Relationship between severity of liver dysfunction and the relative ratio of liver to aortic enhancement (RE) on MRI using hepatocyte-specific contrast

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Directed by Professor Myeong-Jin Kim

The Doctoral Dissertation submitted to the Department of Medicine, the Graduate School of Yonsei University in partial fulfillment of the requirements for the degree of Doctor of Philosophy

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Young Chul Kim

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ABSTRACT

Relationship between severity of liver dysfunction and the relative ratio of liver to aortic enhancement (RE) on MRI using hepatocyte-specific contrast

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Purpose: To evaluate differences in liver enhancement among patients with low and high morbidity risks and to determine the relationship between severity of liver dysfunction and the relative ratio of liver to aortic enhancement (RE) on MRI using hepatocyte-specific contrast.

Material and Methods: A total of 126 patients underwent MRI and blood serum testing including serology, bilirubin, international normalized ratio, and creatinine tests. Radiologists analyzed a region of interest in the liver and aorta on pre-contrast, and 10- and 20 minute-delayed hepatobiliary phase MR images. Liver enhancement after 10- (LE_{10min}) and 20 minutes (LE_{20min}) compared between the low- and high-risk groups by independent t-test. Regression analysis was used to assess the relationship between the Model for End-stage Liver Disease (MELD) score and RE.

Results: All 126 patients were classified into either the low-risk group (MELD <8; n=85) or high-risk group (MELD ≥ 8; n=41). The mean LE_{10min} and LE_{20min} were significantly higher in the low risk group (471.61; 95% CI: 449.79–493.43 and 510.69; 95% CI: 486.51–534.87, respectively) than in the high-risk group

(401.6776; 95% CI: 364.75–438.61 and 413.81; 95% CI: 370.91–456.70). There was a moderate inverse correlation between MELD score and the relative ratio of liver enhancement (RLE) (r=-0.5442; 95% CI: -0.6480 to -0.4207; p<0.01), but a high positive correlation between MELD score and RE (r=0.7470; 95% CI: 0.6665–0.8102; p < 0.01).

Conclusion: Although liver enhancement was significantly greater in low-risk patients compared to high-risk patients, RE may be a better predictor of liver function than RLE.

Key words : Gadolinium ethocybenzyl diethylenetriamine pentaacetic acid (Gd-EOB-DTPA), Hepatocytes, Liver Function Test, Magnetic Resonance Imaging (MRI)

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I. INTRODUCTION

Impaired liver function can significantly influence postoperative outcomes of major surgery, as well as urgently require liver transplantation. Thus, it is important to predict the severity of liver dysfunction in patients with chronic liver disease. To date, the most useful prognostic predictors include the Child–Turcotte–Pugh (CTP) and the Model for End-stage Liver Disease (MELD) scoring systems. Although the CTP scoring system is used in various clinical applications, there are limitations to its use as a means of assessing liver function due to subjective parameters such as the extent of ascites and hepatic encephalopathy.[1] Currently, the modified MELD scoring system is used to determine the priority of liver donor allocation in the United Network for Organ Sharing (UNOS).[2] The MELD scoring system is a quantitative predictor of morbidity and mortality calculated based on three objective variables: serum

creatinine (Cr), bilirubin, and international normalized ratio of prothrombin time (INR).[3, 4] The MELD score is reported to be superior to the CTP score in predicting three-month mortality and morbidity of patients with chronic liver disease.[5-10] According to previous studies, patients with a MELD score ≥ 8 had a higher postoperative morbidity rate than patients with a MELD score < 8.[11, 12]

Gadolinium ethocybenzyl diethylenetriamine pentaacetic acid (Gd-EOB-DTPA), also known as Gadoxectic acid or Primovist® (Bayering Schering Pharma AG, Berlin) is a newer, liver-specific contrast material that has unique biphasic excretory pathways.[13-19] Gd-EOB-DTPA is thought to be taken up by organic anion transporting polypeptides (OATPs) and excreted into the biliary tract through canalicular multiorganic anion transporters.[20, 21] Subsequently, approximately 50% of the injected dose of Gd-EOB-DTPA is eliminated by the biliary system, while the remaining portion is excreted renally via glomerular filtration.[22] Therefore, we hypothesized that poor liver function might induce impaired hepatocyte uptake and renal excretion of Gd-EOB-DTPA in the hepatobiliary phase.

The purpose of this study was to evaluate the differences in liver enhancement between patients with low and high morbidity risk using MRI with a hepatocyte-specific agent, and to analyze the correlation between severity of liver dysfunction and the relative ratio of liver to aortic enhancement (RE).

