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Impact of Hand Grip Strength on the Clinical Course of Patients with Acute Variceal Bleeding: A Single-Center Prospective Observational Study

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ABSTRACT

Objective: This study aimed to assess handgrip strength (HGS) in patients with acute variceal bleeding (AVB) following endoscopic therapy, compare it with compensated cirrhotic patients, and investigate its association with clinical outcomes, including rebleeding and short-term mortality.

Materials and Methods: In this single-center, prospective observational study, 34 AVB patients and 21 compensated cirrhotic were enrolled between March 2025-August 2025. Exclusion criteria included hepatocellular carcinoma, hepatic encephalopathy, malignancies, major organ failure, cerebrovascular disease, or neuromuscular disorders. HGS was measured using a digital dynamometer at discharge for AVB patients and during outpatient visits for controls. Baseline demographics, laboratory data, Child–Turcotte–Pugh (CTP) and model for end-stage liver disease (MELD-Na) scores, and 6-week outcomes were recorded. Statistical analyses compared HGS and clinical parameters between groups and evaluated correlations.

Results: HGS was significantly lower in the AVB group compared with compensated cirrhotic (p=0.0004). Within 6 weeks, nine patients experienced rebleeding, and seven patients died. Those with adverse outcomes demonstrated significantly reduced HGS, lower albumin, hemoglobin, and blood pressure, and higher MELD-Na and CTP scores. HGS negatively correlated with prognostic scores, hospital stay, international normalized ratio, and bilirubin, while positively correlating with albumin.

Conclusion: HGS is markedly reduced in patients with AVB and is associated with rebleeding, short-term mortality, and prolonged hospitalization. As a simple bedside measure of sarcopenia and nutritional status, HGS may serve as a valuable prognostic marker. Early identification of low HGS can guide nutritional and rehabilitative interventions to potentially improve outcomes in this high-risk patient population.

Keywords: Acute variceal bleeding, Hand dynamometer, Mortality, Muscle strength, Rebleeding, Sarcopenia

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INTRODUCTION

Acute variceal bleeding (AVB) represents a medical emergency, carrying a 6-week mortality rate of 10–20%.[1] Even patients who survive an initial acute bleeding episode remain at high risk, with rebleeding and mortality rates reaching approximately 60% and 33%, respectively, within the 1st year in the absence of secondary prophylaxis.[2] The combined use of non-selective β-blockers and endoscopic therapy has been shown to reduce the risk of rebleeding and improve survival outcomes.[3] Nevertheless, studies indicate that the risk of recurrent bleeding after endoscopic treatment remains between 7.8% and 29%, and fatal rebleeding events may still occur.[4] Thus, identifying risk factors that can predict poor outcomes in patients undergoing endoscopic therapy is of critical clinical importance.[5]

Malnutrition and sarcopenia are major complications of decompensated cirrhosis, primarily arising from metabolic dysfunction and nutritional imbalance.[6] Therefore, timely identification of prognostic indicators in cirrhotic patients is essential to improve outcomes in this vulnerable population. Muscle function has been shown to be strongly linked to both the progression and prognosis of cirrhosis.[7] In particular, handgrip strength (HGS), measured with a dynamometer, has been proposed as a sensitive marker of muscle function, with advantages including simplicity, feasibility, and good reproducibility. Patients with cirrhosis demonstrate markedly lower HGS compared to non-cirrhotic individuals[8] and reduced HGS has been validated as being associated with disease severity, malnutrition, sarcopenia and progression.[9,10]

The presence of substantial evidence demonstrating that sarcopenia and reduced HGS are associated with poor prognosis in liver cirrhosis has raised the necessity of investigating HGS in patients with AVB and its potential relationship with clinical outcomes. To date, only a limited number of studies have assessed HGS in patients undergoing endoscopic therapy for AVB. In our study, we aimed to evaluate HGS in patients with AVB compared with compensated cirrhotic patients, and to investigate the association of HGS, a bedside tool, with post-endoscopic clinical course, including rebleeding and related outcomes.

