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Analysis of Factors Influencing Disease Outcomes in Patients with Liver Cirrhosis and Upper Gastrointestinal Hemorrhage After Endoscopic Hemostasis

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Abstract: *Objective:* To investigate the factors influencing disease outcomes in patients with liver cirrhosis and upper gastrointestinal hemorrhage (UGH) after endoscopic hemostasis. *Methods:* A retrospective analysis was conducted on clinical data of patients with liver cirrhosis and UGH admitted to our hospital from January 2023 to December 2024. All patients received endoscopic hemostasis. According to their disease outcomes, 215 patients were divided into a good prognosis group (172 cases) and a rebleeding group (43 cases, with rebleeding occurring one year after treatment). The influencing factors for disease outcomes were analyzed. *Results:* The rate of good prognosis in patients with liver cirrhosis and UGH was 80.00%, and the one-year rebleeding rate after treatment was 20.00%. Univariate analysis showed that age, liver cirrhosis stage, liver function grade, hepatic encephalopathy, esophageal varices, complications, and shock index were influencing factors for disease outcomes in these patients ($P < 0.05$). Binary logistic regression analysis showed that age, decompensated liver cirrhosis, hepatic encephalopathy, esophageal varices, and complications were independent risk factors for rebleeding one year after treatment ($P < 0.05$). *Conclusion:* One year after endoscopic hemostasis, patients with liver cirrhosis and UGH have a relatively high rebleeding rate. Factors such as bleeding volume influence disease outcomes. Therefore, increased attention and targeted interventions are needed to achieve better prognostic outcomes.

Keywords: Liver cirrhosis; Upper gastrointestinal hemorrhage; Endoscopic hemostasis; Disease outcome; Influencing factors

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1. Introduction

Liver cirrhosis is caused by long-term liver injury, with common etiologies including hepatitis virus infection and alcoholism. It is a common chronic liver disease characterized by progressive and diffuse development [1]. In recent years, the incidence of liver cirrhosis has been continuously increasing. Early symptoms are atypical, but as the disease progresses, it leads to persistent liver function impairment and portal hypertension. In the advanced stages, symptoms such as upper gastrointestinal hemorrhage (UGH) often occur [2]. UGH is a major cause of hospitalization and death in

patients with liver cirrhosis. UGH can exacerbate liver injury, potentially lead to complications such as ascites and hepatic encephalopathy, and may also cause hemorrhagic shock, resulting in a poor prognosis. Clinically, endoscopic hemostasis is commonly used to treat UGH, as it has a favorable hemostatic effect [3]. However, liver cirrhosis complicated by UGH is a critical condition that progresses rapidly and is prone to shock. This study observes the disease outcomes of patients with liver cirrhosis and UGH after endoscopic hemostasis and analyzes related influencing factors, aiming to develop targeted interventions to improve prognosis. The details are as follows.

2. Materials and methods

2.1. General information

A total of 215 patients diagnosed with liver cirrhosis and upper gastrointestinal bleeding in our hospital from January 2023 to December 2024 were enrolled. Among them, there were 146 males and 69 females. The age ranged from 38 to 80 years, with a mean of 60.55 ± 5.74 years. The body mass index (BMI) ranged from 19.5 to 28.9 kg/m², with a mean of 24.12 ± 1.87 kg/m².

2.2. Diagnostic criteria

All cases simultaneously met the diagnostic criteria specified in the *Guidelines for the Diagnosis and Treatment of Liver Cirrhosis* [4] and the *Expert Consensus on the Emergency Diagnosis and Treatment Process for Acute Upper Gastrointestinal Bleeding* [5].

2.3. Inclusion criteria

(1) Meeting the clinical diagnostic criteria and confirmed as liver cirrhosis complicated by UGH based on clinical symptoms, laboratory examinations, and endoscopy; (2) All patients received endoscopic hemostasis; (3) Time from disease onset to hospital admission less than 12 hours; (4) Normal function of organs including the heart, lungs, and kidneys; (5) Complete clinical data.

2.4. Exclusion criteria

(1) Presence of bleeding from other sites; (2) Upper gastrointestinal bleeding not caused by liver cirrhosis; (3) Presence of cancer; (4) Presence of systemic infectious diseases; (5) History of cardiovascular or cerebrovascular diseases, or history of gastrointestinal surgery in the recent period.

2.5. Methods

Clinical data of the patients were collected, including gender, age, bleeding volume, type of liver cirrhosis, liver function grade, underlying diseases, comorbidities, complications (ascites, Dieulafoy's ulcer bleeding, etc.), and shock index.

2.6. Observation indicators

Patients were followed up for one year after the completion of endoscopic hemostasis treatment. Disease outcomes were observed, and related risk factors were analyzed.

2.7. Statistical analysis

Clinical data were entered into SPSS version 25.0 software. Categorical data were analyzed using the Chi-square (χ^2) test and expressed as counts (percentages). Binary logistic regression was used for multivariate analysis. The significance level was set at 0.05.

