

Renal vein measurement using ultrasonography in  
patients with cirrhotic ascites and  
congestive heart failure

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金子 真大

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指導教員 森山 光彦



# Renal vein measurement using ultrasonography in patients with cirrhotic ascites and congestive heart failure

Masahiro Kaneko<sup>1</sup> · Naoki Matsumoto<sup>1</sup> · Mariko Kumagawa<sup>1</sup> · Yukinobu Watanabe<sup>1</sup> · Midori Hirayama<sup>1</sup> · Hiroshi Nakagawara<sup>1,2</sup> · Toshiki Yamamoto<sup>1</sup> · Masahiro Ogawa<sup>1</sup> · Mitsuhiro Moriyama<sup>1</sup>

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## Abstract

**Purpose** Ascites can cause compression of the inferior vena cava (IVC), leading to increased renal venous pressure and renal congestion. Previously, the left renal vein diameter in liver cirrhosis patients with ascites was measured using computed tomography, showing that enlargement of the left renal vein diameter affects the prognosis. Herein, the diameter and flow velocity of the renal veins were measured using ultrasonography.

**Methods** Abdominal ultrasonography was performed on 186 patients. The patients were divided into four groups: normal liver ( $n = 102$ ), liver cirrhosis (LC) without ascites ( $n = 37$ ), LC with ascites ( $n = 30$ ), and congestive liver ( $n = 17$ ). Ultrasonographic measurements for diameter and flow velocity of the IVC, left renal vein main trunk, and segmental renal vein were performed.

**Results** The left renal vein diameter increased in the following order: normal liver, LC, LC with ascites, and congestive liver groups ( $P < 0.001$ ). IVC flow velocity was lower and left renal vein diameter was larger in the congestive liver and LC with ascites groups. These results suggest that the two groups have different pathological conditions, but the mechanism of renal congestion is similar. In patients with LC, IVC compression due to ascites might cause blood stagnation and renal congestion.

**Conclusion** The left renal vein and IVC can be measured using ultrasonography. It might help in furthering our understanding of the pathophysiology of renal congestion in these patients.

**Keywords** Ascites · Ultrasonography · Cirrhosis · Renal congestion · Congestive heart failure

## Introduction

In congestive heart failure (CHF), increased central venous pressure is associated with a decline in renal function due to renal venous hypertension, renal congestion, and renal failure. This condition also has a poor prognosis and significant morbidity, and may lead to death [1, 2]. Fluid retention associated with CHF leads to an increase in renal interstitial

pressure, which compresses renal blood vessels and tubules, and impairs glomerular filtration and renal medullary blood flow [3]. Renal congestion alone is associated with increased mortality and morbidity in patients with heart failure. Renal congestion is also linked to cirrhotic ascites, which frequently causes compression of the inferior vena cava (IVC), an increase in renal venous pressure, and dilation of the renal veins. In a previous study using computed tomography (CT) to measure renal vein diameter, it was shown that patients who had refractory cirrhotic ascites and left renal vein dilation had high mortality rates [4]. To date, a few studies have measured the renal vein diameter or renal flow velocity using the harmless and painless color Doppler ultrasonography [5, 6].

The aim of this study was to assess the clinical significance of ultrasonography in measuring renal vein diameter and flow velocity in patients with and without liver disease. These measurements might be used as prognostic markers

✉ Naoki Matsumoto  
matsumotosg@yahoo.co.jp

Masahiro Kaneko  
m1659k@yahoo.co.jp

<sup>1</sup> Division of Gastroenterology and Hepatology, Department of Medicine, Nihon University School of Medicine, 30-1 Oyaguchi Kamimachi, Itabashi-ku, Tokyo 173-8610, Japan

<sup>2</sup> Department of Gastroenterology, Japan Community Health Care Organization Yokohama Chuo Hospital, Yokohama, Japan

and may ultimately assist in treating cases of liver disease, renal disease, and CHF.

## Materials and methods

### Patient selection and eligibility

This cross-sectional, observational study involved 186 consecutive patients examined between April 2015 and March 2017 at our hospital. The patients were divided into four groups: normal liver ( $n=102$ ), liver cirrhosis (LC) without ascites ( $n=37$ ), LC with ascites ( $n=30$ ), and congestive liver ( $n=17$ ). The inclusion criteria were: age  $\geq 20$  years; patients in the normal liver group had no history of chronic liver disease or heart disease and were negative for hepatitis B surface antigens and hepatitis C virus antibodies, while patients with LC were divided into two groups, i.e., those with no or minimal ascites, and those with more-than-minimal ascites. Patients on artificial dialysis, patients with only one kidney, and patients with a gastrosplenic or splenorenal shunt were excluded. In the normal liver and LC groups, patients with heart disease were excluded. In the congestive liver group, patients with other chronic liver diseases were excluded. LC was diagnosed based on the history of chronic liver disease, physical examination, blood examination, and findings of LC and/or portal hypertension (nodular liver, splenomegaly, and collaterals including gastroesophageal varices) on CT, ultrasonography, or gastrointestinal endoscopy. The diagnosis of congestive liver was based on the elevation of aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, or  $\gamma$ -glutamyl transferase levels, dilation of the hepatic veins, and decreased respiratory movement of the IVC. In all cases, clinical data were obtained from hospital records. The Nihon University Institutional Review Board approved this study. Informed consent was obtained by an opt-out method, because this study was retrospective in design and targeted cases from routine clinical practice.

### Clinical assessment and blood tests, and various imaging studies

In this study, clinical assessments and general blood tests (total bilirubin, blood urea nitrogen, creatinine, sodium levels, and serum albumin) were performed for each patient on the same day as that of the ultrasound examination.

The presence or absence of esophageal varices was confirmed in patients with liver cirrhosis who underwent upper gastrointestinal endoscopy within 3 months before and after ultrasonography.

In patients with congestive liver, N-terminal prohormone of brain natriuretic peptide (NT-proBNP) in the blood,

cardiothoracic ratio (CTR) on chest X-ray, and ejection fraction (EF) and tricuspid pressure gradient (TRPG) using transthoracic echocardiography were confirmed within 3 months before and after ultrasonography.

In patients with liver cirrhosis and congestive liver, CT was performed within 3 months before and after ultrasonography, and the left renal vein diameter was measured on CT images. The left renal vein diameter was measured on CT at the main trunk of the renal vein [4]. Measurements were performed using non-contrast CT.

