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RESEARCH

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Hepatic arterial hemodynamics and model for end-stage liver disease (MELD) scores in chronic liver disease: insights from Doppler ultrasonography

Sasmita Tuladhar[1], Shailendra Katwal[2*], Ghanshyam Gurung[3] and Umesh Khanal[3]

Abstract

Background Doppler ultrasonography is essential to provide insights into hemodynamic alterations and liver function changes in pre-cirrhotic and cirrhotic patients. Utilizing Doppler examinations, this study aims to explore the correlation between hepatic arterial hemodynamics and Model for end-stage liver disease (MELD) scores in chronic liver disease patients.

Methods A study of 50 chronic liver disease patients included sonographic assessments, measuring liver, portal vein size, and flow. Hepatic artery velocity, resistive index (RI), pulsatility index (PI), and acceleration time (AT) were evaluated. Biochemical parameters (serum bilirubin, creatinine, INR) were used to calculate MELD scores, compared with different Doppler sonographic parameters.

Results The study found a mean peak systolic velocity (PSV) of 107.42 ± 48.10, with end-diastolic velocity (EDV) of 26.40 ± 14.68, RI of 0.74 ± 0.06, and PI of 1.47 ± 0.24. The mean MELD score was 19.28 ± 6.09. Correlations between MELD scores and PSV, EDV, RI, PI, and AT did not yield statistically significant correlations. 80% of subjects displayed high RI (> 0.7) values in the hepatic artery, and a significant correlation was found between portal vein thrombosis and hepatic artery PSV and RI ($p < 0.05$).

Conclusions Hepatic artery RI and PSV show a significant correlation with portal vein thrombosis. Doppler ultrasonography, while not directly tied to MELD scores, is valuable for non-invasive liver disease monitoring when invasive methods are impractical. Further research is needed to unravel the relationships between hemodynamic changes, MELD scores, and clinical outcomes in a broader patient population.

Keywords Chronic liver disease, Doppler sonography, Hepatic artery velocity, MELD score

Background

Chronic liver diseases (CLDs) involve persistent inflammation, leading to biochemical and histopathological abnormalities. Cirrhosis, characterized by fibrosis and nodules, results from various causes, with alcohol being predominant [1]. A significant global health issue, liver cirrhosis ranks 9th in causes of death, with a substantial impact in Nepal despite limited national data [2]. A study by AK Mishra et al. found that alcoholic liver disease (60.8%) was the most common cause of chronic liver

*Correspondence:
Shailendra Katwal
shailendrakatwal@gmail.com
[1] Department of Radiology, Kanti Children Hospital, Kathmandu, Nepal
[2] Department of Radiology, Dadeldhura Subregional Hospital, Dadeldhura, Tufandada, Nepal
[3] Department of Radiology, Maharajgunj Medical College, Kathmandu, Nepal

disease in 130 patients admitted to Bir Hospital's liver unit [3]. Although the community prevalence of HBV and HCV infections is low, they account for 40% and 14% of liver cirrhosis, respectively. A study of 430 liver biopsies from 1990 to 1997 revealed that alcohol and hepatic IVC disease (HVD) were the two predominant causes of liver diseases in Nepal. Alcohol was responsible for 44.6% of cirrhosis and 20.4% of other liver diseases, while HVD for 34% of cirrhosis and 29.3% of other liver diseases [4]. Patients with chronic liver disease on orthostatic liver transplantation waiting lists face improved prognostic evaluation through scoring systems like Child–Pugh, MELD, and MELD-Na. The shift from Child-Turcotte-Pugh (CTP) to MELD in allocation policies has reduced waitlist mortality, with the MELD score proving to be an excellent predictor of 3-month mortality based on objective biochemical parameters [5, 6].