II. MATERIALS AND METHODS

1. Patient population

This study was approved by the institutional review board, which granted a waiver of informed consent for this retrospective analysis. We retrospectively reviewed 150 MRI images obtained from 126 patients with liver cirrhosis who underwent Gd-EOB-DTPA-enhanced MRI between April 2008 and May 2009. Among the 126 patients, 17 had more than two MRI examinations. Data collected from medical records included patient sex, age, type of hepatitis, mental status, degree of ascites, and liver function tests. Results of blood serum tests were recorded so long as they were performed within two weeks of MRI. These tests included levels of bilirubin, international normalized ratio (INR), creatinine (Cr), albumin, aspartate aminotransferase (AST), alanine transaminase (ALT), and alkaline phospatase (ALP). The MELD score was calculated using the following formula: MELD = 3.78 x $\log_e$(bilirubin [mg/dL])+ 11.20 x loge(INR) + 9.57 x loge(creatinine [mg/dL]) + 6.4.

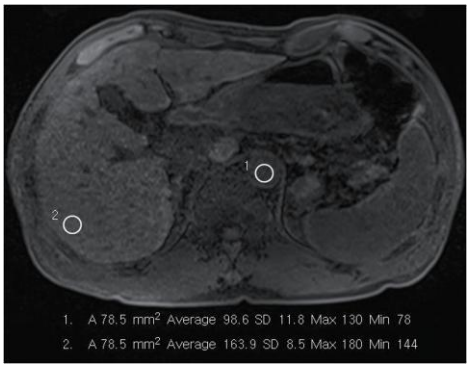
2. MRI examination

MRI testing was performed using a 3.0 Tesla scanner (MAGNETOM TrioTim; Siemens, Erlangen, Germany) with a six-element body phased-array coil on the anterior side of the patient and another six-element spine coil on the posterior side of the patient. We recommended fasting four hours prior to imaging. The pre-contrast T1-weighted MR images were obtained using a breath-hold transverse three-dimensional (3D) gradient echo (GRE) sequence with fat saturation (TR/TE, 2.54/0.95 msec; flip angle, 13°; field of view, 30 × 40 cm; matrix, 256×192; one signal acquired; section thickness, 2 mm; zero intersection gap).

Contrast-enhanced MRI was performed using a breath-hold 3D GRE sequence with a fat saturation sequence (volumetric interpolated breath-hold examination; VIBE sequence), following an IV bolus of 0.025 mmol Gd-EOB-DTPA per kilogram of body weight followed by a 20 ml saline flush. This sequence was repeated four times with data acquisition in the hepatic arterial, portal venous, and equilibrium phases. An automatic infusion system (Spectris MR injection system, Medrad Europe, Maastricht, Netherlands) operating at an injection rate of 2 mL/s was used. The actual pulse sequence was started manually when the fluoroscopic sequence revealed that the contrast material had reached the abdominal aorta. Hepatobiliary phase images were obtained 10 and 20 minutes after the injection using the same sequence and parameters used for pre-contrast and dynamic MR imaging.

3. Quantitative analysis

Two radiologists blinded to the clinical data, analyzed in consensus the pre-contrast, 10 minute- and 20 minute-delayed hepatobiliary phase images obtained by the same 3D fat-saturated GRE sequence parameter on a PACS (Centricity 3.x, GE Healthcare Integrated Imaging Solutions, Wisconsin, USA). Quantitative analysis was performed using the signal intensity (SI) of the liver parenchyma as measured by drawing regions of interest (ROI) and excluding visible bile ducts and large vessels. The same six ROIs in the liver parenchyma and aorta were measured in the pre-contrast liver ($L_{precontrast}$), pre-contrast aorta ($A_{precontrast}$), 10 min-delay (LE_{10min} and AE_{10min}), and 20 min-delay (LE_{20min} and AE_{20min}) hepatobiliary phase images (Fig.1 and 2).



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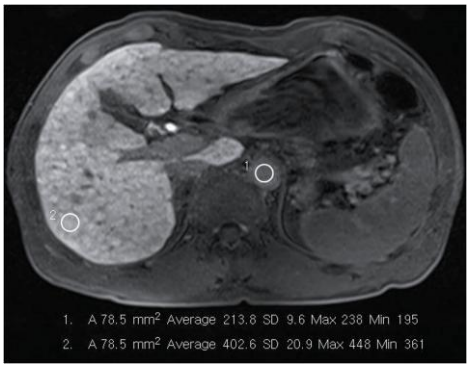