### Ultrasonographic measurement of the IVC and renal veins

Ultrasonographic measurements of the diameter and the flow velocity of the IVC, left renal vein main trunk, and segmental renal vein in the supine position were taken. The ultrasonographic equipment used in this study included LOGIQ S8 (GE Healthcare Japan, Tokyo, Japan), Xario, Aplio 300 (Canon Medical Systems, Tokyo, Japan), and Aloka  $\alpha 10$  (Hitachi Aloka Medical Ltd., Tokyo, Japan). The IVC parameters were measured with a sagittal scan. The largest diameter during exhalation was measured. The venous flow velocity was measured three times when the heart rate was stable, and the velocity recorded was the average of the three measurements. The IVC was measured at the caudal end of the liver. The left renal vein diameter and flow velocity were measured at the main trunk of the renal vein, which is downstream from the confluence of branched veins. The left renal vein was measured, because the right renal vein sometimes branches into multiple renal veins connecting to the IVC (occurs in approximately 15% of patients). The left renal vein, which runs ventral to the aorta, was measured on a transverse scan. The segmental renal vein was measured on the left intercostal scan (Fig. 1).

All tests were performed by two specific physicians who were board-certified Fellows of the Japan Society of Ultrasonics in Medicine (FJSUM) (M.K., N.M.; 5 and 15 years of imaging experience, respectively).

### Statistical analyses

The data are shown as mean  $\pm$  standard deviation. Patient characteristics were compared using the Mann–Whitney  $U$  test and the  $\chi^2$  test. Normal distribution was assessed with the Kolmogorov–Smirnov test.  $P$  values  $< 0.05$  were considered statistically significant for all analyses. All statistical analyses were performed using EZR (Saitama Medical Center, Jichi Medical University, Saitama, Japan), a graphical user interface for R (R Foundation for Statistical Computing, Vienna, Austria). EZR is a modified version of R Commander, enabling statistical functions frequently used in biostatistics.

**Fig. 1** Measurement of renal veins. **a** The diameter and flow velocity of the left renal vein were measured at the kidney side of the superior mesenteric artery crossing. **b** The segmental renal vein was measured at the left intercostal space using color Doppler ultrasonography

## Results

### Blood test results by patient group

One hundred eighty-six patients satisfied the study inclusion criteria. Table 1 summarizes patient background, clinical characteristics, and blood test data. In the LC and LC with ascites groups, total serum bilirubin levels were higher than that in the normal liver group ( $P < 0.001$ ). Serum albumin levels decreased in the following order: normal liver group, LC group, congestive liver group, and LC with ascites group ( $P < 0.001$ ). Serum sodium levels were lower in the LC and LC with ascites groups compared to the normal liver group ( $P < 0.001$ ).

### Success rate of ultrasonographic measurement of left renal vein diameter and flow velocity

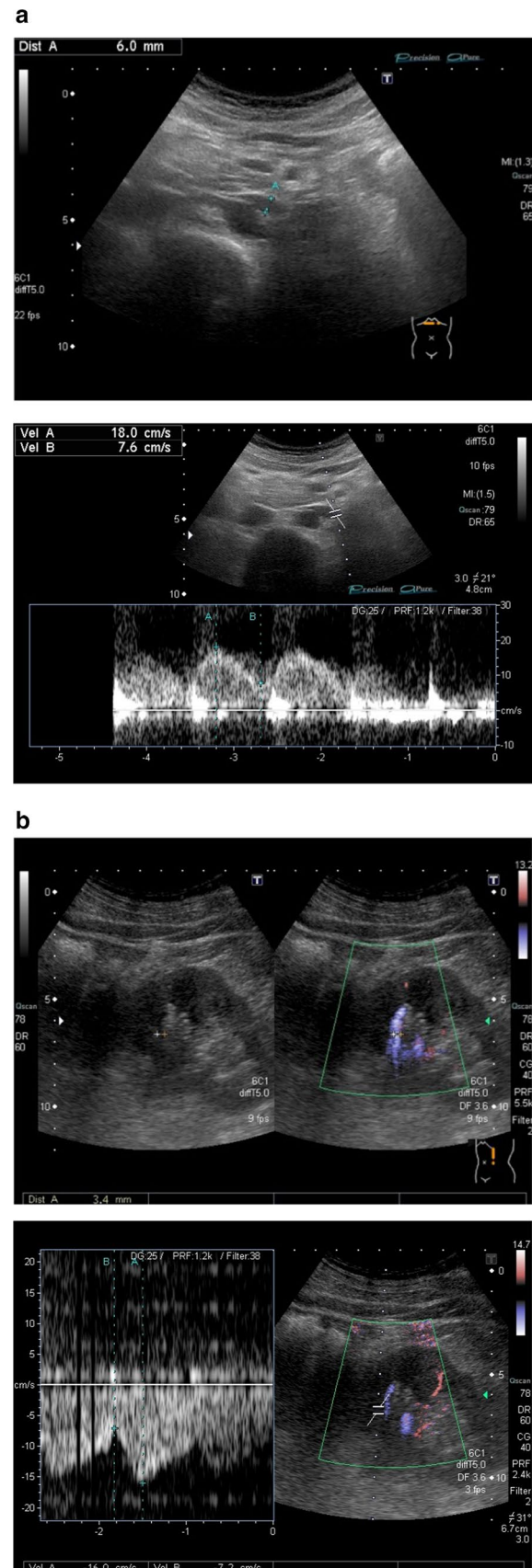
The success rate of measuring left renal vein diameter was 96% in the normal liver group, 97% in the LC group, 63% in the LC with ascites group, and 100% in the congestive liver group. The success rate of measuring left renal vein flow velocity was 91%, 76%, 37%, and 88%, respectively. The success rate of measuring segmental renal vein diameter was 94%, 95%, 100%, and 94%, respectively. The success rate of measuring segmental renal vein flow velocity was 91%, 81%, 87%, and 77%, respectively. In the normal liver, LC without ascites, and congestive liver groups, the success rate of measuring left renal vein diameter was more than 70% (Fig. 2).

### Normally distributed left renal vein diameters in normal liver group

The diameter and flow velocity of the left renal vein were normally distributed in the normal liver group ( $P = 0.012$  and  $< 0.001$ , respectively) (Fig. 3). In this group, the diameter of the left renal vein did not correlate with height, weight, or diameter of the IVC ( $P = 0.085$ ,  $0.922$ , and  $0.468$ , respectively).

