A sonographic Doppler study of the hepatic vein, portal vein and hepatic artery in liver cirrhosis: Correlation of hepatic hemodynamics with clinical Child Pugh score in Singapore

Azizah M Afif, Jason Pik-Eu Chang, Yan Y Wang, Simin D Lau, Fuzhen Deng, Shy Y Goh, Mar K Pwint, Chin C Ooi, Nanda Venkatanarasimha and Richard HG Lo

Abstract

Objective: Liver cirrhosis has been a rising complication of chronic liver disease in Singapore. Ultrasound has been widely accepted as a non-invasive imaging modality for the evaluation of hepatic haemodynamics. This study aims to correlate the Doppler ultrasound values with the progression of liver cirrhosis to allow further understanding and possible prediction of clinical events for timely intervention.

Methods: Study sample of 56 eligible patients with liver cirrhosis was divided according to their Child-Pugh clinical score into Child's A (n = 29 patients), B (n = 19 patients) and C (n = 8 patients). The maximum portal vein velocity, maximum hepatic vein velocity, maximum hepatic artery velocity and hepatic artery resistive index were assessed by Doppler ultrasound.

Results: The incidence of ascites increases with the severity of cirrhosis. Flattening of the hepatic vein waveforms was dependant on degree of liver cirrhosis. Maximum hepatic vein velocity was higher in cirrhotic patients (where $p = 0.05$). Maximum portal vein velocity was found to be lower in cirrhosis (where $p < 0.001$) and mean maximum portal vein velocity decreases as severity of cirrhosis worsens. Hepatic artery resistive index was significantly higher in cirrhosis (where $p < 0.001$). Significant association was found between maximum hepatic vein velocity and maximum hepatic artery velocity and significant negative correlation was observed with the maximum portal vein velocity and hepatic artery resistive index.

Conclusion: The study demonstrated that these parameters can supplement the evaluation of liver cirrhosis and will be able to distinguish the different grades of liver cirrhosis using Doppler ultrasound.

Keywords

Ultrasound, Doppler, liver cirrhosis, hepatic vein, portal vein, hepatic artery

Date received: 28 April 2017; accepted: 15 June 2017

Introduction

Liver cirrhosis is a consequence of progressive liver fibrosis resulting from chronic liver disease. It is a common problem worldwide and leads to significant morbidity and mortality from liver cancer and liver failure.^{1,2} Cirrhosis is seen in about 20% of hepatitis B Virus (HBV) carriers in Singapore and in the

Singapore General Hospital, Singapore, Singapore

Corresponding author:

Azizah M Afif, Singapore General Hospital, Block 2, Level 1, Ultrasound, Singapore 169608, Singapore.
Email: succubus311281@gmail.com

Asia Pacific region with the annual incidence of cirrhosis among chronic HBV patients reported to be approximately 1.0 to 2.4%.^{3,4} Severity of liver disease in patients with cirrhosis can be graded by Child-Turcotte-Pugh (CP) score,⁵ where the parameters of serum albumin and bilirubin, prothrombin time, presence of ascites and hepatic encephalopathy are accorded individual numerical points (ranging from 0 to 2) which are summed up to give the CP score. This scoring can be easily calculated at the patient's bedside and is useful in determining the short-term mortality rate in patients with cirrhosis and predicting the waiting list mortality of patients listed for liver transplantation.⁴

Ultrasound (US) has been shown to be a cost-effective, readily and widely available non-invasive imaging tool in evaluating patients with liver cirrhosis. Routine US evaluation can detect the development of portal hypertension, hepatocellular carcinoma and other complications of cirrhosis that may have a negative effect on the patient's prognosis. Doppler US is a very useful complement in evaluating the haemodynamic changes seen within a cirrhotic liver. Variation in hepatic haemodynamics demonstrated in liver cirrhosis has been shown in previous studies to correlate with the severity of cirrhosis. However, the extent of changes in Doppler US flow parameters in predicting the severity of liver cirrhosis remains unclear.