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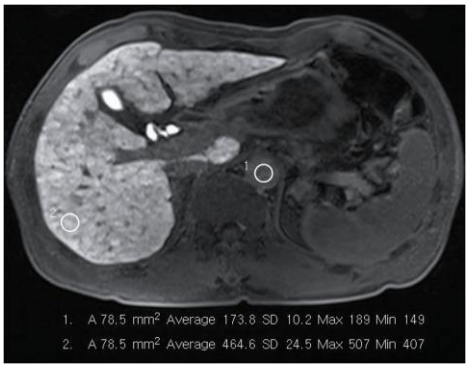

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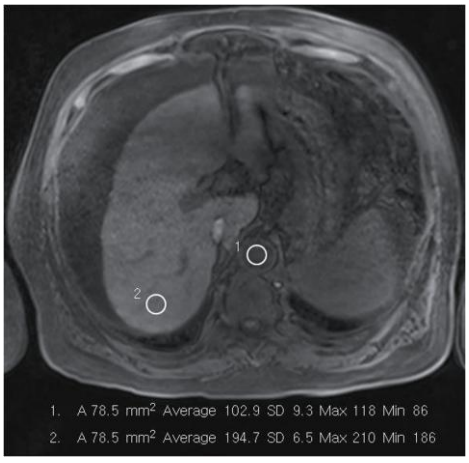
Figure 1. A 49-year old male who was classified in the low-risk group (MELD score: 0.98). a: SI values of $A_{precontrast}$ and $L_{precontrast}$ were measured by drawing two separate circular region of interest (ROI) in the aorta and liver on a precontrast axial T1-weighted image (T1WI). b: SI values of AE_{10min} and LE_{10min} were measured in the aorta and liver at the 10 minute-delayed hepatobiliary phase of contrast-enhanced axial T1WI with gadolinium-ethoxybenzyl-diethylenetriamine pentaacetic acid (Gd-EOB-DTPA). c: SI values of AE_{20min} and LE_{20min} were measured in the aorta and liver at the 20 minute-delayed hepatobiliary phase.



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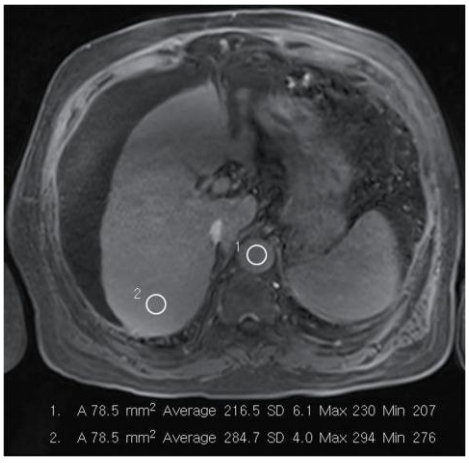

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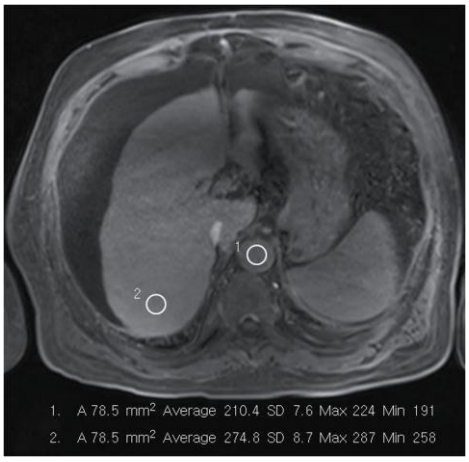

(c)

Figure 2. A 63-year old male who was classified in the high-risk group (MELD score: 26.58). (a) SI values of $A_{precontrast}$ and $L_{precontrast}$ were measured by drawing two separate circular region of interest (ROI) in the aorta and liver on a precontrast axial T1-weighted image (T1WI). (b) SI values of AE_{10min} and LE_{10min} were measured in the aorta and liver at the 10 minute-delayed hepatobiliary phase of contrast-enhanced axial T1WI with Gd-EOB-DTPA. (c) SI values of AE_{20min} and LE_{20min} were measured in the aorta and liver at the 20 minute-delayed hepatobiliary phase.

The degree of liver enhancement during the first 10 minutes (DLE_{10min}) and aortic enhancement during the first 10 minutes (DAE_{10min}) were calculated by subtracting the SI during the pre-contrast phase from SI in the 10 min-delayed hepatobiliary phase. In other words, $DLE_{10min} = LE_{10min}-L_{precontrast}$ and $DAE_{10min} = AE_{10min}-A_{precontrast}$. The degree of liver enhancement between the 10- and 20-minute periods ($DAE_{10\sim20min}$) as well as aortic enhancement from 10-20 minutes ($DAE_{10\sim20min}$) were calculated by subtracting the SI measured in the 10 min-delayed hepatobiliary phase from the SI in the 20 min-delayed hepatobiliary phase. That is, $DLE_{10\sim20min} = LE_{20min}-LE_{10min}$ and $DAE_{10\sim20min} = AE_{20min}-AE_{10min}$.