Doppler ultrasonography is crucial for evaluating pre-cirrhotic and cirrhotic patients, especially those with chronic liver disease. It assesses hepatic blood flow to detect hemodynamic changes associated with cirrhosis and portal hypertension [7]. Understanding different flow patterns can reinforce cirrhosis diagnosis, aid in staging, and provide prognostic information for therapy direction. The three major vessels interrogated at liver Doppler US are hepatic arteries, hepatic veins, and portal veins [8]. The arterial resistance index (RI) is a widely used Doppler US parameter for estimating intrahepatic vascular resistance in clinical studies. A RI > 0.70 is linked to liver fibrosis [6], while cirrhosis causes portal inflow to decrease and hepatic arterial inflow increases due to the "hepatic arterial buffer response"[7].

Percutaneous biopsy is the gold standard for diagnosing cirrhosis and fibrosis, but it is an invasive procedure and can cause false negative results in 20–30% of patients due to sampling mistakes [9]. Early detection of chronic liver disease prevents complications like variceal bleeding and hepatic encephalopathy. Staging liver cirrhosis guides therapy. This study explores the correlation between MELD score and hepatic arterial hemodynamics, utilizing Doppler ultrasonography for prognostic insights in patients awaiting transplantation.

Methods

This prospective, observational analytical study was carried out in the Department of Radiology and Imaging from August 2018 to July 2019. Ethical approval for conducting the study was taken from the Institutional Review Committee (IRC) (approval number 44 (6/11) E^2 075/076). This study followed the STROCSS (Strengthening The Reporting Of Cohort Studies in Surgery) 2021 checklist for cross-sectional studies [10]. By the Declaration of Helsinki, the study is registered retrospectively in the research registry with registration number, researchregistry9394.

Sample size

A sample size of 28 was calculated using the bivariate correlative analysis formula with a correlation coefficient of 0.5%, level of significance of 5%, and power of study of 80%. The study was conducted in 50 patients (Fig. 1).

Inclusion and exclusion criteria

The study includes patients aged 18 and above with decompensated chronic liver disease, sonographic features of chronic liver disease, histological diagnosis (if available), and history of hepatitis B or C with sonographic evidence. Exclusion criteria include acute liver disease, known liver neoplasms, focal liver mass, metastatic liver disease, and previous hepatic surgery.

Data collection

Informed written consent was taken from the patients meeting the inclusion criteria. Biochemical parameters such as serum bilirubin, serum creatine, and PT/INR were obtained, and the MELD score was calculated as per the formula. MELD = 3.78 [Ln.Sr.Bil. (mg/dl)] + 11.2 [Ln.INR] +9.57 [Ln.Sr.Creat.] + 6.43

All examinations were performed using a convex probe (3.75–5 MHz) on MEDISON ACCUVIX A30. All the patients were examined using B-mode and Doppler US after a fasting period of ≥ 4 h. Three highly experienced radiologists, each with over a decade of expertise,

