepatic dysfunction is associated with measurable changes in renal parenchymal signal intensity, including both cortical and medullary regions, during the later phase of contrast distribution.

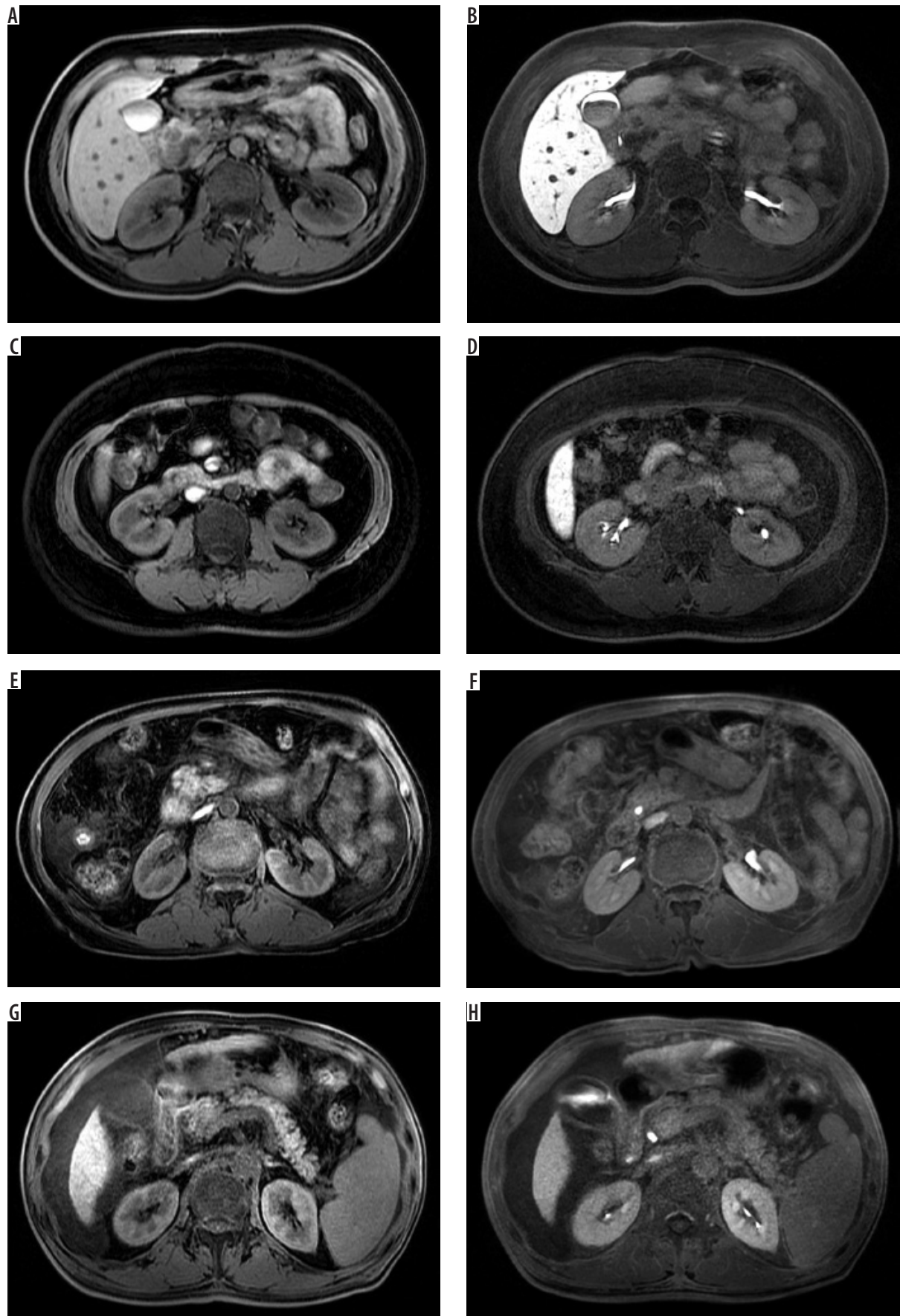


Figure 6. Renal images of each group under identical window width and level settings on unenhanced and hepatobiliary phase scans. **A, C, E, G)** Unenhanced images of the normal control, grade A, grade B, and grade C groups, respectively; **B, D, F, H)** Hepatobiliary phase images of the normal control, grade A, grade B, and grade C groups, respectively

Effects of liver function on renal cortical signal intensities

In this study, $SI_{\text{cortex-pre}}$ differed significantly among the normal control group and the various Child-Pugh grades ($p < 0.05$); however, pairwise comparisons did not reach statistical significance. This may be related to the conservative nature of pairwise analyses and the relatively small sample size, which warrants further validation with larger cohorts or alternative analytical approaches in future studies.

