Predictive Factor for Hepatic Encephalopathy in Cirrhotic Patients Who Presented with Acute Variceal Bleeding

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Background: *Cirrhotic patients who were hospitalized due to acute variceal bleeding and subsequently developed hepatic encephalopathy during hospital stay had dreadful outcome and high mortality rate. Recommendations regarding management and prevention of encephalopathy in these patients are not available in the current clinical practice guidelines. Defining high-risk patients could possibly prevent or early detect hepatic encephalopathy and help develop prophylactic management.*
Objective: *To evaluate the potential of certain clinical predictors of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding.*
Material and Method: *The medical records of cirrhotic patients with diagnosis of acute variceal bleeding by endoscopy were retrospectively examined for clinical parameters. Potential predictive factors for hepatic encephalopathy were identified by univariate and multivariate analysis.*
Results: *One hundred seventy four cirrhotic patients who presented with acute variceal bleeding were enrolled in the present study. Hepatic encephalopathy was developed in 25 patients (14.4%). Multivariate analysis showed cirrhosis Child C, serum potassium <3.5 mmole/L, WBC >10,000 cells/mm^3, and hemoglobin <8 gm/dL on the day of admission were significant factors predicting hepatic encephalopathy in cirrhotic patients presenting with acute variceal bleeding (adjusted odds ratio 36.7, 9.25, 4.91, and 4.52, respectively). Cirrhotic patients presented with acute variceal bleeding who developed hepatic encephalopathy had higher rate of infection (40% vs. 5.4%), respiratory failure (20% vs. 2%), unit of red blood cell transfusion (3.8±1.8 units vs. 2.6±1.8 units), volume of fresh frozen plasma transfusion (1,000 (0-4,000) cc vs. 500 (0-5,000) cc), length of stay (9.0±3.5 days vs. 5.6±1.8 days), and mortality rate (8% vs. 0.7%), than non-hepatic encephalopathy p-value <0.05.*
Conclusion: *Cirrhotic patients presented with acute variceal bleeding with cirrhosis Child C, serum potassium <3.5 mmole/L, WBC >10,000 cells/mm^3, and hemoglobin <8 gm/dL were significant predictors for development of hepatic encephalopathy. Cirrhotic patients with acute variceal bleeding who developed hepatic encephalopathy had high morbidity and mortality rates.*

Keyword: *Predictor, Cirrhosis, Acute variceal bleeding, Hepatic encephalopathy*

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Acute variceal bleeding is a medical emergency. The mortality rate within six weeks after the first episode is approximately 20%. Hepatic encephalopathy is the predictor for recurrent episode of bleeding and the mortality of cirrhotic patients presented with acute variceal bleeding[1]. Acute variceal bleeding is the direct precipitating factor for development of hepatic encephalopathy, as a result of increasing nitrogen and ammonia production after variceal bleeding. Acute variceal bleeding, which occasionally causes arterial hypotension, hypoxemia, anemia, and dehydration can indirectly precipitate hepatic encephalopathy[2].

Cirrhotic patients with acute variceal bleeding who developed hepatic encephalopathy more likely resulted in the worst outcome and increased mortality rate[1,3,4]. Nevertheless, the prevention of hepatic encephalopathy in cirrhotic patients with acute variceal bleeding may improve the prognosis and decrease mortality rate. Praveen Sharma et al study[5] showed that using lactulose to prevent the development of hepatic encephalopathy in patients with cirrhosis and acute variceal bleeding was very effective. However, of all the clinical practice guidelines, the recommendations regarding to management and prevention of encephalopathy in patients with acute variceal bleeding, the use of lactulose to prevent

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hepatic encephalopathy in these patients were still not existed[1,6-8].

Not only acute variceal bleeding, there are also multiple factors involve in the development of hepatic encephalopathy. The purpose of the present study was to evaluate the potential of certain clinical parameters to predict development of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding.

Material and Method

The present study was a retrospective cross-sectional study based on the patients 18 to 80 years of age with cirrhosis and presented with acute variceal bleeding. All patients that were hospitalized due to acute variceal bleeding, which was confirmed by esophagogastroduodenoscopy, and were treated at Hat Yai Hospital between May 2010 and June 2012, were included in this study. The patients with one or more of the following features, previously treated with lactulose within one month, abnormal neurologic symptom by other neurologic disease, acute variceal bleeding during the admission with other condition and presented with the hepatic encephalopathy on the day of admission, were excluded. The study protocol was approved by the Ethic Committee of Hat Yai Hospital.

All data collection was done retrospectively by reviewing the in-patient charts for clinical history including age, gender, cause and history of cirrhosis. Physical signs on the day of admission were reviewed, including body temperature, blood pressure, ascites, or splenomegaly. Initial laboratory data were collected, including compete blood count, creatinine, sodium, potassium, liver functional test, coagulopathy, and ascitic fluid analysis. Child Pugh Score at the time of admission, units of blood transfusion, length of stay, and treatment outcome were all recorded.