DLE and DAE were used to calculate the ratio of liver enhancement (RLE) and aortic enhancement (RAE) using the following equations: $RLE = (DLE_{10\sim20min}/DLE_{10\ min})$; $RAE = (DAE_{10\sim20min}/DAE_{10\ min})$. Finally, we calculated the relative ratio of liver to aortic enhancement (RE) using the following equation:

$$RE = \frac{RLE}{RAE} = \frac{[(DLE_{10\sim20min}/DLE_{10\ min})}{(DAE_{10\sim20min}/DAE_{10\ min})}$$

4. Statistical analysis

All patients were assigned to low- and high-risk groups for postoperative morbidity based on their MELD score. Low-risk patients were defined as having a MELD score < 8 and high-risk patients had a MELD ≥ 8. All statistical analyses were conducted using MedCalc for Windows version 9.5 (MedCalc Software, Mariakerke, Belgium). Among the low- and high-risk groups, the $L_{precontrast}$, $A_{precontrast}$,LE_{10min}, AE_{10min}, LE_{20min}, $AE_{20min,}$ DLE_{10min}, $DLE_{10\sim20min}$, DAE_{10min}, and $DAE_{10\sim20min}$ were compared and analyzed using an independent, two-tailed Student's t test. Regression analysis was used to estimate the correlation between MELD score and RE liver among patients as well as

between MELD score and RLE. Correlation coefficients of 0.3 or less were considered a weak relationship, while those of 0.30-0.70 and greater than 0.70 indicated moderate and high-strength relationships. A z-test was used to compare two correlation coefficients. Additionally, receiver operating characteristic (ROC) analysis was used to evaluate the diagnostic performance of RE in predicting the severity of liver dysfunction. For all tests, a *p*-value of less than 0.05 was considered statistically significant.

III. RESULTS

Among the study population of 126 patients, the mean age was 57.62 (SD ±1.96). The patients were predominantly male (male to female ratio = 108:18). Ninety-seven patients (76.98%) had chronic viral hepatitis B. There were no significant differences between the low and high-risk groups with regard to clinical characteristics such as age, gender, and hepatitis etiology. The clinical characteristics of all 126 patients are summarized in the Table 1. Among the 41 patients in the high-risk group, the mean MELD score was 12.35 (95% confidence interval CI: 8.01–41.52). The low-risk group consisted of 85 patients with a mean MELD score of 4.87 (95% CI: -1.56–7.99). The mean MELD score in the high-risk group was significantly higher than that in the low-risk group (p<0.0001). The mean Cr level of the low-risk group, 0.918 mg/dL (95% CI: 0.875-0.9614), was significantly lower than that of the high-risk group (1.3908 mg/dL; 95% CI: 0.9973-1.7843; p=0.0009).

The mean $L_{precontrast}$ value, as measured by drawing a ROI on pre-contrast axial T1-weighted images, was not statistically different between the low-risk group (204.68; 95% CI: 196.92–212.44) and high-risk group (204.92; 95% CI: 191.21–218.63) (Fig. 1 and 2). The mean LE_{10min} of the low-risk group was higher than that of the high-risk group (471.61; 95% CI: 449.79-493.43 and 510.69; 95% CI: 486.51–534.87, respectively). Similarly, the mean LE_{20min} was higher in the low-risk group compared to the high-risk group (401.6776; 95% CI: 364.75–438.61 and 413.81; 95% CI: 370.91-456.70, respectively). Variations in SI and LE between groups at various time intervals post-Gd-EOB-DTPA injection are presented in Table 2 and Figure 3. Between the low and high-risk groups, there were no significant differences in mean $A_{precontrast}$, AE_{10min} and AE_{20min} (Table 3; Fig. 4).

Table 1. Characteristics of low- and high-risk patients based on the Model for End-Stage Liver Disease (MELD) scoring system.

	Total	Low-risk group	High-risk group	*P*-value
Number of patients	126	85	41	-
Number of MRIs	150	101	49	-
Mean age	57.62±1.97	58.35±2.14	55.78±3.28	0.1832
Sex (Male/Female)	108/18	70/15	38/3	0.1999
Viral hepatitis B	97	66 (77.65%)	31 (75.61%)	0.9768
Viral hepatitis C	10	9 (10.59%)	1 (2.44%)	0.2173
Other etiology*	19	10 (11.76%)	9 (21.95%)	0.2179

Note: The low-risk group (n=85) by definition had a MELD <8. The high-risk group (n=41) by definition had a MELD ≥ 8.