### Correlation between the IVC and left renal vein diameters, height, and age

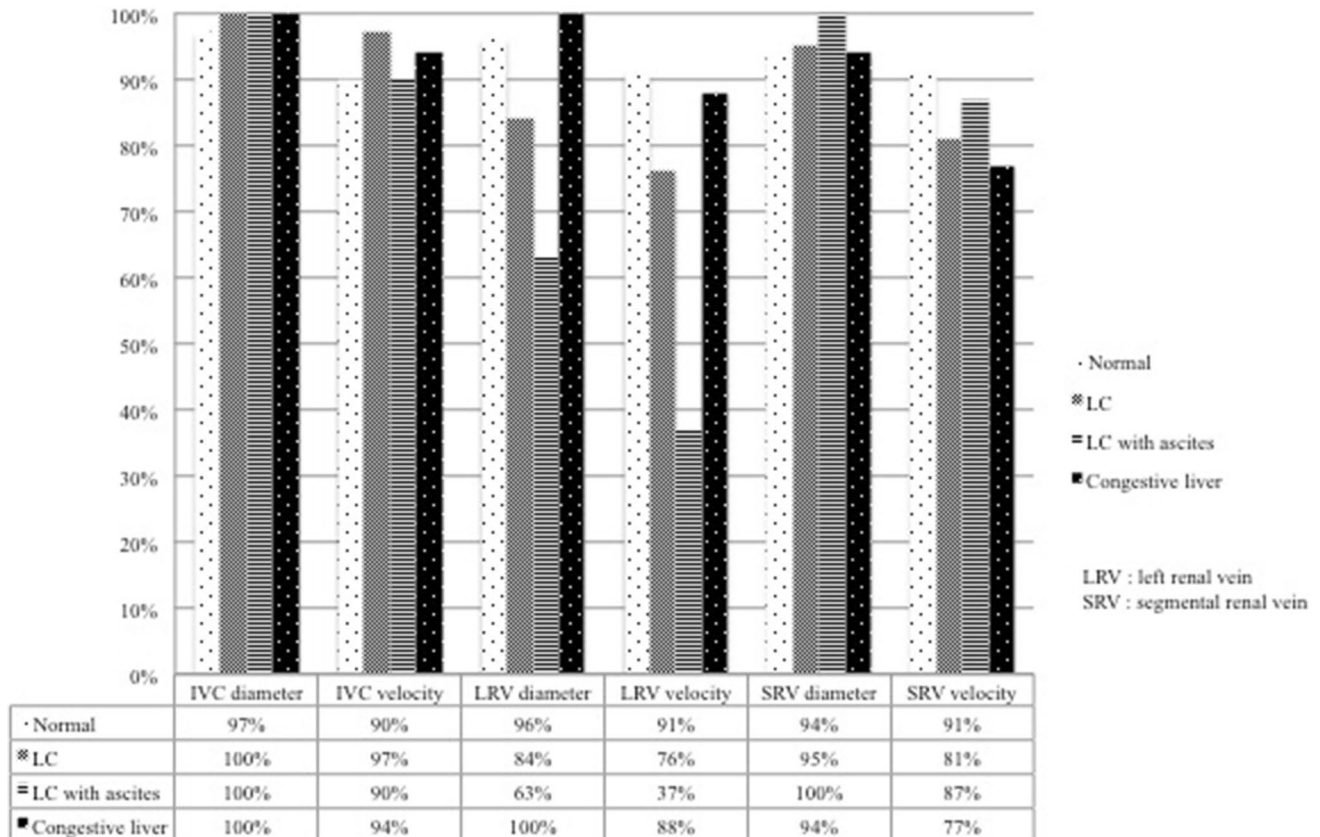
In the normal liver group, the diameter of the IVC correlated with height ( $r = 0.347$ ,  $P = 0.001$ ) and age ( $r = -0.436$ ,