The patients who developed hepatic encephalopathy diagnosed by using West Haven criteria during the admission were enrolled in the hepatic encephalopathy group.

Statistical analysis

Statistical analysis was performed after all the data were completely collected. The number and percentage of categorical data, the mean, and standard deviation (SD) of continuous data were calculated. Chi-square and Fisher's exact tests were used for comparison of categorical variables. Continuous variables data were analyzed by using t-test or Mann-Whitney tests; odds ratio and 95% confidence interval were calculated. The *p*-value of <0.05 was considered statistically significant. Logistic regression for multivariate analysis was performed to identify independent parameters associated between clinical parameters and development of hepatic encephalopathy in cirrhosis patients with portal hypertension bleeding.

Results

One hundred seventy four cirrhotic patients presented with acute variceal bleeding and fulfill criteria of study design were enrolled. Hepatic encephalopathy was developed in 25 patients (14.4%). Of all 174 patients, 148 (85.1%) were male. The mean age was 50.6 years old (Table 1). The most common cause of cirrhosis was alcoholic cirrhosis (79.3%). There was thrombocytopenia (platelet count below 100,000 cells/mm^3) in 110 patients (63.2%).

Table 1. Demographic data of 174 cirrhotic patients with acute variceal bleeding

Demographic data	Number (%)
Generalized characteristic	
Age (years), mean ± SD	50.6±11.3 (range 25-80)
Male	148 (85.1)
History of underlying cirrhosis	121 (69.5)
History of hepatocellular carcinoma	14 (8.0)
Cause of cirrhosis	
Alcoholic cirrhosis	138 (79.3)
Hepatitis B infection	26 (20.7)
Hepatitis C infection	26 (20.7)
Hepatitis B and C coinfection	6 (3.4)
Physical exammination (at index of admitted)	
Body temperature >37.8°C	19 (10.9)
Blood pressure <90/60 mmHg	33 (19.0)
Shiffting dullness or fluid thrill positive	54 (31.0)
Initial laboratory investigation	
WBC >10,000 cells/mm^3	63 (36.2)
Platelet <100,000 cells/mm^3	110 (63.2)
Hemoglobin <8 gm/dL	70 (40.2)
Creatinine >1.5 mg/dl	16 (9.2)
Sodium <135 mmole/L	5 (2.9)
Potassium <3.5 mmole/L	24 (13.8)
Ascites fluid PMN >250 cells/µl	4 (2.3)
Child classcification	
Child A	69 (39.7)
Child B	84 (48.3)
Child C	21 (12.1)

WBC = white blood cell count; PMN = polymorphonuclear neutrophil

Cirrhotic patients who presented with acute variceal bleeding developed hepatic encephalopathy in 25 patients. Thirteen patients (52%) developed hepatic encephalopathy within 24 hours after admission and 10 patients (40%) developed hepatic encephalopathy within 24 to 48 hours after admission. Thirteen patients (52%) who had hepatic encephalopathy were diagnosed with cirrhotic Child C. Prevalence of hepatic encephalopathy development in cirrhotic Child A (69 patients), B (84 patients), and C (21 patients) were 1.4% (1 patient), 13.1% (11 patients), and 61.9% (13 patients), respectively.

Eight significant clinical parameters were associated with the development of hepatic encephalopathy. They were fever (body temperature >37.8°C), shifting dullness or fluid thrill positive, WBC in complete blood count >10,000 cells/mm^3, hemoglobin <8 gm/dL, creatinine >1.5 mg/dL, serum potassium <3.5 mmole/L, ascitic fluid polymorphonuclear neutrophil (PMN) >250 cells/µL, and cirrhosis Child C on the day of admission (Table 2).

Univariate analysis showed that seven clinical parameters were significant predictors of the development of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding (Table 3). Those included ascitic fluid PMN >250 cells/µl (odds ratios 20.18, 95% CI 2.00-202.72, p = 0.011), cirrhosis Child C (odds ratios 19.09, 95% CI 6.62-55.10, p<0.001), serum potassium <3.5 mmole/L (odds ratios 5.03, 95% CI 1.89-13.33, p = 0.001), white blood cell count (WBC) in complete blood count >10,000 cells/mm^3 (odds ratios 4.76, 95% CI 1.92-11.81, p = 0.001), body temperature >37.8°C (odds ratios 3.30, 95% CI 1.12-9.73, p = 0.030), hemoglobin below 8 gm/dL (odds ratios 3.13, 95% CI 1.29-7.56, p = 0.011), and fluid thrill positive on the physical examination at the day of admission (odds ratios 1.65, 95% CI 1.11-2.46, p = 0.013).