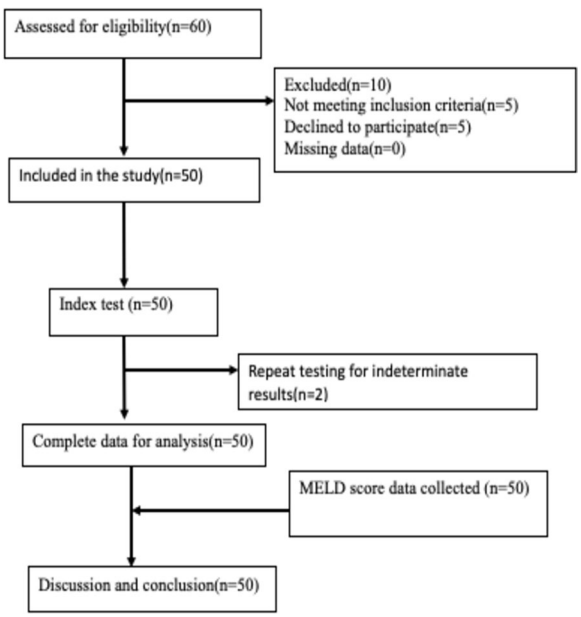

Fig. 1 STROCSS flow diagram for a patient with chronic liver disease

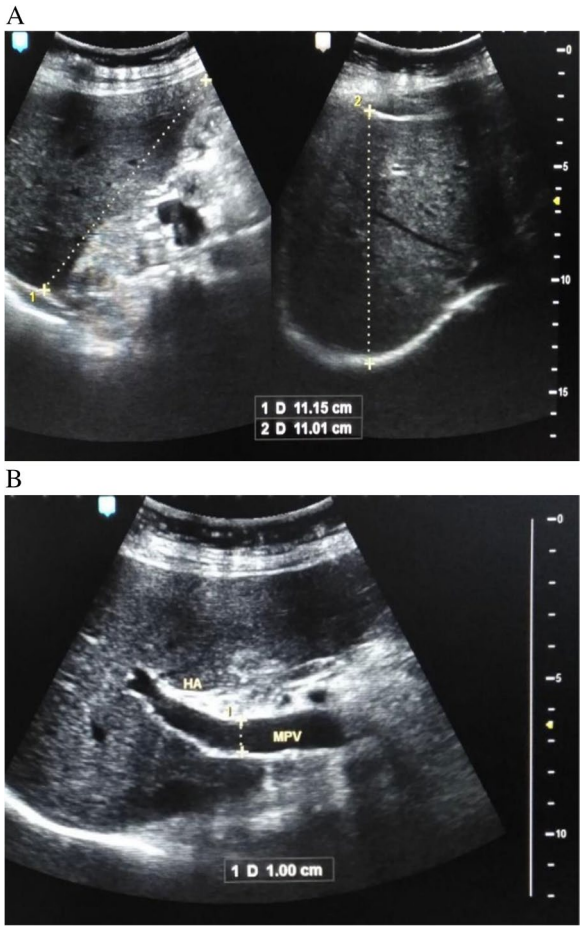

Fig. 2 **A** Measurement of the liver size of a 35-year-old female with chronic liver disease showed craniocaudal dimension of 11.15 cm. **B** Measurement of the portal vein at the porta hepatis of the same 35-year-old female with chronic liver disease (Diamete *r* = 10 mm). *MPV* main portal vein, *HA* hepatic artery

performed the ultrasound examinations with their specialized skills. On average, they performed 900 hepatic Doppler ultrasound procedures each year, showcasing their skill and deep familiarity with the technique. The patients were scanned in a supine and right anterior oblique position with the transducer in the oblique position in the right upper quadrant of the abdomen. The liver size was measured by taking the longitudinal length of the liver in the right midclavicular line extending from the hepatic dome to the lower hepatic margin according to the method described by Khammas et al.[11] (Fig. 2A). Measurement of the portal vein diameter was taken in quiet respiration at the hilum of the liver just before bifurcation into right and left. The diameter was taken by putting the two cursors in the internal wall of the portal vein; the wall of the portal vein was excluded from the measurement (Fig. 2B). In the color Doppler study, the presence and absence of flow in portal vein either due to sluggish flow or thrombus were also noted.

A standardized scanning protocol was adopted for all patients with scanning performed via a right intercostal space to visualize and interrogate the hepatic artery at porta hepatis. After evaluating the vascular configuration via B mode in the axial and longitudinal planes, Doppler US findings were evaluated following optimization of the flow rate interval, flow filter, and Doppler gain for minimizing noise and aliasing artifacts (Fig. 3A). Peak systolic velocity of the hepatic artery was measured in centimeters per second at the porta hepatis using a Doppler angle of less than or equal to 60 degrees for angle correction (Fig. 3B). The maximum velocity, minimum velocity, resistive index (RI), pulsatility index (PI), and acceleration time (AT) of the common hepatic artery (HA) were measured.