The objective of this study therefore is to evaluate the variation in Doppler flow hemodynamics in the hepatic portal, arterial and systemic circulation within cirrhotic livers as compared to normal livers, and to correlate these parameters with the clinical severity of liver cirrhosis. This may aid in providing additional information to better understand the observed changes in hepatic haemodynamics with progression of liver cirrhosis. This information may help to predict clinical events such as portal vein (PV) thrombosis, and to assist in timely intervention.

Materials and methods

Informed consent was obtained from all participants prior to inclusion in the study, with approval obtained from local Centralized Institutional Review Board (CIRB) and following their guidelines.

Patients

The study group of consecutive patients with liver cirrhosis was recruited from clinical referrals by hepatologists in our institution for abdominal US scan over a three-year period up to end of 2013. The abdominal US scan included Doppler US examination of the hepatic vessels in these patients. The severity of liver cirrhosis

was determined by the referring clinician using the CP score based on clinical and biochemistry parameters.

The control group comprises patients undergoing routine health screening with normal clinical and biochemistry parameters, as well as normal liver appearance on US. Patients with confirmed cardiac and respiratory disease (which may cause Doppler waveform changes^{6,7}), large hepatic mass compressing on any hepatic vessels, previous liver resection or hepatic surgery with hepatic vessel alterations, and patients undergoing dialysis were excluded from the study.

Methods

Equipment. Only two US systems were used in the study for scanning both the study group and control group participants – Toshiba Aplio XG (*Toshiba Medical Systems Corporation, Shimoishigami, Otawara-shi, Tochigi, Japan*) with 3.5 MHz curvilinear transducer and Philips iU22 (*Philips Medical System, Bothell, WA, USA*) with curvilinear broadband 1–5 MHz transducer. Both systems were equipped with automated velocity tracing of spectral waveforms to measure and compute maximum peak velocity and resistive index in order to reduce inter-operator variability. Doppler angle of insonation was maintained at less than 60° to ensure accuracy.

Operators. Six experienced sonographers able to scan independently performed Doppler US examinations on the participants in the study, and all were blinded to the severity of liver disease in terms of individual patient's clinical status and liver function biochemistry. Automated velocity tracing of spectral waveforms function was utilised to obtain the Doppler parameters in order to reduce inter-operator variability in the measurements.

Doppler measurements. All the participants were fasted for 7 to 8 hours prior to the Doppler US scan. A standardised scanning protocol was adopted for all patients with scanning performed via a right intercostal space to visualise and interrogate the hepatic vessels. Vessel interrogations of the right portal vein (RPV), main hepatic artery (MHA) and right hepatic vein (RHV) were performed with the patient lying in a left posterior oblique position to ensure absence postural influence.⁸ B-mode documentation of ascites, if present, was also obtained.

The RPV was chosen for evaluation as the left portal vein can be subjected to paraumbilical recanalisation^{7,9,10} in some cases of cirrhosis and within the liver. The RPV was interrogated with the patient in shallow normal respiration (Figure 1), and the maximum portal vein velocity (MPVV) was obtained

Figure 1. Right lobe of liver with Doppler gate (sample volume) located at the centre of the RPV vessel; interrogation was performed with the patient in shallow respiration.

Figure 2. Automated Doppler tracing of the RPV waveform with arrowhead indicating MPVV.

(Figure 2). It has been reported that minor degrees of respiratory undulations can be difficult to appreciate when scanning in suspended respiration.^{10–12}

The MHA was selected for interrogation as it supplies both the right and left hepatic arteries, and is seen at the portal triad before entering the hepatic hilum (Figure 3), and measurements of the maximum hepatic artery velocity (MHAV) and hepatic artery resistive index (HARI) were obtained with the patient in suspended respiration (Figure 4). HARI was calculated as the peak-systolic velocity (PSV) minus the end-diastolic velocity (EDV) divided by the PSV of the hepatic artery ($HARI = [PSV - EDV]/PSV$).¹³

Finally, the RHV was selected for interrogation as the middle and the left HVs often join each other before draining into the inferior vena cava (IVC).¹⁴ Measurements in RHV were obtained at a distance of 2–3 cm from its confluence with the IVC with the patient in suspended normal expiration¹⁰ (Figure 5). Three measurements of maximum hepatic vein velocity (MHVV) were obtained due to possible variability of the HV velocity, and the average calculated to represent the mean MHVV (Figure 6). The configuration of the

Figure 3. Right lobe of the liver with Doppler gate (sample volume) at the centre of MHA at the hepatic portal hilum.