*Other etiology includes: Non-viral hepatitis, alcoholic hepatitis, Bud-chiari, SLE, etc.

Table 2. Mean SI of liver in the low- and high-risk groups at pre-contrast, 10 minutes, and 20 minutes post-Gd-EOB-DTPA injection.

	Low-risk group (95% CI)	High-risk group (95% CI)	*P*-value
$L_{precontrast}$	204.68 (196.92–212.44)	204.92 (191.21–218.63)	0.9745
LE_{10min}	471.61 (449.79-493.43)	401.6776 (364.75–438.61)	0.0008
LE_{20min}	510.69 (486.51–534.87)	413.81(370.91-456.70)	<0.0001

Note: Three ROIs were measured at the same location and level in the liver parenchyma on the pre-contrast ($L_{precontrast}$), 10- (LE_{10min}), and 20 min-delay hepatobiliary phase images (LE_{20min}).

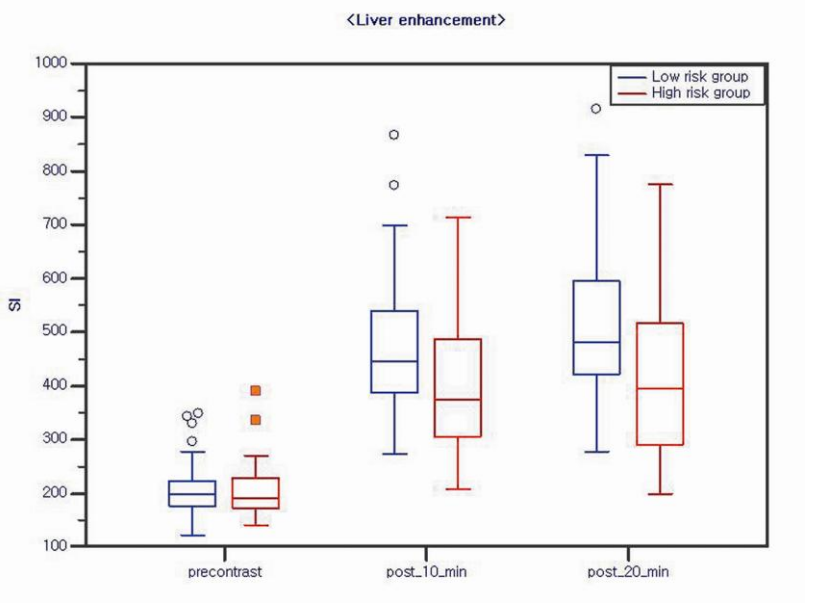

Figure 3. Box plots demonstrating liver signal intensity (SI) changes in the low- and high-risk groups at pre-contrast, 10 and 20 minutes after Gd-EOB-DTPA. Each box stretches from the 25th percentile at the lower edge to the 75th percentile at the upper edge. The mean value is depicted as a short line across the box.

Table 3. Mean SI of aorta in the low- and high-risk groups at the pre-contrast, 10 min, and 20 minutes post-Gd-EOB-DTPA injection.

Time point	Low risk group (95% CI)	High risk group (95% CI)	*P*-value
$A_{precontrast}$	113.03 (108.82–117.24)	105.27 (97.84–112.71)	0.0538
AE_{10min}	301.30 (286.80–315.79)	284.27 (263.47–305.06)	0.1828
AE_{20min}	256.87 (243.31–270.47)	229.63 (229.63-271.06)	0.5914

Note: Three ROIs were measured in the aorta on the pre-contrast ($A_{precontrast}$), 10- (AE_{10min}) and 20 min-delay hepatobiliary phase images (AE_{20min}).