**Table 1** Patient characteristics

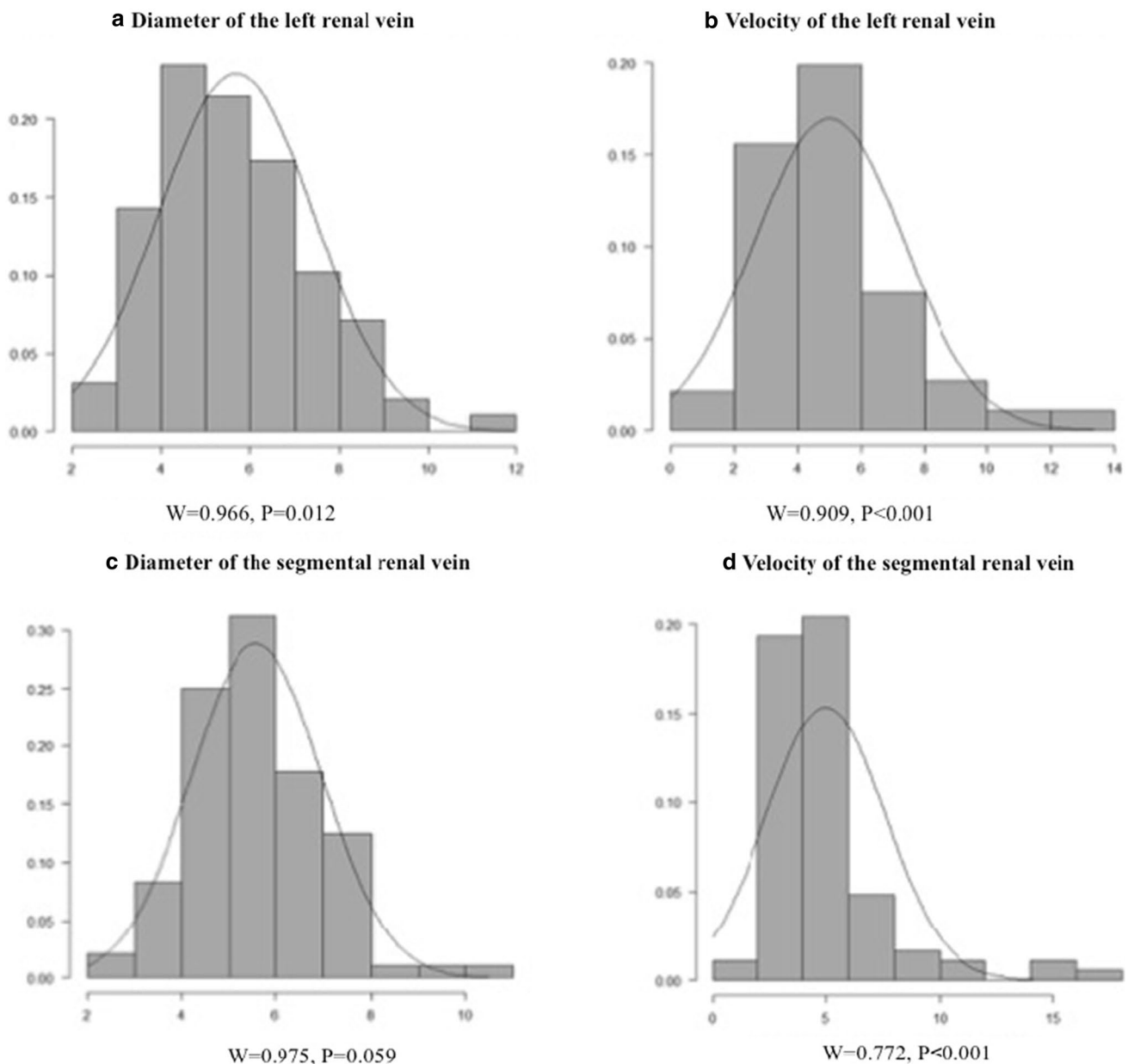
	Normal	LC	LC with ascites	Congestive liver	<i>P</i> value
<i>n</i>	102	37	30	17	
Age (years)	64.8 (26–93)	68.9 (48–85)	69 (48–88)	71.5 (48–85)	0.168
Sex (male/female)	54/48	20/17	22/8	11/6	
Height (cm)	160.3 (130–183)	159.2 (142–176)	160.2 (144–176)	157.6 (179–142)	
BMI	21.8 (15.8–32)	21.6 (17–27.3)	23.1 (16.8–32.5)	19.9 (17.4–23)	0.221
Background (HBV/HCV/alcohol/other)		2/17/7/11	0/12/9/9		
Esophageal varices (non/F0/F1/F2/F3)		(6/0/9/1/0)	(3/1/7/8/3)		
Total bilirubin (mg/dl)	0.57 (0.18–2.2)	1.04 (0.3–2.21)	2.7 (0.4–23.1)	1.61 (0.37–8.99)	<0.001
Urea nitrogen (mg/dl)	15 (5.4–44.3)	16.9 (8.1–43.9)	19.9 (0.5–71.4)	24.9 (10.5–57.4)	0.036
Serum creatinine (mg/dl)	0.77 (0.39–2.02)	0.84 (0.4–2.66)	1.0 (0.4–3.6)	1.48 (0.4–6.35)	0.109
Serum sodium (mEq/l)	141 (134–149)	139.8 (132–145)	138 (129–143)	137.5 (130–142)	<0.001
Serum albumin (g/dl)	4.1 (2.8–4.1)	3.58 (2.6–4.8)	2.7 (1.8–3.6)	3.09 (1.7–3.7)	<0.001
NT-proBNP (pg/ml)				3617 (67–476,022)	
CTR (%)				64 (56–76)	
EF (%)				51 (33.2–74)	
TRPG (mmHg)				38 (6.4–54.7)	

*BMI* body mass index, *HBV* hepatitis B virus, *HCV* hepatitis C virus, *LC* liver cirrhosis, *NT-proBNP* N-terminal prohormone of brain natriuretic peptide, *CTR* cardiothoracic ratio, *EF* ejection fraction, *TRPG* transtricuspid pressure gradient



**Fig. 2** Success rates for ultrasonographic measurements of left renal vein diameter and flow velocity. In the normal liver, LC without ascites, and congestive liver groups, the success rate for measuring

left renal vein diameter was more than 70%. In the LC with ascites group, the success rate for measuring left renal vein diameter and velocity was low ( $P=0.013$ ,  $P=0.016$ )



**Fig. 3** Normally distributed left renal vein and segmental renal vein diameter and flow velocity measurements in the normal liver group. The diameter and flow velocity of the left renal vein were normally distributed in the normal liver group ( $P=0.012$  and  $P<0.001$ , respectively)

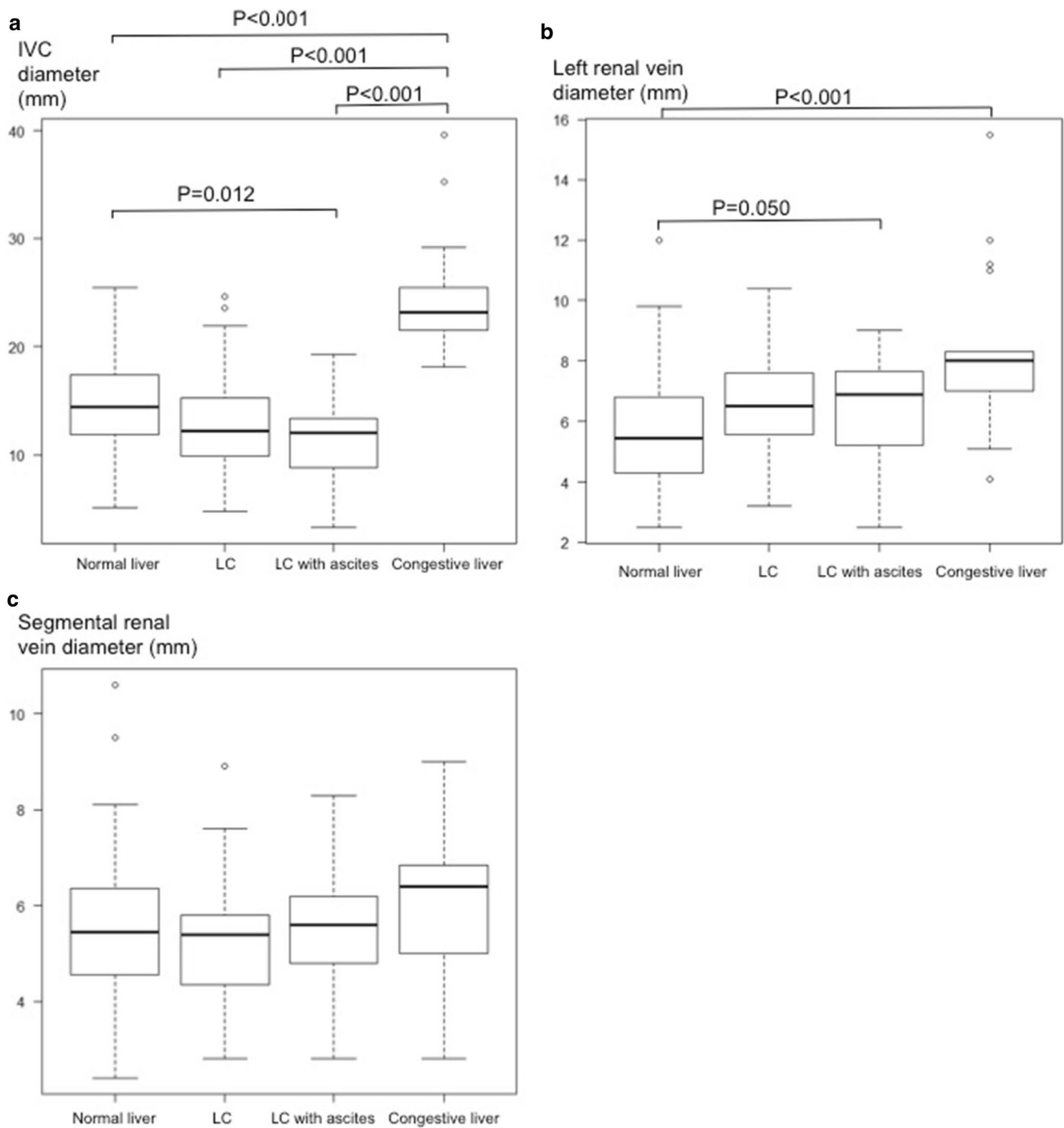
$P<0.001$ ), while the diameter of the left renal vein did not correlate with height ( $r=0.186, P=0.091$ ) or age ( $r=-0.024, P=0.816$ ).