3. Results

3.1. Disease outcomes

Among the 215 patients with liver cirrhosis complicated by UGH, 172 cases (80.00%) had a good prognosis, while 43 cases (20.00%) experienced rebleeding one year after treatment.

3.2. Univariate analysis of disease outcomes

Comparisons between the good prognosis group and the rebleeding group showed significant differences in age, liver cirrhosis stage, liver function grade, hepatic encephalopathy, esophageal varices, complications, and shock index ($P < 0.05$). See **Table 1**.

Table 1. Univariate analysis of disease outcomes [*n* (%)]

Variable	Good prognosis group(*n* = 172)	Rebleeding group(*n* = 43)	χ^2 value	*P* value	Variable
Age (years)	< 60	116 (67.44)	15(34.88)	15.318	< 0.001
	≥ 60	56 (32.56)	28(65.12)		
Gender	Male	118(68.60)	28(65.12)	0.192	0.661
	Female	54 (31.40)	15(34.88)		
Bleeding volume (mL)	< 500	103(59.88)	25(58.14)	0.043	0.835
	≥ 500	69 (40.12)	18(41.86)		
Type of liver cirrhosis	Alcoholic	41 (23.84)	9 (20.93)	0.163	0.687
	Non-alcoholic	131(76.16)	34(79.07)		
Liver cirrhosis stage	Compensated	120(69.77)	15(34.88)	17.917	< 0.001
	Decompensated	52 (30.23)	28(65.12)		
Child-Pugh grade	A–B	125(72.67)	17(39.53)	16.847	< 0.001
	C	47 (27.33)	26(60.47)		
Anemia	Yes	43 (25.00)	13(30.23)	0.489	0.484
	No	129(75.00)	30(69.77)		
Hypertension	Yes	114(66.28)	27(62.79)	0.186	0.667
	No	58 (33.72)	16(37.21)		
Diabetes mellitus	Yes	108(62.79)	24(55.81)	0.707	0.401
	No	64 (37.21)	19(44.19)		
Coronary heart disease	Yes	95 (55.23)	26(60.47)	0.383	0.536
	No	77 (44.77)	17(39.53)		
Hepatic encephalopathy	Yes	21 (12.21)	20(46.51)	26.227	< 0.001
	No	151(87.79)	23(53.49)		
Esophageal varices	Yes	60 (34.88)	28(65.12)	13.005	< 0.001
	No	112(65.12)	15(34.88)		
Complications	Yes	28 (16.28)	21(48.84)	20.723	< 0.001
	No	144(83.72)	22(51.16)		
Shock index	> 1.5	19 (11.05)	13(30.23)	9.996	0.002
	≤ 1.5	153(88.95)	30(69.77)		

3.3. Multivariate analysis of disease outcomes

Taking the patient's prognosis status as the dependent variable, the indicators with statistical significance in the univariate analysis were incorporated into the binary logistic regression equation. The results showed that the independent risk factors for rebleeding one year after treatment in patients with liver cirrhosis complicated by UGH were age, decompensated liver cirrhosis, hepatic encephalopathy, esophageal varices, and complications ($P < 0.05$). See **Table 2**.

Table 2. Multivariate analysis of disease outcomes

Variable	β value	SE value	Wald value	*P* value	OR value	95% CI for OR
Age	1.097	0.362	9.183	0.003	2.995	1.473–6.089
Decompensated liver cirrhosis	0.989	0.420	5.545	0.019	2.689	1.180–6.124
Child-Pugh grade C	1.106	0.649	2.904	0.089	3.022	0.847–10.784
Hepatic encephalopathy	1.208	0.520	5.397	0.021	3.347	1.208–9.274
Esophageal varices	1.163	0.384	9.173	0.003	3.200	1.507–6.791
Complications	1.302	0.315	10.696	0.001	2.802	1.511–5.194
Shock index > 1.5	0.965	0.537	3.229	0.073	2.625	0.916–7.520

4. Discussion

Patients with liver cirrhosis are prone to UGH due to factors such as rupture of esophageal and gastric varices caused by portal hypertension, decreased liver function, and reduced synthesis of coagulation factors. UGH not only aggravates the condition and further impairs organ function but also easily leads to complications such as hepatic encephalopathy, with a high mortality rate [6]. The mortality rate of acute UGH within 6 weeks is approximately 15%, and recurrent or multiple episodes of bleeding significantly increase mortality [7]. Rebleeding after endoscopic hemostasis indicates persistently worsening portal hypertension, incomplete sealing of vascular damage, and poor liver function. Rebleeding after treatment suggests that the patient is in a state of blood loss, anemia, and hypovolemia, with further decreased tolerance to blood loss, making them prone to recurrent shock in a short period, inducing or exacerbating complications such as infection and hepatic encephalopathy, leading to a poor prognosis. Paying attention to the disease outcomes of patients with liver cirrhosis complicated by UGH and implementing corresponding intervention measures based on risk factors is very important for improving patient prognosis.