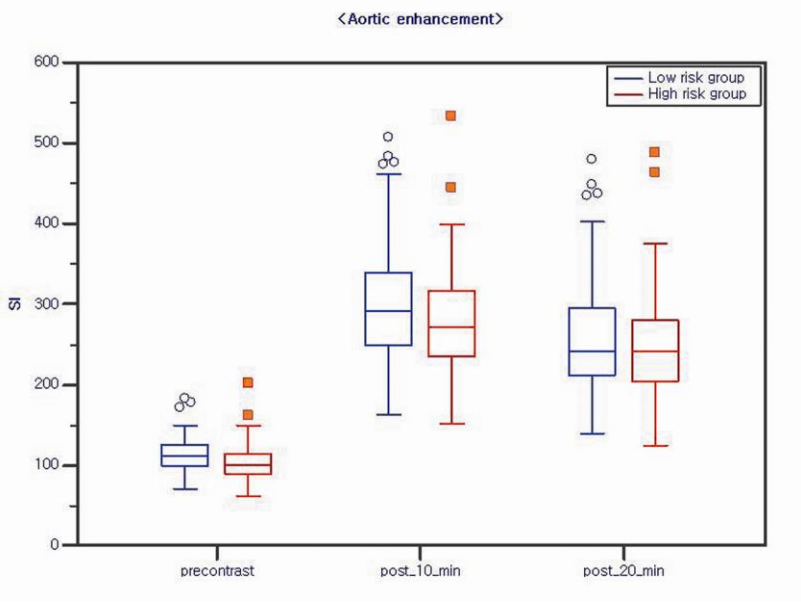

Figure 4. Box plots demonstrating aorta signal intensity (SI) changes in the low- and high-risk groups at pre-contrast, 10 and 20 minutes after Gd-EOB-DTPA. Each box stretches from the 25th percentile at the lower edge to the 75th percentile at the upper edge. The mean value is depicted as a short line across the box.

Table 4. Mean degree of liver and aortic enhancement in the low- and high-risk groups.

	Low-risk group (95% CI)	High-risk group (95% CI)	P-value
DLE_{10min}	266.93 (249.61 – 284.24)	196.82 (164.77 – 228.87)	< 0.0001
$DLE_{10\sim20min}$	39.08 (34.09 – 44.07)	12.13 (2.26 – 22.00)	< 0.0001
DAE_{10min}	188.27 (176.94 – 201.28)	178.9898 (163.63 – 194.35)	0.3606
$DAE_{10\sim20min}$	-44.41 (-50.02 – -38.81)	-55.92 (-66.80 – -41.05)	0.0837

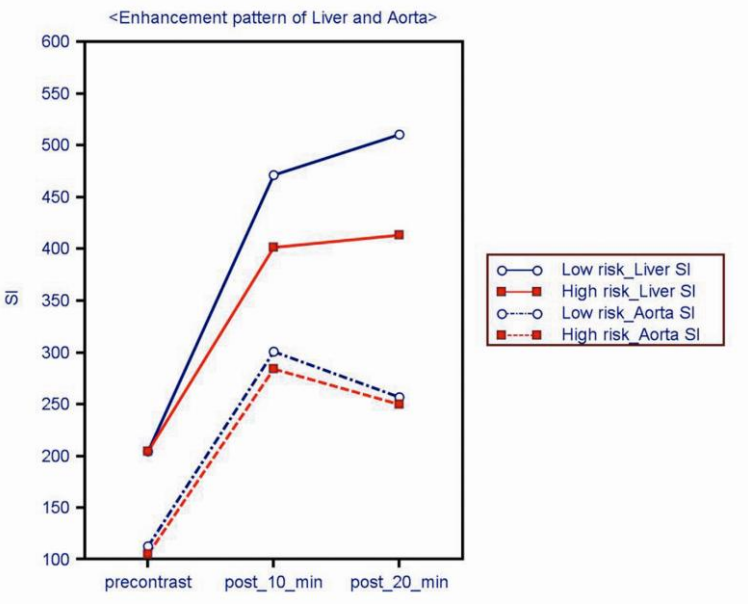

Figure 5. Graph demonstrating four lines corresponding to the mean SI changes of liver and aortic enhancement at pre-contrast, 10 and 20 minutes after Gd-EOB-DTPA in the low- and high-risk groups. Two time intensity curves of liver enhancement demonstrate an increased slope in both groups. Time courses of aortic enhancement demonstrate different slopes.

As shown in Figure 5, there were increased slopes in the liver enhancement curves of both groups. The mean LE_{20min} of both groups was higher than LE_{10min}. The $DLE_{10\sim20min}$ of both risk groups was higher than DLE_{20min} (Table 3). There were no significant differences in the mean DAE_{10min} and $DAE_{10\sim20min}$ between groups (Table 4).

For the lowest MELD score of -1.56, the ratio of liver enhancement (RLE) was 0.2978 and the ratio of aortic enhancement (RAE) was -0.2319. For the highest MELD score of 41.52 RLE and RAE were both -0.2934. There was a moderate inverse correlation between MELD score and RLE (r = -0.5442, 95% CI: -0.6480 to -0.4207, $p < 0.01$). There was a weak direct correlation between MELD score and RAE (r = 0.2073, 95% CI: 0.04868 to 0.3558, p = 0.0109). When RLE was divided by RAE, the RE values were -1.28 and 2.23. There was a stronger positive correlation between MELD score and RE (r = 0.7470, 95% CI: 0.6665 to 0.8102, $p < 0.01$) (Fig. 6). The correlation coefficient between MELD score and RE was significantly higher than that between MELD score and RLE (p=0.0023). In addition, strong positive correlation coefficient between MELD score and RE (r = 0.7409, 95% CI: 0.6604 to 0.8041; $p < 0.01$) was shown in 17 patients who performed more than two MRI examinations.