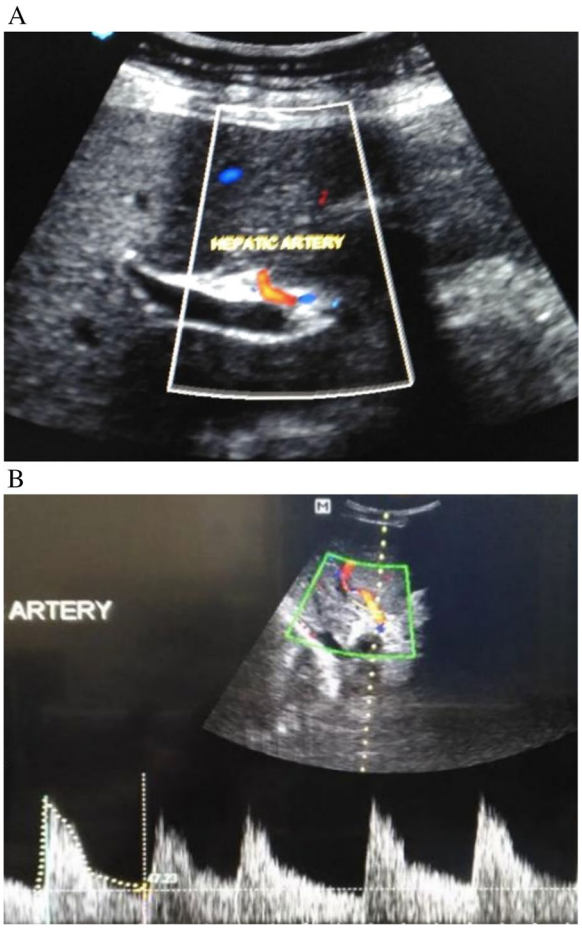

Fig. 3 **A** Hepatic artery at the porta hepatis in color Doppler study of the same 35-year-old female with chronic liver disease. **B** Spectral Doppler evaluation of the hepatic artery of the same 35-year-old female with chronic liver disease (PSV = 164.27 cm/s, EDV = 47.23 cm/s, PI = 1.36, RI = 0.71, AT = 56 ms)

The Hepatic artery Doppler parameters were calculated from the following equation:

$$\begin{aligned}\text{Resistance index} &= [(\text{peak systolic velocity} \\ &\quad - \text{end - diastolic velocity}) \\ &\quad /\text{peak systolicvelocity}] \\ \text{Pulsatility index} &= ((\text{peak systolic velocity} \\ &\quad - \text{end - diastolic velocity}) \\ &\quad /\text{mean averagevelocity}\end{aligned}$$

To mitigate selection bias, only participants meeting inclusion criteria are included. Measurement bias is minimized through standardized protocols, and quality control is ensured through regular meetings and continuous monitoring.

The study involved patients and the public in research objectives, design, and outcome measures. They facilitated recruitment, provided feedback, and participated in meetings, dissemination, and knowledge translation activities.

Statistical analysis

The biochemical parameters and calculated MELD score were entered in the datasheet. Analysis of data was performed upon completion of the study using IBM SPSS version 24 and Microsoft Excel software. Statistical analysis was done by linear regression analysis using Pearson's two-tailed test. Results were expressed in mean ± SD and comparisons with p value < 0.05 were considered statistically significant. There were no missing data in this study.

Results

The study included 50 patients diagnosed with chronic liver disease, aged 18–72 years, referred for routine follow-up scans. The etiologies of chronic liver disease included alcoholic liver disease, hepatitis B, hepatitis C, and an unknown cause of cirrhosis. The study found that alcoholic liver disease was the most common, followed by hepatitis B and C (Table 1).

Out of 50 patients, 22 had ascites, 7 had splenomegaly, and 1 had hepatomegaly. No additional ultrasonographic features were found in the remaining 18 patients. Liver size ranged from 10.65 to 19.02 cm, with a mean of 14.5232 ± 1.73 cm. Females had a mean size of 14.2752 ± 1.28, while males had a mean size of 14.7344 ± 2.03670 cm. No loss to follow-up case noted.

Portal vein size was measured, yielding a mean diameter of 11.83 ± 1.766 mm. Out of 50 patients, 10 exhibited dilated portal veins (> 13 mm) among them nine with alcoholic liver disease and one with hepatitis B. Color Doppler assessment identified 5 patients with hypoechoic filling defects and absent flow, indicating portal vein thrombosis.