Figure 4. Automated velocity tracing of MHA waveform measuring MHAV and HARI. MHAV: maximum hepatic artery velocity; HARI: hepatic artery resistive index.

Figure 5. Right lobe of liver with Doppler gate (sample volume) at RHV 2–3 cm from its confluence with the IVC with the patient in normal suspended expiration.

HV Doppler waveforms was also analysed and categorised as being triphasic or monophasic patterns. Triphasic waveform pattern (Figure 6) is characterised by the presence of four components: a retrograde 'A' wave, an antegrade 'S' wave followed by a transitional

‘V’ wave (which may be antegrade, retrograde, or neutral), and finally an antegrade ‘D’ wave. In contrast, flat, blunted or monophasic waveform is considered when no phasicity in the waveform is observed.⁶

Statistical analysis. The primary objective of the study was to evaluate the Doppler flow parameters of hepatic vessels and investigate any correlation of observed changes with the severity of liver cirrhosis based on CP classification. Statistical analysis was performed using SPSS 17.0 (SPSS Inc., Chicago, IL, USA). Differences in categorical and continuous variables between the studied groups were analysed by using Mann-Whitney U and later the correlation of the parameters were correlated with each other to examine if the parameters may affect each other, by using Spearman’s correlation (ρ). Results are expressed in mean $\pm$ SD and comparisons with p -value ≤ 0.05 were considered as statistically significant.

Figure 6. Normal RHV triphasic waveform in a control group patient with automated velocity tracing to obtain MHW.

Results

Fifty-seven consecutive patients with liver cirrhosis were recruited for the study. One patient was excluded as there was total thrombosis of the right portal vein. The study group therefore comprised 56 eligible patients (38 male and 18 female patients) with mean age 61.4 ± 23.6 years. Severity of liver cirrhosis was based on the CP score⁵: Child-Pugh grade A ($n=29$), Child-Pugh grade B ($n=19$), and Child-Pugh grade C ($n=8$). The control group comprised 26 health screening patients with normal clinical and biochemistry results (12 male and 14 female patients) with mean age of 52.3 ± 25.7 years.

Table 1 outlines patient demographics, laboratory and clinical parameters of the subject population.

Portal vein

The portal vein flow velocity (MPVV) was demonstrated to be significantly lower in the study group participants as compared to the control group.

The mean MPVV for the study group patients measured at 13.9 ± 7.6 cm/s vs. 19.8 ± 5.1 cm/s in the control group ($p < 0.001$) (Figure 7). With increasing severity of cirrhosis within the study group patients, the mean MPVV decreases as the CP score increases where: 16.5 ± 3.6 cm/s ($p = 0.01$) in Child’s A, 14.2 ± 4.2 cm/s ($p < 0.001$) in Child’s B, and 3.7 ± 14.3 cm/s ($p = 0.001$) in Child’s C (Figure 8).