MATERIALS AND METHODS

Study Population

For our study, patients who presented to Samsun Training and Research Hospital between March 2025-August 2025 with a diagnosis of variceal bleeding or compensated liver cirrhosis were evaluated. The patient group consisted of cases with AVB, while the control group included compensated cirrhotic patients attending the gastroenterology outpatient clinic. Patients without AVB on endoscopy, as well as those with concomitant hepatocellular carcinoma, hepatic encephalopathy (HE), other malignancies, cardiac or renal failure, history of cerebrovascular disease, or neuromuscular disorders, were excluded from the study. Following these criteria, a total of 34 patients with AVB were enrolled as the patient group, and 21 compensated cirrhotic patients without any prior variceal bleeding were included as the control group. All patients underwent standard initial management in the emergency department, which included intravenous (IV) fluid replacement, blood transfusion to maintain hemoglobin (Hb) levels above 8 g/dL, prophylactic antibiotics (ceftriaxone 1 g IV), and administration of terlipressin. Emergency endoscopy was carried out within 12 h in every case. Patients presenting with hematemesis, melena, or hematochezia, together with a Hb drop and endoscopic evidence of actively bleeding varices or the presence of a nipple sign, were categorized as having AVB. The diagnosis of cirrhosis had been confirmed in all participants by abdominal ultrasonography performed within the preceding 6 months. Patients were hospitalized for a minimum of 5 days and monitored for at least 6 weeks, during which episodes of rebleeding and mortality were documented.

Clinical, Laboratory and HGS Assessment

Demographic characteristics, endoscopic findings, cirrhosis etiology, baseline biochemical parameters, endoscopic interventions, and length of hospital stay were collected. Child-Turcotte-Pugh (CTP) and model for end-stage liver disease (MELD-Na) scores were calculated using admission data.

The CTP score was determined based on bilirubin, albumin, international normalized ratio (INR), and the presence and severity of ascites and encephalopathy, as previously described.[11] The MELD-Na score was calculated using the formula: MELD + 1,32 × (137−Na)−(0.033×MELD×[137−Na]).[11,12]

HGS was measured using a digital hand dynamometer (Camry Digital Hand Dynamometer, Model: EH101, Fig. 1), which has been validated for clinical use.[13] Participants were instructed to sit upright in a chair with a backrest but without armrests, maintaining both feet flat on the floor and knees flexed at 90°. The test arm was positioned with 90° elbow flexion and the forearm in a neutral pronation-supination position.[14] Before the test, the procedure was explained, and a blinded examiner provided standardized verbal encouragement for participants to exert their maximum grip strength with the dominant hand. Three trials were performed with a 1-min rest interval, and the highest value, expressed in kilograms, was recorded. In the patient group, measurements were performed immediately before hospital discharge, following endoscopic control of variceal bleeding and completion of inpatient treatment. In the control group, measurements were obtained during outpatient clinic visits.

Figure 1. Camry EH101 dynamometer.

First, the baseline biochemical and demographic characteristics, as well as HGS, were compared between the AVB group and the compensated cirrhosis control group. Rebleeding was defined as new onset hematemesis, melena, or hematochezia, with endoscopic evidence of recurrent bleeding. Post-discharge, 6 weeks rebleeding or mortality were recorded. Then, patients were grouped according to the presence or absence of in 6 weeks rebleeding, or mortality, and their CTP, MELD, and hand grip strength scores were compared.

Ethical Approval

This study was approved by the Ethics Committee of Samsun University Non-Interventional Clinical Research (date: 05.03.2025, decision no: 2025/5/23). All participants were fully informed about the study procedures, and written informed consent was obtained in accordance with the Declaration of Helsinki.

Statistical Analysis

Descriptive statistics for numerical variables were presented as mean ± standard deviation and median, while categorical variables were summarized as counts and percentages. Group comparisons for normally distributed continuous variables were carried out using the independent samples t-test, whereas the Mann–Whitney U test was employed for non-normally distributed variables. Categorical variables were compared between groups using Pearson's Chi-square test. Correlation analyses were performed using Spearman's rank correlation test.