Table 1 Clinico-demographic profile of chronic liver disease referred to radiology department

Characteristics	Number (%)
Sex	
Male	28 (56%)
Female	22 (44%)
Age	
> 60 year	15 (30%)
50–59 year	19 (38%)
40–49 year	11 (22%)
30–39 year	4 (8%)
< 30 year	1 (2%)
Etiology of chronic liver disease	
Alcoholic liver disease (ALD)	43 (86%)
Hepatitis B	4 (8%)
Hepatitis C	2 (4%)
Unknown	1 (2%)

Table 2 Hepatic artery Doppler parameters

	PSV	EDV	RI	PI	Sec
Mean	107.42	26.49	0.74	1.47	0.87
Median	99.50	23.29	0.75	1.51	0.08
Mode	82.00[a]	19.90[a]	0.71[a]	1.37[a]	0.07
Standard deviation	48.1	14.68	0.60	0.24	0.065
Skewness	2.50	3.23	−0.79	−0.19	6.46
Standard error of Skewness	0.34	0.34	0.34	0.34	0.34
Range	290.4	88.80	0.27	1.34	0.47
Minimum	26.60	7.2	0.58	0.86	0.05
Maximum	317.00	96.00	0.85	2.2	0.52

[a] Multiple modes exist. The smallest value is shown

The study examined hepatic artery spectral Doppler sonography, revealing a mean PSV of 107.416 ± 48.09986 and EDV of 26.4 ± 14.67847. Normal and high RI values were found, with a maximum of 42 patients having high RI values. Portal vein thrombosis was present in 4 patients, and none had low RI values. The PI value ranged from 0.86 to 2.2, and the acceleration time had a mean of 0.0865 ± 0.06457 (Table 2).

MELD score was obtained in all patients applying the values of biochemical tests to the standard formula. The mean MELD score was 19.28 ± 6.085. Out of 50 patients, the maximum number of patients were in category II followed by those in category III.

Pearson's correlation coefficient with a two-tailed test was applied to estimate the correlation between PSV,

Table 3 Correlation between hepatic artery Doppler parameters and MELD score

	PSV	EDV	RI	PI	Sec	Meld score
PSV						
Pearson correlation	1	0.757**	−0.380**	−0.421**	−0.169	0.224
Sig(2-tailed)		0.00	0.006	0.002	0.241	0.118
Number	50	50	50	50	50	50
EDV						
Pearson correlation	0.757**	1	−0.638**	−0.504**	−0.171	−0.006
Sig(2-tailed)	0.000		0.000	0.000	0.236	0.966
Number	50	50	50	50	50	50
RI						
Pearson correlation	−0.380**	−0.638**	1	0.451**	0.126	0.142
Sig(2-tailed)	0.006	0.000		0.001	0.383	0.327
Number	50	50	50	50	50	50
PI						
Pearson correlation	−0.421**	−0.504**	0.451**	1	−0.362**	−0.102
Sig(2-tailed)	0.002	0.000	0.001		0.010	0.481
Number	50	50	50	50	50	50
Sec						
Pearson correlation	−0.169	−0.171	0.126	−0.362**	1	0.043
Sig(2-tailed)	0.241	0.236	0.383	0.010		0.766
Number	50	50	50	50	50	50
MELD score						
Pearson correlation	0.224	−0.006	0.142	−0.102	0.043	1
Sig(2-tailed)	0.118	0.966	0.327	0.481	0.766	
Number	50	50	50	50	50	50

**Correlation is significant at the 0.01 level (2-tailed)

EDV, RI, PI, and AT (sec) with MELD score (Table 3). The study assessed correlations between MELD scores and Doppler parameters. PSV-MELD correlation had a small effect size ($r=0.224$) with $p=0.118$, indicated by a flat linear regression curve. Similarly, the RI-MELD correlation showed a small effect size ($r=0.145$) with $p=0.327$ and a flat regression curve. For PI-MELD, $r=-0.102$, $p=0.481$; for AT-MELD, $r=0.043$, $p=0.766$; both exhibited low effect sizes and flat curves. Overall, Doppler parameters exhibited low effect sizes ($r<0.1$) and p values >0.05, indicating no significant correlation with the MELD score.