The median MPVV for the control group was 19.7 cm/s whilst that for study group was 15.5 cm/s (Figure 7). Similar to the mean MPVV changes observed within the study group patients, the median MPVV for Child’s A was 16.5 cm/s, B was 13.5 cm/s

Table 1. Laboratory and clinical parameters in subject population

	Cirrhotics, N = 56			Controls, N = 26	p
Age (years)	61.4 ± 11.0			51.4 ± 12.0	<0.001
Gender (male)	38 (67.9%)			12 (46.2%)	0.062
Child class (A/B/C)	29 (51.8%)	19 (33.9%)	8 (14.3%)	–	–
Albumin (g/L)	33.5 ± 7.4			42.8 ± 2.7	<0.001
Bilirubin ($\mu\text{mol/L}$)	30.2 ± 22.9			15.9 ± 3.7	<0.001
Prothrombin time (s)	12.1 ± 2.0			–	–
ALT (U/L)	37.1 ± 31.1			20.0 ± 5.5	<0.001
Platelet count ($\times 10^9/\text{L}$)	129.4 ± 72.5			261.6 ± 57.3	<0.001

Figure 7. MPVV in study group vs control group where $\ddagger p < 0.001$ (mean $\pm$ SD). MPVV: maximum portal vein velocity.

Figure 8. MPVV in study group Child-Pugh A, B, and C vs. control group (mean $\pm$ SD), where $*p = 0.01$, $\dagger p < 0.001$, $\ddagger p = 0.001$, respectively, for each Child-Pugh group. MPVV: maximum portal vein velocity.

and C was 9.8 cm/s (Figure 8) showing progressive reduction of portal vein velocity as severity of cirrhosis worsens. In the study group, we encountered two patients with partial portal vein thrombosis – one with Child's B and another with Child's C cirrhosis, with MPVV of 11.3 cm/s and 15.5 cm/s, respectively.

Reversed portal vein velocity was found in three out of eight (37.5%) participants with Child's C cirrhosis (MPVV measuring -11.5 cm/s, -13.3 cm/s and -13.8 cm/s).

Hepatic artery

The MHAV was not found to be significantly different between the study group and the control group patients ($p = 0.06$), nor between the different subsets of patients (Child's A, B and C) within the study group.

Figure 9. HARI in study group Child-Pugh A, B, and C vs. control group (mean $\pm$ SD), where $\ddagger p < 0.001$ for each Child-Pugh group. HARI: hepatic artery resistive index.

Figure 10. HARI in study group vs. control group, where $\ddagger p < 0.001$ (mean $\pm$ SD). HARI: hepatic artery resistive index.

In contrast, HARI was significantly higher in the study group as compared to the control group (0.8 ± 0.06 ; $p < 0.001$). In addition, HARI was demonstrated to be significantly higher in all the CP subsets of cirrhosis where $p < 0.001$ (Figure 9).

With reference to Figure 10, the median HARI value for the study group patients was 0.81 in contrast to the control group individuals where the median HARI did not exceed 0.71.

Hepatic Vein

In the control group, the HV demonstrated normal triphasic waveform pattern (Figure 6) in all 26 participants. However, in the study group, the HV in 11 patients (19.6%) showed flattened monophasic waveform patterns (Figures 11 and 12). Further stratification

of this group of 11 patients revealed four were Child's A (out of 29 patients (13.8%) classified as Child's A), three were Child's B (out of 19 patients (15.8%) in this category), and four were Child's C (out of eight patients (50%) in this category).

The incidence of blunted waveform showed correlation with the severity of cirrhosis as presented in Table 2. Thus, HV waveform flattening was significantly more commonly seen in patients with advanced cirrhosis (i.e. Child's C).

With regard to the HV velocity, it was found that the study group MHVV in liver cirrhosis was significantly higher as compared to the control group (Figure 13), where $p = 0.05$.

Table 3 summarises hepatic haemodynamics in both the control group and the study group (with further stratification of study group into Child's A–C cirrhosis).

Correlation between parameters. There was significant positive correlation in cirrhotic individuals of

Figure 11. Patient 2 in the study group with blunted waveform and loss of triphasic waveform pattern.

Figure 12. Patient 4 in study group showing markedly blunted monophasic waveform pattern.

MHV with MHAV where $p = 0.039$, $\rho = 0.229$. There was significant negative correlation between MPVV and HARI where $p < 0.001$, $\rho = -0.47$. MHAV and HARI also had correlation where $p = 0.001$, $\rho = 0.352$.