The diagnostic performance of RE in predicting the severity of liver dysfunction was assessed by calculating the mean of the area under the ROC curve (Az= 0.8, 95% CI: 0.73–0.86, p<0.0001). The best cutoff RE value for distinguishing between low-and high-risk groups was -0.25 (sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV): 59.2%, 87.1%, 69% and 81.5%).

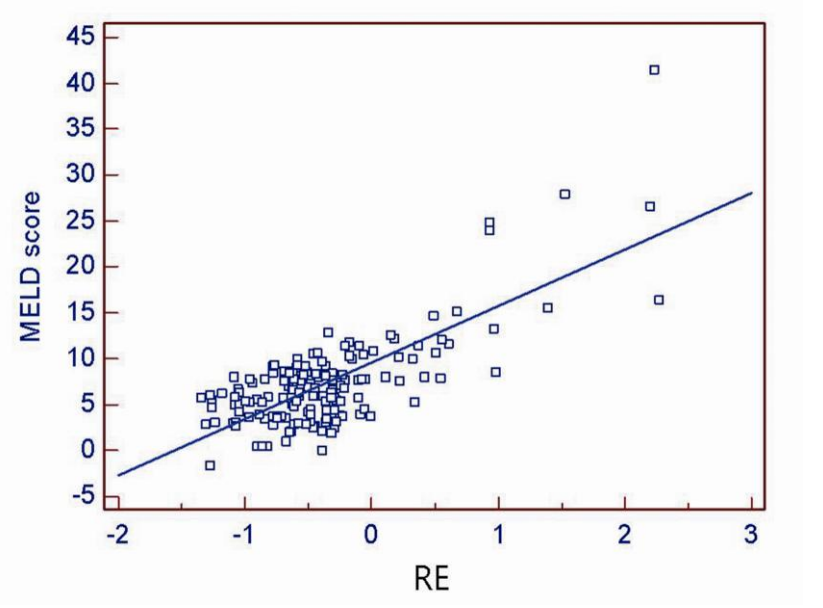

Figure 6. Scatterplot demonstrating high positive correlation between RE and MELD score (r = 0.7470; 95% CI: 0.6665 to 0.8102; p < 0.01). The correlation coefficient of greater than 0.70 indicates a strong relationship.

IV. DISCUSSION

Gd-EOB-DTPA is transported into hepatocytes via organic anion transporting polypeptides (OATPs) and excreted into bile by multidrug resistance–associated protein 2 (MRP2). After injection of contrast, liver enhancement peaked at 30 minutes in both the normal liver function and chronic liver dysfunction groups. Liver enhancement increased significantly at 20 minutes in patients with normal liver function; however, there was no significant difference in enhancement between 25 and 30 minutes.[23] Therefore, liver enhancement increases exponentially with time and liver enhancement is higher in the normal liver compared to the chronically diseased liver.[23-26] The degree of liver enhancement is affected not only by OATPs that regulate contrast uptake, but also by MRP2 that regulates contrast elimination. MRP2 up-regulation in cirrhosis can lead to decreased liver enhancement in cirrhotic compared to the normal rat liver.[25]

Our results demonstrate that both LE_{10min} and LE_{20min} of the low-risk group were higher than that of the high-risk group. Liver enhancement curves showed increased slope (Fig. 5). There were significant differences in the mean degree of liver enhancement at 10 minutes (DLE_{10min}), and 20 minutes ($DLE_{10\sim20min}$) between the low- and high-risk groups (Table 4). However, when evaluating the relationship between MELD and RLE, there was a moderate inverse correlation between MELD and RLE (r = -0.5442; 95% CI: -0.6480 to -0.4207; $p < 0.01$).

The MELD score includes serum Cr level as well as bilirubin and INR of patients with underlying liver disease. Several researchers have evaluated the degree of liver enhancement between normal liver and chronic liver disease patients, and the kidney function of both groups was found to be almost identical.[23, 25] In our study, the mean Cr level of the low-risk group was significantly lower than that in the high-risk group. The distribution of Gd-EOB-DTPA in ECF space is influenced not only by the concentration

gradient, but also by renal function post-intravenous administration. The enhancement slope, peak time and maximal concentration of aortic enhancement are different between the healthy kidney and kidney with renal failure and high Cr levels.[27]

Several researchers have defined ROI over the heart, spleen, porta hepatis and aorta.[24, 26-28] We drew the ROI in the transverse section of the aorta. In our study, there was no significant difference in the mean $A_{precontrast}$, AE_{10min} and AE_{20min} of aorta between low- and high-risk patients (Table 3; Fig. 4). Moreover, there was no significant difference in between the mean DAE_{10min} and $DAE_{10\sim20min}$ in both groups (Table 4). There was a moderate inverse correlation between MELD score and RLE (r = -0.5442, 95% CI: -0.6480 to -0.4207, p < 0.01). However, after RLE was divided by RAE, there was a strong positive correlation between MELD score and RE (r = 0.7470, 95% CI: 0.6665 to 0.8102; p < 0.01).