Theoretically, as liver function deteriorates, the biliary excretion of Gd-EOB-DTPA during the hepatobiliary phase may decrease, potentially resulting in increased renal excretion [12,13]. In the present study, however, $SI_{\text{cortex-post}}$ increased only in patients with Child-Pugh grades A or B. This may be explained by several factors: (1) Although a greater proportion of the contrast agent may remain in the circulation due to impaired hepatic clearance, the actual concentration differences after systemic dilution may be limited. Additionally, all participants had normal renal function, which may have resulted in minimal differences in renal cortical perfusion; hence, $SI_{\text{cortex-post}}$ differences were observed only between some groups. (2) The compensatory capacity of renal excretion may be limited. (3) Liver dysfunction may be associated with changes in renal function. Although all participants had normal renal function, the influence of the liver-kidney axis on the results cannot be completely excluded.

No significant differences were observed in the RE_{cortex} among the groups. Several factors may account for this finding. First, the relatively small number of patients with Child-Pugh grade C, which may have increased intragroup variability and broadened the distribution of RE_{cortex} values, thereby reducing the statistical power to detect differences between groups. Second, in patients with Child-Pugh grades A and B, the reduction in hepatic excretion is dynamically balanced by compensatory renal excretion, resulting in only minor changes in RE_{cortex} that are insufficient to reach statistical significance. Consequently, RE_{cortex} did not differ significantly between the groups.

Effects of liver function on renal medullary signal intensities

No significant differences were observed in $SI_{\text{medulla-pre}}$ between the normal control group and the different Child-Pugh grades. This may be explained by the following reasons: (1) The renal medulla is primarily composed of renal tubules, collecting ducts, and vasa recta, with its function mainly involved in urine concentration and electrolyte regulation. It receives only approximately 10% of the total renal blood flow and is therefore potentially less influenced by perfusion-related factors [14]. (2) Its T1-weighted signal intensity is largely determined by rela-

tively stable tissue characteristics, such as water and protein content [15], which may be less affected by the Child-Pugh grade.

Similarly, no significant differences were found in $SI_{\text{medulla-post}}$ or RE_{medulla} across groups, which may be closely related to the structural and functional features of the medulla. Renal tubules are predominantly located in the medullary region and do not reabsorb Gd-EOB-DTPA. In cases of liver dysfunction, compensatory renal excretion of Gd-EOB-DTPA primarily relies on the renal cortex rather than the medulla. Moreover, at the time of hepatobiliary phase imaging (20–30 min after injection) [16,17], the concentration of the contrast agent in the medulla may not have reached its peak or may have already been partially cleared, resulting in minimal changes in $SI_{\text{medulla-post}}$ and RE_{medulla} .

Effects of liver function on corticomedullary signal differences

The results showed no significant differences in $SI_{\text{cortex-medulla}}$ among the groups. This may be because corticomedullary signal differences on T1-weighted imaging are primarily determined by the tissue water content and anatomical structure. Moreover, all patients included in this study had normal renal function without significant intergroup differences, resulting in a consistent baseline signal. Although contrast enhancement may influence the corticomedullary signal to some extent, the magnitude of the signal change was relatively small across groups and did not reach statistical significance.

Clinical implications of renal signal assessment in the hepatobiliary phase

In clinical practice, the hepatobiliary phase is routinely obtained during Gd-EOB-DTPA-enhanced liver MRI, and the renal parenchymal signal can be assessed without additional scans. This provides incidental information for liver evaluation. However, hepatobiliary phase enhancement primarily reflects contrast filtration and redistribution rather than direct measures of renal function. Compared with dedicated renal imaging techniques, its sensitivity for detecting subtle abnormalities is limited. Therefore, renal signal changes observed during the hepatobiliary phase should be considered supplementary findings.

The number of patients with Child-Pugh grade C was relatively small, which may have reduced the statistical power and limited the ability to detect subtle differences between the groups. In addition, the signal intensity was assessed using semi-quantitative measurements rather than dynamic or fully quantitative techniques, which may have affected the sensitivity. Studies with larger cohorts and more quantitative imaging methods are required to further clarify these findings.

Conclusions

Higher Child-Pugh grades were associated with differences in hepatic signal intensity in both unenhanced and hepatobiliary phase imaging; however, differences in renal parenchymal signal intensity were limited, with significant differences observed only in certain cortical parameters. These findings suggest that the association between hepatic dysfunction and renal parenchymal signal intensity is limited.

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Disclosures

1. Institutional review board statement: This study was approved by the Medical Ethics Committee of the Second Affiliated Hospital of Kunming Medical University, with a waiver of informed consent for participants (Ethics approval reference number: No. PJ-2025-178).
2. Assistance with the article: None.
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4. Conflicts of interest: None.

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