In the control group, there was no correlation among the parameters studied.

Discussion

Liver cirrhosis is known to affect and alter the hepatic vascularisation, and hepatic circulation can differ with varying grades of cirrhosis. Using Doppler interrogation, we have observed haemodynamic changes with statistically significant values in our study group of patients with severe liver cirrhosis.

Normal portal vein velocity (MPVV) ranges between 20 cm/s and 40 cm/s. Reduced portal vein velocity was seen in our study with a mean MPVV of 13.9 cm/s in cirrhotic patients (Figure 7). Further stratification of the study group of patients into subsets of liver cirrhosis using the CP classification clearly showed a greater degree of reduced portal vein flow velocity with worsening of liver cirrhosis – mean MPVV for Child's A patients was 16.5 cm/s, Child's B was 14.2 cm/s and

Figure 13. MHVV in study group vs. control group where $*p = 0.05$ (mean $\pm$ SD). MHVV: maximum hepatic vein velocity.

Table 2. Hepatic vein waveforms in study population n (%)

Waveform	Non-cirrhotic controls, n = 26	Liver cirrhosis participants Child-Pugh		
		A, n = 29	B, n = 19	C, n = 8
Triphasic	26 (100%)	25 (86.2%)	16 (84.2%)	4 (50%)
Blunted	0	4 (13.8%)	3 (15.8%)	4 (50%)

Table 3. Hepatichaeodynamics in relation to different stages of liver cirrhosis

	Control group	Study Group	Child Pugh A	Child Pugh B	Child Pugh C
MHVV [cm/s]	39.5 ± 12.2	52.0 ± 25.9*	50.4 ± 22.7	57.8 ± 30.8*	44.2 ± 24.9
MHVV Range	16.5–61.3	16.6–145.0	18.3–93.5	25.5–145.0	16.6–98.1
MPVV [cm/s]	19.8 ± 5.1	13.9 ± 7.6‡	16.5 ± 3.6†	14.2 ± 4.2‡	3.7 ± 14.3‡
MPVV Range	11.7–31.1	(–) 13.8–24.7	10.0–24.7	8.2–23.4	(–) 13.8–21.6
MHAV [cm/s]	44.3 ± 15.8	54.7 ± 26.7	47.3 ± 16.9	52.6 ± 20.6	86.3 ± 44.7†
MHAV Range	19.6–81.7	13.8–177.0	21.2–86.1	13.8–109.0	32.8–177.0
HARI	0.5 ± 0.1	0.8 ± 0.06‡	0.8 ± 0.07‡	0.8 ± 0.05‡	0.8 ± 0.06‡
HARI Range	0.3–0.7	0.7–0.9	0.7–0.9	0.7–0.9	0.7–0.9

Man U Whitney test * $p \leq 0.05$; † $p \leq 0.01$; ‡ $p \leq 0.001$.

MHVV: maximum hepatic vein velocity; MPVV: maximum portal vein velocity; MHAV: maximum hepatic artery velocity; HARI: hepatic artery resistive index.

Child's C was 3.7 cm/s (Figure 8). This is in concordance with published studies by Taourel et al.¹⁵ and Ljubicić et al.¹⁶ Intrahepatic fibrosis in liver cirrhosis results in higher resistance to the portal vein flow, thereby reducing portal inflow. In our study group, reversal of portal vein flow was detected in three Child's C patients; partial portal vein thrombosis was observed in one Child's B and one Child's C patient. High incidence of portal vein thrombosis has been attributed to the flow stasis that develops as portal hypertension worsens thereby causing development of thrombus.^{17,18} There may not be specific symptoms but chronic intrahepatic microthrombosis, termed "parenchymal extinction", has been postulated as a pathogenic mechanism in cirrhosis which may aggravate fibrosis inducing atrophy.¹⁹ Cirrhosis nodules are found to have a "porto-centric" pattern with enlargement of cirrhotic nodules adjacent to major portal systems. The levels of both pro- and anti-coagulation proteins are known to be reduced under conditions of hepatic synthetic impairment in cirrhotic patients which may also further account for the higher rate of thrombosis occurring in severe cirrhosis.²⁰