RESULTS

The mean age and sex distribution of the 34 patients with AVB and the 21 patients with compensated liver cirrhosis in the control group were comparable (Table 1). Regarding cirrhosis etiology, hepatitis B infection was predominant in the AVB group, whereas steatotic liver disease was more frequent in the compensated cirrhosis group (Table 1). As shown in Table 1, the most common site of varices in the AVB group was the cardia and esophagus gastro-esophagial varices-1, while esophageal varices were most frequent among compensated cirrhosis patients. Other demographic variables and the types of interventions performed for variceal bleeding are presented in Table 1.

Table 1. Characteristics and demographic data of all studied patients

Variable	Number/mean (%)	
	Acute variceal bleeding group n=34	Compensated cirrhosis group n=21
Age	57.2	60.2
Male: Female	26 (76):8 (24)	16 (77):5 (23)
Etiology		
MASLD	12 (35)	8 (38)
HBV	13 (38)	7 (33)
Alcohol	2(5)	4 (19)
PBC-AIH	2 (5)	1 (4)
Other	5 (14)	1 (4)
Location of varices		
Esophagus	14 (41)	19 (90)
GOV-1	16 (47)	2 (21)
GOV-2	3 (8)	-
IGV-1	1 (2)	-
Grade of varices		
F3	31(91)	1 (4)
F2	3 (8)	16 (74)
F1	-	4 (19)
Ascites +	17 (50)	-
Endoscopic procedure		
Band ligation	30 (88)	-
Histoacryl injection alone	3 (8)	-
Combined treatment	1 (2)	-

MASLD: Metabolic associated steatotic liver disease, HBV: Hepatitis B virüs, PBC-AIH: Primary biliary cholangitis-autoimmune hepatitis, GOV: Gastroesophageal varices, IGV: Intragastric varices.

When comparing baseline biochemical parameters between the AVB and compensated cirrhosis groups, platelet count, albumin, Hb, systolic blood pressure, and HGS were significantly lower in the AVB group, whereas bilirubin, ALT, CTP, and MELD-Na scores were significantly higher (Table 2).

Within 6 weeks after discharge, a total of nine patients experienced rebleeding. In this subgroup, MELD-Na scores were significantly higher, and HGS was significantly lower (Table 3). Furthermore, platelet count, Hb, and initial systolic blood pressure were significantly reduced in patients with rebleeding (Table 3).

During the 6-week follow-up, seven patients with AVB died. In those who died, baseline bilirubin, ALT, creatinine, and MELD-Na scores were significantly higher, while systolic blood pressure, albumin, diastolic blood pressure, and HGS were significantly lower (Table 4).

Table 2. A comparison of basic parametric and handgrips of the groups

Variables	Acute variceal bleeding group n=34 mean±SD	Compensated cirrhosis group n=21 mean±SD	*p*
Age, year	57.2±13	60.2±9	0.25
Male: Female*	26:8	16:5	0.98
WBC	7400±4400	6470±2280	0.28
Hemoglobin g/dL	7.6±1.8	10.6±1.4	**0.000**
Platelet	90.000±57.000	115.000±185.000	**0.02**
INR	1.35±0.21	1.31±0.2	0.48
Total bilirubin mg/dL	2.76±5.1	0.85±0.4	**0.01**
ALT IU	26±20	19.2±11.4	**0.02**
Albumin g/dL	2.7±0.4	3.5±0.5	**0.000**
Creatinine mg/dL	0.97±0.4	0.75±0.2	**0.02**
Sodium mg/dL	133±4.3	137±2.9	**0.000**
Systolic blood pressure mm/Hg	106±23	120±12	**0.01**
Diastolic blood pressure mm/Hg	60.7±10	63.8±8	0.24
Pulse, min	91±11	85±6	**0.01**
CTP	7.5±1.4	6.1±1.2	**0.000**
MELD-Na	15.7±7.6	11.5±3.1	**0.003**
Handgrip (kg)	20.9±12	29±7	**0.004**

WBC: White blood count, INR: International normalized ratio, ALT: Alanine aminotransferase, CTP: Child-Pugh score, MELD: Model for end-stage liver disease, SD: Standard deviation