This study shows that the proportion of patients with liver cirrhosis complicated by UGH having a good prognosis is 80.00%, indicating that although endoscopic hemostasis can achieve satisfactory results, some patients still experience rebleeding due to various factors after treatment, necessitating strengthened prevention and control. Multivariate analysis revealed that the independent risk factors for rebleeding one year after treatment in these patients include the following: (1) Age: Elderly patients have significantly reduced physiological reserve capacity, more underlying diseases, and sluggish compensatory responses. At the same degree of cirrhosis, elderly patients have poorer liver function compensation, progressive portal hypertension, which further drives variceal rupture and rebleeding. They have poor tolerance to acute blood loss, shock, and hypoxia, and also respond poorly to hemostatic treatments, with a significantly increased incidence of complications [8]. (2) Decompensated liver cirrhosis: In the decompensated stage, liver fibrosis is irreversible, and portal venous pressure continues to rise and progressively worsen. It is difficult to reverse the hypertensive state after endoscopic hemostasis, so rebleeding may still occur after treatment. Additionally, severe liver function impairment causes coagulation dysfunction, making it difficult for bleeding to stop spontaneously. (3) Hepatic encephalopathy: The occurrence of hepatic encephalopathy indicates severe impairment of liver synthetic and detoxification functions, as well as irreversible damage to central nervous function, which can easily lead to deep coma, suppression of respiration and circulation, and a high risk of death [9]. (4) Esophageal varices: Increased tension in esophageal and gastric varices reduces the vessel wall's capacity

to withstand pressure and the supportive capacity of surrounding tissues, leading to failure of endoscopic hemostasis and a tendency for recurrent bleeding, increasing the risk of death [10]. (5) Complications: Ascites and hypoalbuminemia lead to unstable blood volume. Coupled with the hyperdynamic circulation of liver cirrhosis, varices are subjected to high perfusion and high pressure over a long period, making them very prone to rebleeding. Infection increases the burden on the liver, promotes acute deterioration of liver function, and increases the risk of stress ulcers and variceal bleeding. At the same time, infection induces a systemic inflammatory response, leading to mucosal bleeding and rupture of varices. Dieulafoy's ulcer bleeding (mostly related to portal hypertensive gastropathy-associated erosions/ulcers, post-variceal ligation ulcers, stress-related mucosal lesions, etc.) is an important non-variceal cause of rebleeding after treatment in these patients. This is mainly because portal hypertension persists without relief, leading to a vicious cycle of mucosal congestion and ischemia, as well as severe destruction of the gastric mucosal defense barrier, resulting in non-healing ulcers. Coagulation dysfunction can cause continuous oozing or intermittent major bleeding from small ulcers, with recurrence shortly after hemostasis. At the same time, complications repeatedly induce ulcer activity, increasing the risk of rebleeding.

Non-independent risk factors also included Child-Pugh grade C and shock index > 1.5. The reasons for this are as follows: Child-Pugh grade C indicates poor liver function and compensatory capacity, and continuously worsening liver injury affects the secretion of coagulation factors and immunoglobulins, causing systemic internal environment disturbances. This increases the difficulty of endoscopic hemostasis, affects the effectiveness of endoscopic hemostatic treatment, and makes persistent bleeding or rebleeding very likely, thus easily leading to a poor prognosis. A shock index > 1.5 indicates a state of severe hemorrhagic shock, which can easily induce acute kidney injury, hepatorenal syndrome, and even multiple organ failure, carrying a high risk of death [11].

In response to the above risk factors, corresponding intervention measures should be formulated: A multidisciplinary approach involving the Hepatology Department and Gastroenterology Department should be adopted to develop targeted intervention plans for high-risk patients with multiple risk factors, with dynamic adjustments. After successful endoscopic hemostasis, continue intravenous administration of drugs such as somatostatin and proton pump inhibitors to consolidate the hemostatic effect; at the same time, administer drugs to lower portal pressure. Strengthen bowel management and dietary interventions, actively control predisposing factors such as esophageal varices and hepatic encephalopathy, enhance the prevention and control of complications, and strengthen cerebral function protection and liver function support. Regularly assess liver function, coagulation function, and perform gastroscopy to reduce triggers for bleeding recurrence and avoid rebleeding. Instruct patients and their families to recognize warning signs of bleeding and seek medical attention promptly if any abnormalities are detected.

5. Conclusion

In summary, factors such as age and decompensated liver cirrhosis directly affect the disease outcomes of patients with liver cirrhosis complicated by UGH after endoscopic treatment. Clinically, attention should be paid to these relevant risk factors, and active interventions should be implemented to reduce the rebleeding rate after treatment and achieve better prognostic outcomes.

Disclosure statement

The author declares no conflict of interest.

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