With three of the eight (37.5%) Child's C patients in our study group demonstrating reversed portal vein flow and none in Child's A or B categories, it is evidently clear that severe liver cirrhosis is contributory to portal vein flow reversal as seen in our study. This corresponds to similar findings by Mittal et al.⁹ who reported reversal of portal vein flow (hepatofugal flow) only in patients with more advanced liver disease. The observation of reversed portal vein flow in cirrhosis has been elegantly explained by Sherlock²¹ who

illustrated the concept of the obstruction of the intra-hepatic portal and hepatic arterial flow in advanced liver disease. This may result in blood entering the liver finding it easier to exit via the portal vein instead of the normal route via HVs. With reversal of portal vein flow, it has been pointed out that diversion of hepatic arterial blood away from the liver parenchyma and into the main portal vein would deprive the liver cells of oxygen and other nutrients.

The increase in HARI in liver cirrhosis was found to be statistically significant in our study. This helps in differentiating cirrhotic participants (where HARI values were above 0.7) from the non-cirrhotics (HARI values were less than or equal to 0.7). HARI therefore has been shown to be a useful discriminator of cirrhosis. Sacerdoti et al.²² stated that the hepatic arterial resistance in cirrhosis could be altered through the same mechanisms that were responsible for the increase in portal resistance, i.e. distortion of hepatic architecture caused by cirrhosis, reduction of vascular space and other still undefined factors.

HV spectral waveform was also demonstrated to be altered in cirrhotic patients in our study. Eleven (19.6%) of the 56 patients in the study group of cirrhotic patients demonstrated flattened HV Doppler spectral waveform. We have also found that the incidence of dampening of HV waveforms correlates with the severity of cirrhosis, and as there is greater likelihood of observing hepatic venous outflow obstruction in higher grade cirrhosis. Studies by Colli et al.,¹ von Herbay et al.²³ and Arda et al.²⁴ reported incidence of flattened HV waveform in patients with cirrhosis in 57%, 43% and 18.2% respectively. The phenomenon

Table 4. Recommended values in Doppler Ultrasound Liver (Ultrasound Section, Singapore General Hospital)

Parameter	Normal range	Values in cirrhosis	Significance
Portal vein velocity	20–30 cm/s	≤ 15 cm/s	$p < 0.001$
Hepatic artery resistive index	0.5–0.7	≥ 0.71	$p < 0.001$
Hepatic vein velocity	≤ 60 cm/s	> 60 cm/s	$p = 0.05$
Hepatic vein waveform	Triphasic	Blunted, monophasic	–

has been attributed to increased parenchymal stiffness impairing HV compliance, thereby resulting in the loss of HV phasicity.²⁵

Increased HV velocity was seen in our study group of cirrhotic patients with a mean MHVV of 52 cm/s in cirrhotic participants vs. 39.5 cm/s in the non-cirrhotic group. This shows that elevated HV velocity can be a useful parameter to help identify cirrhotic individuals, coupled with blunting of HV spectral waveform.

Based on our findings from this study on the cohort of local patients, we have therefore adopted a recommended set of values for the various parameters in our clinical practice. These values are as detailed in Table 4.

We also analysed our data to see if we could demonstrate any correlation between the hepatic flow parameters in our study group of patients – MPVV, MHVV, MHAV and HARI. Interestingly, our study data showed a statistically significant positive correlation of MHVV with MHAV in the study group patients – as the velocity of the hepatic artery increases, the HV velocity also increases indicating that change in the hepatic artery velocity has an impact on the HV outflow velocity. Hence, we conclude that arterialisat-ion occurring in the cirrhotic liver will increase hepatic inflow which will also result in a higher hepatic outflow. In contrast, we did not detect any statistically significant correlation of MHAV with portal flow or MPVV.