Table 3. The comparison of the groups in terms of rebleeding in 6 weeks

Acute variceal bleeding group	Rebleed in 6 weeks n=9 mean±SD	Not rebleed in 6 weeks n=25 mean±SD	*p*
Age, year	57.4±16.2	57.2±12.1	0.9
Hemoglobin g/dL	6.1±0.9	8.1±1.7	**0.000**
Platelet×10^3	58.6±18.6	101±62.2	**0.004**
INR	1.4±0.25	1.3±0.2	0.4
Total bilirubin mg/dL	2.6±2.4	2.7±5.8	0.9
ALT IU	32.6±20	23.8±14.6	**0.007**
Albumin g/dL	2.4±0.2	2.9±0.4	**0.001**
Creatinine mg/dL	0.9±0.3	0.9±0.4	0.8
Systolic blood pressure mm/hg	93±16	110±23	**0.02**
Diastolic blood pressure mm/hg	55±9	62.8±10.9	0.06
CTP	8.1±1.2	7.3±1.4	0.1
MELD-Na	18.3±10.8	14.8±6.1	**0.003**
Handgrip (kg)	13.4±4.5	23.6±12.9	**0.02**

WBC: White blood count, INR: International normalized ratio, ALT: Alanine aminotransferase, CTP: Child-Pugh score, MELD: Model for end-stage liver disease, SD: Standard deviation

In addition, significant correlations were observed between HGS and several clinical parameters in the AVB group. Specifically, decreasing HGS was associated with longer hospital stay, as well as higher MELD-Na and CTP scores. Negative correlations were identified between HGS and INR, and total bilirubin, while a positive correlation was observed between HGS and albumin (Table 5).

DISCUSSION

In our study, we investigated whether HGS was lower in patients with AVB compared with those with compensated cirrhosis, and whether HGS was associated with the clinical course of these patients. HGS was found to be significantly lower in the AVB group compared to the compensated cirrhosis group. Moreover, among patients with AVB who were discharged, those who experienced rebleeding or death within 6 weeks had lower HGS values. In addition, HGS demonstrated negative correlations with prognostic scores such as CTP and MELD-Na, as well as with the length of hospital stay. Taken together, these findings suggest that HGS, a simple bedside tool for the assessment of sarcopenia and malnutrition in cirrhotic patients, is lower in those with AVB and may be associated with poorer clinical outcomes and longer hospital stay.

AVB is one of the most important causes of decompensation in chronic liver disease due to its substantial risk of morbidity and mortality. The early identification of patients at high risk for rebleeding and mortality during follow-up, as well as the use of tools that can help predict clinical outcomes, may provide significant advantages in clinical practice. Therefore, the early recognition of malnutrition and sarcopenia – both major contributors to decompensation and mortality in cirrhosis that also worsen clinical outcomes – is crucial in detecting and managing this vulnerable patient population.[7]

Sarcopenia is characterized by a reduction in skeletal muscle mass along with a decline in muscle function, which may be reflected as reduced strength or diminished physical performance.[15] In patients with liver cirrhosis, the prevalence of sarcopenia has been reported to range between 30% and 70%, varying according to the assessment method applied and the stage of liver disease.[16] Both sarcopenia and malnutrition become more common as liver disease progresses, with a higher frequency observed among male patients.[17] The underlying mechanisms of sarcopenia in cirrhosis are multifactorial, including inadequate dietary intake, malabsorption, metabolic disturbances, hormonal alterations, hyperammonemia, and increased muscle degradation.[17] Sarcopenia contributes to a higher incidence of complications such as infections, HE, and ascites.[18] Furthermore, it is recognized as an independent predictor of mortality in cirrhotic individuals.[19] Malnutrition and sarcopenia together are linked to elevated risks of decompensation, infection, and increased mortality among patients awaiting liver transplantation.[19] The HGS test has been considered a useful and sufficient tool for assessing sarcopenia in patients with hepatitis C infection, steatotic liver disease, and cirrhosis.[9,20,21] In our study, HGS was found to be markedly lower in the group with decompensated cirrhosis due to variceal bleeding compared with patients with compensated cirrhosis. Consistent with the literature, sarcopenia and malnutrition are known to accelerate the progression toward decompensation.[18,21]