#### **Group-by-group comparison of the IVC, left renal vein, and segmental renal vein diameter**

In the congestive liver group, the diameter of the IVC was larger compared to the normal liver, LC, and LC with ascites groups ( $P<0.001$ ). In the normal liver

group, the diameter of the IVC was larger compared to the LC with ascites group ( $P=0.012$ ). In the normal liver group, the diameter of the left renal vein was smaller compared to the LC with ascites and congestive liver groups ( $P=0.05$  and  $<0.001$ , respectively). The diameter of the left renal vein gradually increased in the following order: normal liver, LC, LC with ascites, and congestive liver groups ( $P<0.001$ ). There was no significant difference in segmental renal vein diameters between groups (Fig. 4).





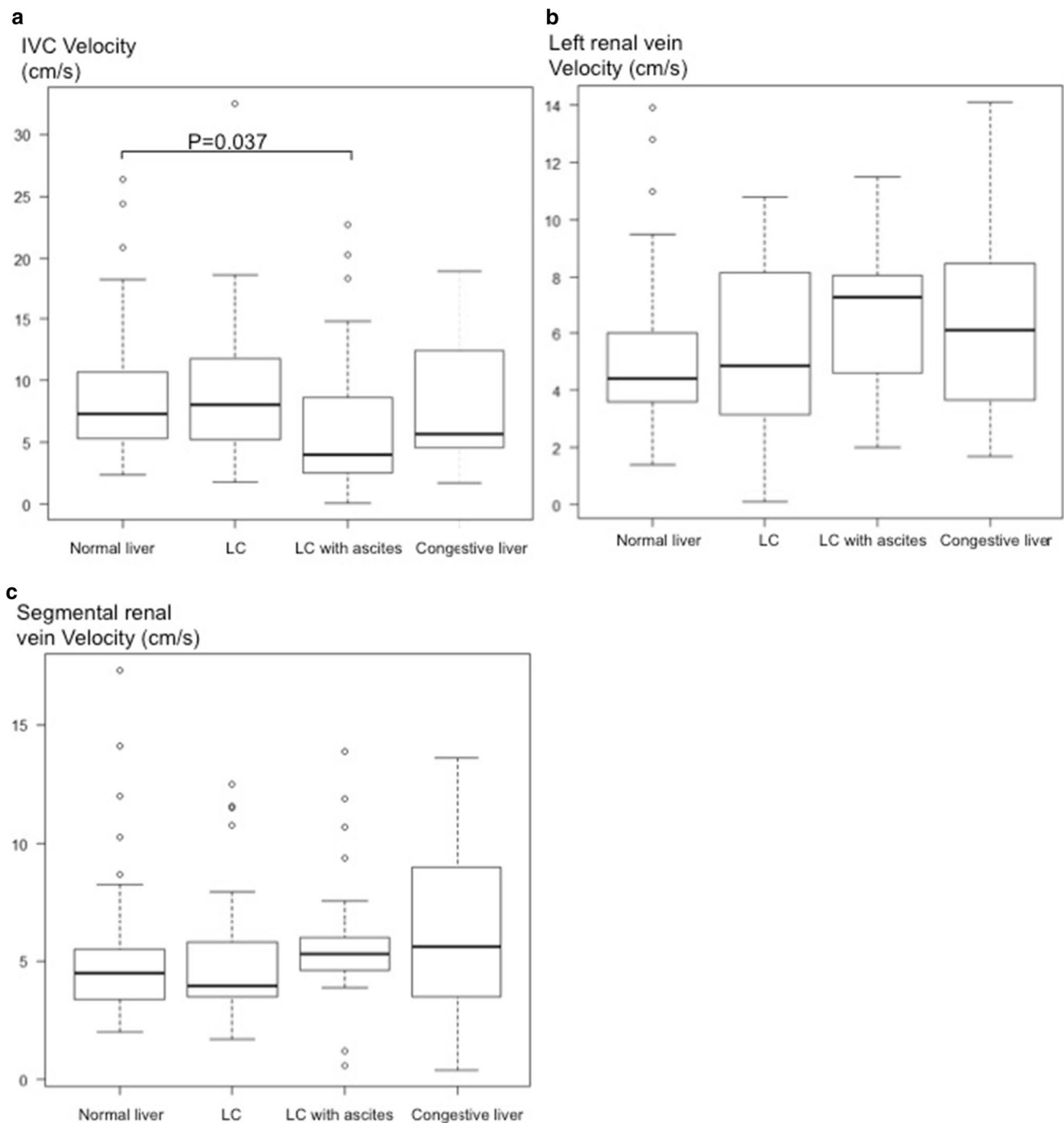
**Fig. 4** Group-by-group comparison of IVC, left renal vein, and segmental renal vein diameter. **a** The diameter of the IVC in the congestive liver group was larger than that in the normal, LC, and LC with ascites groups ( $P < 0.001$ ,  $P < 0.001$ ,  $P < 0.001$ , respectively). The diameter of the IVC in the normal group was larger than that in the LC with ascites group ( $P = 0.012$ ). **b** The diameter of the left renal

vein was gradually increased in the following order: normal, LC, LC with ascites, and congestive liver groups ( $P < 0.001$ ). The diameter of the left renal vein in the normal group was smaller than that in the LC with ascites and congestive liver groups ( $P = 0.05$  and  $P < 0.001$ , respectively). **c** No significant differences were found in the segmental renal vein diameters between groups

### Group-by-group comparison of IVC, left renal vein, and segmental renal vein flow velocities

In the normal liver group, the IVC flow velocity was

greater compared to the LC with ascites group ( $P = 0.037$ ). There was no significant difference in the flow velocity of the left renal vein and segmental renal vein between groups (Fig. 5).