In addition, there was a statistically significant negative correlation of the portal vein velocity (MPVV) with HARI where, as the portal vein inflow decreases, the vascular resistance of the hepatic artery increases. The portal vein provides 75–80% of the liver blood inflow with the other 20–25% inflow contributed by the hepatic artery. It has been suggested that as the portal vein supply decreases, the buffered flow is contributed by the hepatic artery to counter resulting liver parenchymal hypoxia.^{26,27} Angiogenesis develops and the hyperdynamic state causes the hepatic artery resistance to be elevated.²²

Correlation test was also performed on hepatofugal portal vein flow patients, showing mild association of HARI with MHVV ($p = 0.002$, $\rho = -0.553$). This can be explained by the increased portal vein pressure

(due to obstruction of hepatic venules and sinusoids by parenchymal fibrosis) in cirrhotic liver, resulting in opening up of various collateral pathways (arterio-portal and portosystemic shunting)²⁸; reversal of blood flow in the portal vein may develop when intra-hepatic resistance exceeds the resistance of portosystemic collaterals.⁹

There are some limitations in our study. Firstly, two different US systems were used to scan both control and study group patients, owing to significant patient volume and availability of US scanners in the Ultrasound Section of our department. No prior audit was carried out by scanning the same patient/s using these two US scanners to look for any significant difference in the measurement of the parameters being studied; both units had regular preventive maintenance servicing by our hospital Bio-medical Engineering Department qualified personnel with regular phantom tests performed. Nonetheless, we minimised any possible imaging variability by limiting the use to only these two US scanners for the study, both units being the most advanced versions currently available at the time of the study and equipped with the latest and very similar software including automated velocity tracing. Secondly, patients were evaluated by the referring clinician and individual patient's CP score obtained at the time of the clinic visit; to minimise any change in patient's clinical status, US scans in all the patients included in our study were acquired within six months of the clinic visit. Thirdly, the population size of recruited patients with Child's C status was small; statistical analysis therefore may not be so robust and we recommend a multi-centre trial involving a larger patient cohort for any future study.

Summary/conclusion

Doppler US of hepatic vessels is a non-invasive and cost-effective imaging modality, and is now an established routine in the US evaluation of the liver in both normal and cirrhotic patients. Haemodynamic changes in the liver vascularity affecting the portal and HVs and also hepatic artery are well recognised and correlate

with the severity of cirrhosis based on CP score. Our study has shown statistically significant changes in MPVV, MHVV and HV spectral waveform, and HARI with increasing severity of cirrhosis, and can help to prognosticate patients with liver cirrhosis.

Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

Ethical approval

CIRB 2010/705/C

Guarantor

N/A

Contributors

N/A

Acknowledgments

N/A

References

- Colli A, Fraquelli M, Andreoletti M, et al. Severe liver fibrosis or cirrhosis: accuracy of US for detection – analysis of 300 cases. *Radiology* 2003; 227: 89–94.
- Cokkinos DD and Dourakis SP. Ultrasonographic assessment of cirrhosis and portal hypertension. *Curr Med Imaging Rev* 2009; 5: 62–70.
- Guan R. Hepatitis B virus infection in Singapore. *Gut* 1996; 38(Suppl 2): S13–S17.
- Guan R and Lui HF. Treatment of hepatitis B in decompensated liver cirrhosis. *Int J Hepatol* 2011; 2011: 918017.
- Pugh RN, Murray-Lyon IM, Dawson JL, et al. Transection of the oesophagus for bleeding oesophageal varices. *Br J Surg* 1973; 60: 646–649.
- Scheinfeld MH, Bilali A and Koenigsberg M. Understanding the spectral Doppler waveform of the hepatic veins in health and disease. *Radiographics* 2009; 29: 2081–2098.
- Sudhamshu KC, Matsutani S, Maruyama H, et al. Doppler study of hepatic vein in cirrhotic patients: correlation with liver dysfunction and hepatic hemodynamics. *World J Gastroenterol* 2006; 12: 5853–5858.
- Chuo LS, Mahmud R and Salih QA. Color Doppler Ultrasound examination of the main portal vein and inferior vena cava in normal Malaysian adult population: a fasting and post prandial evaluation. *J Cardiovasc Res* 2004; 2(2): <http://ispub.com/IJCVR/2/2/9055> [Online version].
- Mittal P, Gupta R, Mittal G, et al. Association between portal vein color Doppler findings and the severity of disease in cirrhotic patients with portal hypertension. *Iran J Radiol* 2011; 8: 211–217.
- Robinson KA, Middleton WD, Al-Sukaiti R, et al. Doppler sonography of portal hypertension. *Ultrasound Q* 2009; 25: 3–13.
- Görg C, Riera-Knorrenschild J and Dietrich J. Pictorial review: colour Doppler ultrasound flow patterns in the portal venous system. *Br J Radiol* 2002; 75: 919–929.
- Swart J and Sheth S. Role of vascular ultrasound in the evaluation of liver disease. *Ultrasound Clin* 2007; 2: 355–375.
- McNaughton DA and Abu-Yousef MM. Doppler US of the liver made simple. *Radiographics* 2011; 31: 161–188.
- Skandalakis JE, Skandalakis LJ, Skandalakis PN, et al. Hepatic surgical anatomy. *Surg Clin North Am* 2004; 84: 413–435.
- Taourel P, Blanc P, Dauzat M, et al. Doppler study of mesenteric, hepatic, and portal circulation in alcoholic cirrhosis: relationship between quantitative Doppler measurements and the severity of portal hypertension and hepatic failure. *Hepatology* 1998; 28: 932–936.
- Ljubicić N, Duvnjak M, Rotkvić I, et al. Influence of the degree of liver failure on portal blood flow in patients with liver cirrhosis. *Scand J Gastroenterol* 1990; 25: 395–400.
- Boyer TD. Management of portal vein thrombosis. *Gastroenterol Hepatol* 2008; 4: 699–700.
- Nyandak T, Prakash P, Das U, et al. Portal vein thrombosis – clinical profile. *J Indian Academy Clin Med* 2011; 12: 134–140.
- Tsochatzis EA, Senzolo M, Germani G, et al. Systematic review: portal vein thrombosis in cirrhosis. *Aliment Pharmacol Ther* 2010; 31: 366–374.
- Kinjo N, Kawanaka H, Akahoshi T, et al. Portal vein thrombosis in liver cirrhosis. *World J Hepatol* 2014; 6: 64–71.
- Sherlock S. Portal circulation and portal hypertension. *Gut* 1978; 19: 70–83.
- Sacerdoti D, Merkel C, Bolognesi M, et al. Hepatic arterial resistance in cirrhosis with and without portal vein thrombosis: relationships with portal hemodynamics. *Gastroenterology* 1995; 108: 1152–1158.
- von Herbay A, Frieling T and Häussinger D. Association between duplex Doppler sonographic flow pattern in right hepatic vein and various liver diseases. *J Clin Ultrasound* 2001; 29: 25–30.
- Arda K, Ofelli M, Calikoglu U, et al. Hepatic vein Doppler waveform changes in early stage (Child Pugh A) chronic parenchymal liver disease. *J Clin Ultrasound* 1997; 25: 15–19.
- Farrant P and Meire HB. Hepatic vein pulsatility assessment on spectral Doppler ultrasound. *Br J Radiol* 1997; 70: 829–832.
- Zekanovic D, Ljubicić N, Boban M, et al. Doppler ultrasound of hepatic and system hemodynamics in patients with alcoholic liver cirrhosis. *Dig Dis Sci* 2010; 55: 458–466.
- Amitrano L and Guardascione MA. Management of portal vein thrombosis in cirrhotic patients. *Mediterr J Hematol Infect Dis* 2009; 1: e2009014.
- Iranpour P, Lall C, Houshyar R, et al. Altered Doppler flow patterns in cirrhosis patients: an overview. *Ultrasonography* 2016; 35: 3–12.