This study is limited due to its retrospective design, which has inherent biases. Next, the majority of our study population had chronic viral hepatitis B and a low MELD score, making it difficult to extrapolate our results to the general population of patients suffering from chronic liver disease. Our study population was composed of viral hepatitis B (n=97; 76.98 %), C (n=10; 7.94%) and other etiologies (n=19; 15.08%). Hepatitis B virus (HBV) is a double-stranded DNA virus that integrates in the host genome. HCC may develop in non-cirrhotic HBV-infected patients. In contrast, hepatitis C virus (HCV) is a single-stranded RNA virus that does not integrate into the host genome. HCV is known to be involved in the hepatocarcinogenesis, e.g. host protein interactions or the inflammatory process. Moreover, chronic inflammation and hepatocyte regeneration may cause chromosomal damage and cirrhotic change in the liver. The majority of the patients with HCV-related HCCs have underlying liver cirrhosis.[29] Other types of liver disease have a different course. Therefore, prospective studies need to be conducted to

investigate a larger sample size of patients including specific types of hepatitis and higher MELD scores.

V. CONCLUSION

Although the degree of liver enhancement was found to be significantly lower in the low-risk group compared to the high-risk group, the relative ratio of liver to aortic enhancement (RE) may provide significantly better prediction of liver function than the relative ratio of liver enhancement (RLE).

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ABSTRACT(IN KOREAN)

간세포 특이 조영제를 이용한 자기공명영상에서 간 조영증강도와 간 기능 장애의 상관관계

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연구목적: 간세포 특이 조영제를 이용한 자기공명영상에서 간 조영증강도의 차이가 간 기능 장애의 중증도와 상관관계가 있는지를 규명하고자 하였다.

대상과 방법: 총 126명의 환자들이 자기공영촬영과 빌리루빈, 프로트롬빈 시간(INR-PT)과 크레아티닌의 혈청학적 검사를 시행하였다. 영상의학과 전문의가 조영전, 조영후 10분과 20분 지연 간담도기 자기공명상에서 간과 대동맥 관심영역 (region of interest)을 설정하여 정량 분석하였다. 수술 후 발생할 수 있는 신부전, 뇌병증, 감염, 응고장애 등의 이환율이 높은 고위험군과 이환율이 낮은 저위험군에서의 조영제 주입 후 10분 뒤 조영증강도(LE_{10min})와 20분 뒤 조영증강도(LE_{20min})를 독립표본 t-검정(independent t-test)을 이용하여 비교하였다. 회귀분석(Regression analysis)을 사용하여 MELD점수와 간 대 대동맥 조영증강도의 상관관계를 평가하였다.

결과: 총 126명의 환자들은 저위험군(MELD점수 8미만; 85명)과 고위험군(MELD점수 8이상; 41명)으로 분류되었다. 평균 LE_{10min}와

LE_{20min}는 저위험군(각각 471.61; 95% 신뢰구간: 449.79–493.43 그리고 510.69; 95% 신뢰구간: 486.51–534.87)이 고위험군(각각 401.6776; 95% 신뢰구간: 364.75–438.61와 413.81; 95% 신뢰구간: 370.91–456.70)보다 유의하게 높은 수치를 보였다. MELD점수와 간의 조영증강도 간에는 중등도 역상관관계(r=-0.5442; 95% 신뢰구간: -0.6480– -0.4207; p<0.01)였으나, MELD점수와 간 대 대동맥 조영증강도(r=0.7470; 95% 신뢰구간: 0.6665– 0.8102; p < 0.01) 간에는 강한 양의 상관관계였다.

결론: 간 조영증강도는 고위험군보다 저위험군에서 유의하게 높으나 간 대 대동맥 조영증강도가 간 조영증강도보다 더 좋은 예측인자가 될 수 있다.

핵심되는 말: Gadolinium ethocybenzyl diethylenetriamine pentaacetic acid (Gd-EOB-DTPA), Hepatocytes, Liver Function Test, Magnetic Resonance Imaging (MRI)