Interobserver agreement for Doppler assessment of resistance index was evaluated using Cohen's kappa coefficient, revealing a moderate level of agreement ($\kappa=0.6$).

Portal vein thrombosis and hepatic artery PSV and RI

Portal vein thrombosis was identified in 5 patients. Among them, hepatic artery PSV was elevated (> 160 cm/s, cutoff) with a mean of 223.6 cm/s. Independent t-test yielded a p value <0.05, indicating a statistically significant correlation between these variables (Table 4).

Table 4 Independent *t* test for correlation of portal vein thrombosis and hepatic artery RI

		F	Sig	*T*	*df*	Significant (2-tailed)	95% CI of the difference	
							Lower	Upper
PSV	Equal variance assumed	21.46	0.000	9.68	48	0.000	102.29	155.89
RI	Equal variance assumed	4.209	0.046	−3.379	48	0.001	−0.1389	−0.0352

RI was higher in patients without portal vein thrombosis (mean ± SD = 0.75 ± 0.048) than those with (mean ± SD = 0.664 ± 0.098). Independent t-test yielded $p = 0.046$, indicating a significant correlation. Conversely, correlating portal vein thrombosis presence/absence with PI showed no significant correlation ($p = 0.93$). Thus, portal vein thrombosis correlated with elevated PSV and hepatic artery RI ($p < 0.05$), supporting the hepatic arterial buffer response theory.

Discussion

In our study, we found a limited correlation between Doppler parameters of hepatic arterial hemodynamics and Model for End-Stage Liver Disease (MELD) scores in patients with chronic liver disease; nevertheless, it underscores hepatic artery resistance index as a potential marker for portal vein thrombosis.

Assessing hepatic fibrosis is crucial for managing chronic liver disease, but invasive liver biopsy is problematic due to its invasiveness, coagulation issues, and false positives. Transient elastography (TE) and real-time two-dimensional shear wave elastography (2D-SWE) offer noninvasive liver stiffness measurement [12]. However, Doppler ultrasound is more accessible and simpler. This study evaluates hepatic artery Doppler's utility and its correlation with clinical severity, graded by MELD score, in chronic liver disease.

The MELD scoring system was developed to improve the accuracy of Child–Pugh scores in end-stage liver disease patients. It has been used for liver transplantation and is more accurate in estimating 3-month survival [13]. This study aimed to determine the relationship between hepatic arterial Doppler parameters and MELD score.

Our study found a limited correlation between hepatic artery hemodynamic flow parameters (PSV, EDV, RI, PI, AT) and increasing MELD scores. Although we expected these parameters, except RI, to rise with higher MELD scores due to portal hypertension, RI's correlation with liver disease is intricate. Liver disease may manifest as abnormally elevated or decreased resistance in the hepatic artery, with factors like inflammatory edema, arterial compression by regenerative nodules, and stiff noncompliant parenchyma thought to increase resistance. Other factors, such as the "hepatic arterial buffer response" and arteriovenous shunting, are thought to decrease resistance [14].

A study by Park et al.'s [7] found a significant correlation between Hepatic artery velocity (HAV) and MELD scores in 217 patients, with no significant correlations found between hepatic artery resistance index (HARI) and MELD scores, splenomegaly, or ascites. High MELD scores were predicted by HAV > 160 cm/s, with a 120 cm/s threshold offering better sensitivity and specificity for hepatic dysfunction. The study conducted by Topal et al. [6] demonstrated a significant correlation between Hepatic artery resistance index (HARI) and MELD scores, contrary to the findings of studies by Baz et al. [15] and our own study, potentially influenced by the diverse etiology and stages of chronic liver disease.

Glisic et al. [16] found higher HAV and HARI in patients with liver cirrhosis compared to healthy controls. However, their study involved only 80 patients and 20 healthy controls, and their primary focus was on correlating renal artery resistive index with MELD scores rather than HAV. Patients with liver cirrhosis had a higher average HAV of 125.7 ± 67.7 cm/s compared to healthy controls of 79.3 cm/s ± 20.3 cm/s.