**Fig. 5** Group-by-group comparison of IVC, left renal vein, and segmental renal vein flow velocities. **a** The flow velocity of the IVC in the normal group was faster than that in the LC with ascites group

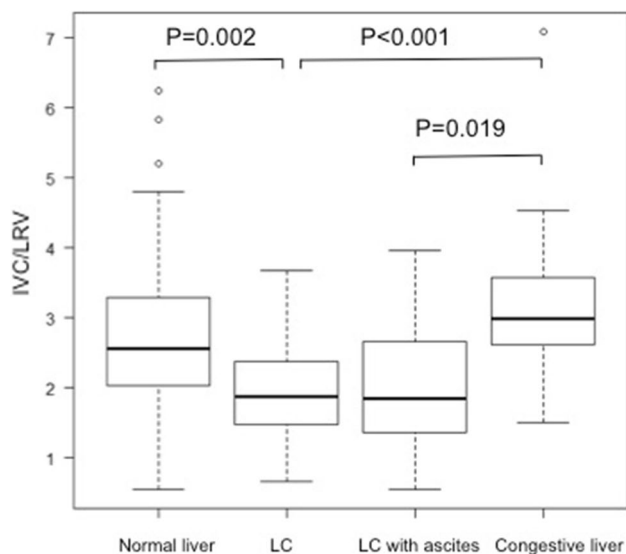
( $P=0.037$ ). **b** The flow velocity of the left renal vein showed no significant differences between groups. **c** The flow velocity of the segmental renal vein showed no significant differences between groups

### Group-by-group comparison of the ratio of IVC/left renal vein diameter

The median of the ratio of IVC/left renal vein diameter was 2.56 in the normal liver group, 1.87 in the LC group, 1.85 in the LC with ascites group, and 2.99 in the congestive

liver group. In the LC group, the ratio of IVC/left renal vein diameter was lower compared to the normal liver group ( $P=0.002$ ). In the congestive liver group, the ratio of IVC/left renal vein diameter was higher compared to the LC group (with or without ascites;  $P=0.019$  and  $P<0.001$ , respectively) (Fig. 6).





**Fig. 6** Group-by-group comparison of the ratio of IVC/left renal vein diameter. In the LC group, the ratio of IVC/left renal vein diameter was lower compared to the normal liver group ( $P=0.002$ ). In the congestive liver group, the ratio of IVC/left renal vein diameter was higher compared to the LC group (with or without ascites;  $P=0.019$  and  $P<0.001$ , respectively)

### Correlation between left renal vein diameter measured by ultrasonography and CT

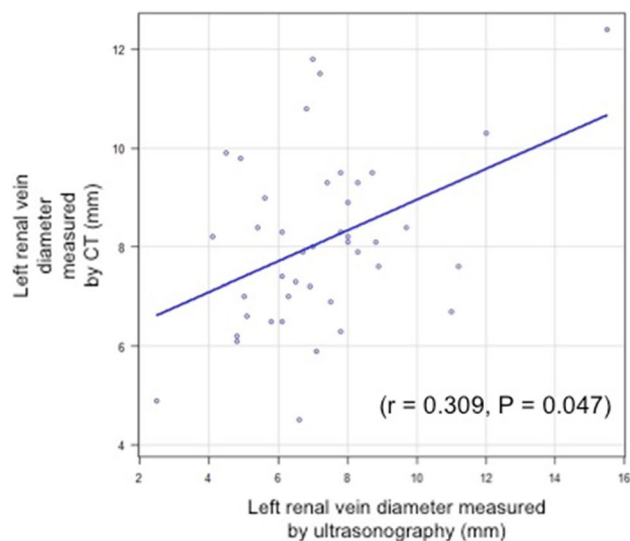
Of the patients with congestive liver and liver cirrhosis (with or without ascites), the left renal vein could be measured in 54 patients using CT. The left renal vein measured by ultrasonography and CT showed correlation ( $r=0.309$ ,  $P=0.047$ ) (Fig. 7).

### Correlation between left renal vein diameter and esophageal varices

There was no correlation between left renal vein diameter and esophageal varices ( $P=0.725$ ).

### Correlation between NT-proBNP, CTR, EF, and TRPG and IVC and left renal vein diameter

Left renal vein diameter and CTR showed correlation ( $r=0.575$ ,  $P=0.040$ ). IVC and CTR tended to correlate ( $r=0.574$ ,  $P=0.051$ ). NT-proBNP, EF, and TRPG did not correlate with IVC ( $P=0.501$ ,  $P=0.908$ , and  $P=0.585$ , respectively) and left renal vein diameter ( $P=0.256$ ,  $P=0.460$ , and  $P=0.244$ , respectively).



**Fig. 7** Correlation between the left renal vein diameter measured using ultrasonography and CT. The left renal vein measured using ultrasonography and CT showed correlation ( $r=0.309$ ,  $P=0.047$ )

## Discussion

In the present study, ultrasonographic measurements of renal vein diameter and flow velocity were taken. The success rate of measuring left renal vein diameter and flow velocity in the LC with ascites group was lower compared to other groups, since ascites may impair visualization of the vessels, causing difficulties in measuring the diameter and flow velocity.

Correlations between IVC diameter, height, and age have been reported in earlier studies; height has a positive correlation and age has a negative correlation with IVC diameter [7, 8]. The results in this study are consistent with those reports. The IVC diameter decreases, because the right atrium pressure decreases during aging [8]. On the other hand, the left renal vein is not affected by age, because it is a peripheral vein and far from the heart. This may explain why the diameter of the left renal vein was not correlated with height and age. Furthermore, this study found no correlation between the IVC and left renal vein diameter. Thus, the diameter and flow velocity of the renal veins are independent of body height or weight.