Table 4. The comparison of the groups in terms of mortality in 6 weeks

Acute variceal bleeding group	Mortality in 6 weeks *n*=7 mean±SD	Survive in 6 weeks *n*=27 mean±SD	*p*
Age	**59.4±7.6**	**56±14.2**	0.5
Hemoglobin g/dL	6.9±1.2	7.8±1.9	0.1
Platelet×10^3	88±40	90±61	0.9
INR	1.4±0.2	1.3±0.2	0.2
Total bilirubin mg/dL	7.5±9.8	1.5±1.6	0.000
ALT, IU	51±30	19±10	0.001
Albumin g/dL	2.3±0.3	2.8±0.4	0.009
Creatinine mg/dL	1.1±0.6	0.9±0.3	0.02
Systolic blood pressure mm/hg	82.7±5.6	112±22	0.03
Diastolic blood pressure mm/hg	50±5	63.5±10.3	0.01
CTP	8.2±1.7	7.3±1.3	0.6
MELD-Na	19.2±10.6	14.8±6.5	0.01
Handgrip (kg)	10.4±5.2	23.7±12	0.000

WBC: White blood count, INR: International normalized ratio, ALT: Alanine aminotransferase, CTP: Child-Pugh score, MELD: Model for end-stage liver disease, SD: Standard deviation.

In a study evaluating sarcopenia, mortality, and the risk of rebleeding in patients with AVB, it was demonstrated that those with sarcopenia at the time of bleeding had a significantly higher risk of rebleeding within 2 years.[5] However, no association between sarcopenia and mortality was observed in the same study. Consistent with these findings, our study also revealed markedly lower HGS in the group that experienced rebleeding within 6 weeks.

The mechanism by which sarcopenia adversely influences variceal bleeding remains not fully elucidated. Clinically, the severity of portal hypertension represents the most significant predictor of AVB. From a pathophysiological perspective, the association between sarcopenia and portal hypertension may be reciprocal.[22] Factors linked to portal hypertension, such as

Table 5. The correlation analysis of the hand grip strength for some parameters

Spearman's Rho for hand grip strength	Age	Platelet	Albumin	Total bilirubin	CTP	BMI	MELD-Na	INR	Hb	ALT	Hospital stay
Correlation coefficient	–0.24	–0.29	0.78	–0.49	–0.61	0.11	–0.56	–0.32	–0.29	–0.11	–0.49
Sig	0.17	0.08	**0.000**	**0.003**	**0.000**	0.52	**0.001**	**0.05**	0.09	0.52	**0.003**

CTP: Child-Pugh score, MELD: Model for end-stage liver disease, BMI: Body-mass index, Hb: Hemoglobin, ALT: Alanine aminotransferase, INR: International normalized ratio

spontaneous portosystemic shunt formation, endotoxemia, and hyperammonemia, play a key role in promoting sarcopenia among cirrhotic patients.[23] Nevertheless, the specific contribution of sarcopenia to portal hypertension-related complications, including variceal bleeding, is still under debate. Skeletal muscle is recognized as an endocrine organ capable of secreting cytokines and polypeptides with autocrine, paracrine, and endocrine actions, many of which are critically involved in inflammatory pathways.[24] Consequently, sarcopenia may foster chronic low-grade inflammation, which tends to worsen with increasing portal hypertension and circulatory impairment, thereby heightening the risk of complications such as variceal bleeding.

The principal limitations of this study include the relatively small sample size and its single-center nature, which may restrict the generalizability of the findings. Nonetheless, a notable strength is that it represents one of the few studies to evaluate HGS specifically in patients presenting with AVB.

CONCLUSION

Since HGS is an indicator of both nutritional status and sarcopenia, this study highlights the importance of early screening and timely nutritional support in patients with AVB. Such interventions, including appropriate dietary strategies and exercise programs, may enhance muscle mass and ultimately improve clinical outcomes.

DECLARATIONS

Ethics Committee Approval: This study was approved by the Ethics Committee of Samsun University Non-Interventional Clinical Research (Date: 05.03.2025, Decision no: 2025/5/23).

Informed Consent: Written informed consent was obtained.

Conflict of Interest: None declared.

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