In our study with 50 patients, the mean MELD score was 19.28 ± 6.085 (range 8–36). Hepatic artery peak systolic velocity ranged from 26–317 cm/s, mean of 107.416 ± 48.09986 cm/s. Unlike Park et al. [7] PSV didn't significantly correlate with the MELD score ($p = 0.118$). Five patients with PSV > 160 cm/s and RI < 0.7 (except 1 with RI 0.84) had portal vein thrombosis. Statistically significant correlation emerged between thrombus presence, hepatic arterial PSV, and RI ($p < 0.05$), reinforcing reduced portal venous inflow triggering hepatic arterial buffer response.

Our study revealed variable MELD scores in patients with PSV > 160 cm/s, placing them in categories II or III. Most patients exhibited elevated RI (> 0.7). In contrast to Glisic et al. [16] our study lacked a control group and didn't address renal artery RI. Popov et al. [17] found that hepatic artery mean velocity, maximum peak systolic velocity, and arterial/portal ratio were higher in MELD scores > 20 versus < 20 patients. However, no direct correlation existed between liver Doppler US parameters and MELD scores. Moreover, no significant differences emerged between liver Doppler US parameters and liver cirrhosis complications.

The study found no significant correlation between PSV and MELD score in patients with a MELD score greater than 20. PSV ranged from 54 to 317 cm/s, while PI ranged from 0.61 to 0.84. The study did not evaluate portal veins or arterial/portal ratios. Afif et al. [18] studied for three years, finding that MHAV was not significantly different between the study and control groups ($p = 0.06$), and there was no significant difference within CP subsets. HARI was higher in the study group vs. control ($p < 0.001$) and also within CP subsets ($p < 0.001$). Limited studies apply the MELD score for hepatic Doppler parameters comparison. Our MELD-based study similarly found no significant hepatic artery velocity correlation, aligning with Afif et al.

Histologic analysis is the gold standard for diagnosing cirrhosis, but liver biopsy is not routine due to alternatives like fibroscan and indirect markers [19]. Liver damage severity was not graded, and the study had diverse chronic liver disease stages and causes. Cardiac output's impact on peripheral artery resistance was not evaluated, and hepatic artery anatomical variations were not accounted for. Normal patients were excluded as controls, as the MELD score's validation only applies to liver disease patients. Small sample size is another limitation. The absence of a control group without liver disease and our reliance on MELD scores might limit broader applicability. While our study provides valuable insights, applying findings across different clinical contexts requires careful consideration.

Conclusions

Hepatic artery Doppler sonography is a noninvasive method for diagnosing and grading chronic liver disease. Although it has limited predictive value for MELD scores, it has high variability and a high resistive index (RI) prevalence. The study highlights the importance of the hepatic artery in assessing disease severity and recommends further research on its diagnostic and prognostic value.

Abbreviations

MELD	Model for end-stage liver disease
RI	Resistance index
PI	Pulsatility index
AT	Acceleration time
PSV	Peak systolic velocity
CLD	Chronic liver disease
IRC	Institutional Review Committee
STROCCS	Strengthening the Reporting of Cohort Studies in Surgery
ALD	Alcoholic liver disease
HAV	Hepatic artery velocity
HARI	Hepatic artery resistance index
SD	Standard deviation
CTP	Child-Turcotte-Pugh

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Author contributions

ST and SK were involved in the conceptualization and review of this article. ST conducted data interpretation, while GG reviewed the article and conducted data interpretation as well. UK contributed to performing literature review, editing, and writing the paper. All authors have thoroughly read and approved the final manuscript.

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author upon reasonable request.

Declarations

Ethics approval and consent to participate

We have conducted an ethical approval based on the Declaration of Helsinki with registration research at the Institutional Review Committee (IRC) of the Institute of Medicine (IOM), Tribhuvan University, Nepal. Reference number: 44(6-11)E^2 /075/076. All data were extracted after taking consent from patients involved in the study.

Consent for publication

Written informed consent was obtained from the patient for the publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Competing interests

The authors declare that they have no competing interests.

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