In addition, left renal vein diameter could be used as an independent factor for the evaluation of renal congestion, because it does not correlate with serum urea nitrogen and creatinine levels, which proves that these are not influenced by renal function [4]. Therefore, left renal vein diameter may be used as an independent parameter.

In a previous report, it was shown that the median overall survival for LC patients with a left renal vein diameter  $\geq 11$  mm, as measured by CT, was less than that for patients with a left renal vein diameter  $< 11$  mm [4]. This proves that a larger left renal vein diameter is associated with a poor prognosis in patients with LC.

The diameter of the left renal vein can be measured with ultrasonography. Compared to CT, ultrasonography has several merits, such as no radiation exposure, easy use at bedside, and repeatable examination.

It is known that heart failure causes renal venous hypertension leading to renal failure. The same mechanism could be underlying cirrhotic ascites. Compression of the IVC due to ascites probably causes congestion, which leads to the dilation of the left renal vein and renal congestion. Experiments on pigs showed that intra-abdominal pressure correlated with renal venous pressure [9, 10]. Studies also showed that an increase in ascetic fluid level increased the renal venous pressure in patients with LC [9]. Moreover, in the supine position, IVC compression by the liver also leads to increased IVC pressure [11]. Thus, in cases of decompensated LC, IVC compression by the liver and the ascetic fluid may cause renal venous hypertension.

Renal congestion has been studied in experimental models [12–15]. These studies showed that histological damage began with interstitial edema in the tubular epithelium, which progressed to necrosis [12]. Furthermore, studies also showed obstruction of the proximal tubule lumen by swollen cells, ischemia associated with the accumulation of cell debris [13], impaired renal cortical flow [14], tubulointerstitial injury, glomerular injury, and hypoxia in the medullary thick ascending limbs [15]. Recently, the significance of color Doppler ultrasonography for the evaluation of renal congestion has been reported [6, 16, 17]. A monophasic waveform in the intrarenal veins (abnormal, as opposed to biphasic or continuous) revealed by color Doppler ultrasonography indicates the presence of renal congestion, which is possibly associated with increased mortality [6, 17] or hospitalization due to heart failure [16]. Treatments for renal congestion have been developed recently [2, 18]. Tolvaptan [18, 19] and cell-free and concentrated ascites reinfusion therapy (CART) may improve renal congestion. Renal congestion also occurs during the anhepatic phase of liver transplantation [20] and in other conditions such as abdominal compartment syndrome [21].

The present study has several limitations. First, the sample size of the LC and congestive liver groups was relatively small and included patients of specifically Japanese ethnicity. Second, various types of ultrasonography equipment were used in this study. The methods of measuring IVC and renal veins were unified to minimize the possibility of error.

Third, the accuracy of the IVC and renal vein measurements was not sufficiently reliable in patients with ascites or obesity, because deep attenuation influences the sensitivity of color Doppler ultrasonography.

## Conclusion

In conclusion, this study showed that ultrasonography can be used for measuring the renal vein diameter and flow velocity. In patients with liver cirrhosis, the diameter of the left renal vein tended to increase and that of the IVC tended to decrease. Compression of the IVC by cirrhotic ascites causes congestion similar to that caused by CHF. Future research on renal congestion will benefit from this new method of measurement.

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## Declarations

**Conflict of interest** Masahiro Kaneko, MD; Naoki Matsumoto, MD, PhD; Mariko Kumagawa, MD; Yukinobu Watanabe, MD; Midori Hirayama, MD; Hiroshi Nakagawara, MD, PhD; Toshiki Yamamoto, MD, PhD; Masahiro Ogawa, MD, PhD; and Mitsuhiko Moriyama, MD, PhD declare that they have no conflicts of interest.

**Ethical statements** All procedures followed were in accordance with the ethical standards of the responsible committee on human experimentation (institutional and national) and with the Helsinki Declaration of 1964 and later versions.

**Informed consent** Informed consent was obtained from all patients for being included in the study.

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## 【和文の要約】 金子真大

### Renal vein measurement using ultrasonography in patients with cirrhotic ascites and congestive heart failure

(肝性腹水とうっ血性心不全患者における超音波を用いた腎静脈の測定)

#### 【目的】

うっ血性心不全による中心静脈圧の上昇は腎静脈圧の上昇、腎うっ血と関連しており、腎うっ血は心不全患者の死亡率の増加に関連しているといわれている。肝硬変 (Liver Cirrhosis : LC) による腹水でも、腹水により下大静脈 (Inferior vena cava : IVC) が圧迫され腎静脈圧が上昇し、腎うっ血が引き起こされる可能性がある。腹水を伴う LC 患者の左腎静脈径を CT で測定した研究では、左腎静脈径の拡大を認める症例で死亡率が高いことが示されている。これまで腎静脈の径と流速を超音波で測定した研究はほとんどなく、今回正常肝例、LC 患者、うっ血肝 (慢性心不全) 患者の腎静脈を測定することで、腎うっ血の病態を理解するために本研究を行なった。

#### 【対象】

2015 年 4 月から 2017 年 3 月の間に腹部超音波検査を施行された患者を対象とした。inclusion criteria として対象期間内に測定担当医師 2 名が施行した腹部超音波症例のうち、20 歳以上の 1633 例とした。exclusion criteria として維持透析例・腎摘出後、脂肪肝・慢性肝障害を除外し、残った 222 例を正常肝、肝硬変、うっ血肝 (慢性心不全) に分けた。さらに exclusion criteria として正常肝では心疾患既往を除外、肝硬変では胃腎・脾腎短絡路を認める症例を除外、うっ血肝では肝疾患既往を除外した。肝硬変は中等量以上の腹水の有無で 2 群に分けた。最終的に正常肝が 102 例、LC が 37 例、腹水を伴う LC が 30 例、うっ血肝が 17 例となった (図 1)。尚本研究は日本大学医学部附属板橋病院の臨床研究倫理審査委員会の承認を得た (RK-150609-6)。

## 【方法】

IVC および腎静脈の測定：IVC は心窩部縦走査で呼気時の最大直径（前後径）を肝下面で測定した。流速は 3 心拍が安定したところで超音波ドプラを用いて測定し、平均血流速度を使用した。左腎静脈の測定は上腹部横走査で行った。大動脈腹側を走行する左腎静脈の径と流速を測定し、径は上腸間膜動脈の交差部位より腎臓側で、最大直径を測定した。区域腎静脈は左肋間走査で測定し、カラードプラを参考にした。すべての測定は日本超音波医学会会員の特定の医師 2 名によって施行された。各種項目を測定し比較検討を行なった。

## 【統計学的手法】

相関の解析は Pearson の積率相関係数、Spearman の順位相関係数を用いた。各群の比較には Kruskal-Wallis 検定を用い、2 群ずつの比較には Bonferroni の多重比較を用いた。0.05 未満の P 値はすべての分析で統計的に優位であるとみなした。統計解析には EZR (R Foundation for Statistical Computing, Vienna, Austria) を用いた。

## 【結果】

- ・ 正常肝における左腎静脈径は年齢および身長と相関は認めなかった (Spearman の順位相関係数)。
- ・ 正常肝における IVC 径は年齢と負の相関 ( $r=-0.436$ ,  $P<0.001$ )、と身長と正の相関 ( $r=0.347$ ,  $P=0.001$ ) を認めた (Pearson の積率相関係数)。
- ・ IVC、左腎静脈、区域腎静脈径の各群の比較 (Kruskal-Wallis 検定)  
うっ血肝の IVC 径は正常肝、LC、腹水を伴う LC と比較して拡大していた ( $P<0.001$ )。腹水を伴う LC の IVC 径は正常肝と比較して縮小していた ( $P=0.012$ )。左腎静脈径は正常肝、LC、腹水を伴う LC、うっ血肝の順で増加しており ( $P<0.001$ )、腹水を伴う LC とうっ血肝の左腎静脈径は正常肝と比べ拡大していた ( $P=0.05$ ,  $<0.001$ )。区域腎静脈径は各群で有意差を認めなかった。
- ・ IVC、左腎静脈、区域腎静脈の流速の各群の比較 (Kruskal-Wallis 検定)  
腹水を伴う LC の IVC の流速は正常肝と比べ低下していた ( $P=0.037$ )。左腎静脈と区域腎静脈の流速は各群で有意差を認めなかった。

## 【考察】

本研究では腹水を伴う LC 患者で IVC 径の縮小と、IVC 流速の低下、また左腎静脈径の拡大を認めた。動物実験では腹腔内圧が腎静脈圧と相関するといわれており、また腹水の増加が腎静脈圧を増加させることも示されている。仰臥位では肝臓による IVC 圧迫で IVC 圧の上昇につながるという報告や、腹水を除去することで IVC の血流の改善がみられたと報告した (Matsumoto N, et al. Journal of Medical Ultrasonics. 2021; 48: 315-322.)。LC 患者では有効循環血漿量が低下しているために IVC 径が縮小傾向であることは推察される。腹水による IVC 圧迫が加わることで IVC の流速低下、血流のうっ滞を認め上流である左腎静脈径が拡大し、結果として腎うっ血を引き起こす可能性があることが示唆された。うっ血肝患者では IVC 径だけでなく、左腎静脈径も拡大していた。これらの超音波所見から腹水を伴う LC とうっ血肝は、病態は異なるが結果として腎うっ血をきたしている可能性が考えられた (図 2)。

腎血流に関して各群での有意差を認めなかった原因として、区域腎静脈は複数本あるため測定にばらつきが生じてしまう点や、腹水あり LC では左腎静脈の流速の測定成功率が 37%と腹水の影響で脈管描出が困難な例もあり、症例数が少ないことが考えられた。

近年腎うっ血を改善する治療としてトルバプタンや腹水濾過濃縮再静注法がある。左腎静脈が拡大しているような腹水を伴う LC 患者では腎うっ血をきたしている可能性を考え、腎うっ血の解除に取り組むことが重要である。実臨床において超音波は CT と比較して放射線被曝がなく、ベッドサイドで繰り返し検査可能であるなど複数の利点があり、今回超音波検査で左腎静脈が測定可能であったことは有益と考えられた。

## 【結論】

本研究では超音波検査を用いて、左腎静脈径、速度、IVC 径を測定することで、LC 患者の腎うっ血の病態把握を行なった。腹水を伴う LC 患者の左腎静脈径は拡大し、IVC 径が縮小していた。腹水による IVC の圧迫により腎うっ血を引き起こす可能性が示唆された。

【本研究の限界、将来の展望】

本研究の限界としては腹部超音波による左腎静脈測定の場合、腹水により深部の脈管描出が困難な例が存在することである。腹部超音波は簡便であるが、評価が難しい症例では CT など他の画像検査と合わせて評価していくのがよいと考える。

将来の展望としては左腎静脈径が拡大を示すような症例は、まず腎うっ血解除目的に腹水除去を行うことを考慮するなど、治療プランニング・モニタリングの一つとして使用できればと考えている。そのためにも今後は治療前後の腎静脈径や IVC 径の変化、また各種腎臓マーカーを比較することが重要と考えている。

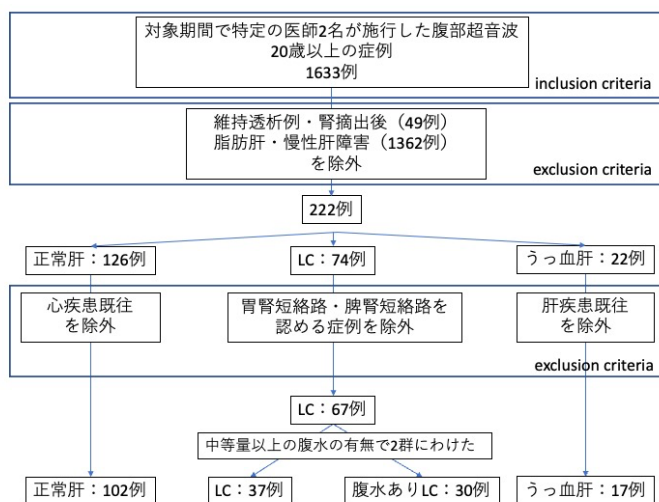


図 1

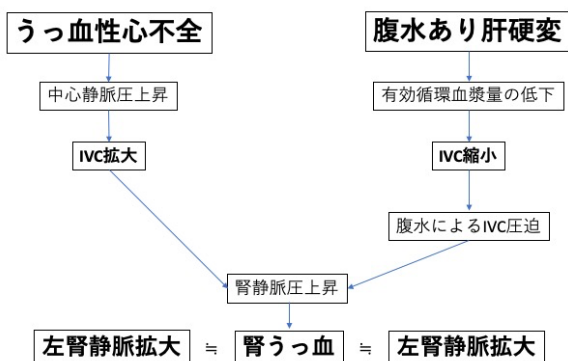


図 2