Multivariate analysis showed that four clinical parameters were significant predictors of the development of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding (Table 4) including cirrhosis Child C, serum potassium <3.5 mmole/L, WBC in complete blood count >10,000 cells/mm^3 and hemoglobin <8 gm/dL

Table 2. Clinical parameter of cirrhotic patients with acute variceal bleeding develop and non-develop hepatic encephalopathy group

Patients characteristic	Hepatic encephalopathy patients (n = 25)	Non hepatic encephalopathy patients (n = 149)	p-value
Generalized characteristic			
Age ≥50 years	14 (56)	63 (42.3)	0.201
Male	22 (88)	126 (84.6)	0.656
History of underlying cirrhosis	19 (76)	102 (68.5)	0.448
History of hepatocellular carcinoma	4 (16)	10 (6.7)	0.114
Cause of cirrhosis			
Alcoholic cirrhosis	21 (84)	117 (78.5)	0.521
Hepatitis B or C infection	8 (32)	17 (11.4)	0.828
Physical exammination (at admited)			
Body Temperature >37.8°C	6 (24)	13 (8.7)	0.023*
Blood pressure <90/60 mmHg	8 (32)	25 (16.8)	0.072
Shiffting dullness or fluid thrill positive	12 (48)	41 (27.5)	0.048*
Initial laboratory investigation			
WBC >10,000 cells/mm^3	17 (68)	46 (30.9)	<0.001*
Platelet <100,000 cells/mm^3	15 (60)	95 (63.8)	0.718
Hemoglobin <8 gm/dL	16 (64)	54 (36.2)	0.010*
Creatinine >1.5 mg/dl	5 (20)	11 (7.4)	0.009*
Sodium <135 mmole/L	1 (4)	4 (2.7)	0.716
Potassium <3.5 mmole/L	9 (36)	15 (10.1)	0.001*
Ascites fluid PMN >250 cells/µl	3 (12)	1 (0.7)	<0.001*
Child pugh score at the admitted			
Child C	13 (52)	8 (5.4)	<0.001*

WBC = white blood cell count; PMN = polymorphonuclear neutrophil

Table 3. Univariate analysis of predictor of hepatic encephalopathy development in cirrhotic patients with acute variceal bleeding

Clinical predictor	Odds ratio	95% CI	*p*-value
Body temperature >37.8°C	3.30	1.12-9.73	0.030*
Shiffting dullness or fluid thrill positive	1.65	1.11-2.46	0.013*
WBC >10,000 cells/mm^3	4.76	1.92-11.81	0.001*
Hemoglobin <8 gm/dL	3.13	1.29-7.56	0.011*
Creatinine >1.5 mg/dl	3.14	0.99-9.97	0.053
Potassium <3.5 mmole/L	5.03	1.89-13.33	0.001*
Ascites fluid PMN >250 cells/µl	20.18	2.01-202.72	0.011*
Child C	19.09	6.62-55.10	<0.001*

WBC = white blood cell count; PMN = polymorphonuclear neutrophil

Table 4. Multivariate analysis of predictor of hepatic encephalopathy development in cirrhotic patients with acute variceal bleeding

Clinical predictor	Adjusted odds ratio	95% CI	*p*-value
Body Temperature >37.8°C	1.87	0.44-8.05	0.398
Shiffting dullness or fluid thrill positive	0.99	0.54-1.82	0.986
WBC >10,000 cells/mm^3	4.91	1.47-16.46	0.010*
Hemoglobin <8 gm/dL	4.52	1.31-15.55	0.017*
Potassium <3.5 mmole/L	9.25	2.42-35.31	0.001*
Ascites fluid PMN >250 cells/µl	4.80	0.12-199.25	0.409
Child C	36.67	4.86-84.57	<0.001*

WBC = white blood cell count; PMN = polymorphonuclear neutrophil

on the day of admission (adjusted odds ratio 36.7, 9.25, 4.91, and 4.52, respectively).

The cirrhotic patients presented with acute variceal bleeding and developed hepatic encephalopathy during the admission were significantly increased in rate of infection (40% vs. 5.4%, $p<0.001$), respiratory failure (20% vs. 2%, $p<0.001$), unit of red blood cell transfusion (3.8±1.8 vs. 2.6±1.8, $p = 0.004$), volume of fresh frozen plasma transfusion (1,000 (0-4,000) vs. 500 (0-5,000), $p = 0.021$), length of stay (9.0±3.5 vs. 5.6±1.8, $p<0.001$) and mortality rate (8% vs. 0.7%, $p = 0.009$) in comparison with the patient without hepatic encephalopathy (Table 5).

Discussion

The incidence of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding in the present study was 14.4%, which is similar with the study from Wen J et al[9] but in contrast with the study from Sharma P et al[5]. Both of these previous studies examined the potency of lactulose for the primary prophylaxis of hepatic encephalopathy in patients with cirrhosis and presented with upper gastrointestinal bleeding. The incidence of hepatic encephalopathy in the patients without lactulose treatment was 16.9% and 40% in the study from Wen J and Sharma P, respectively. The major cause of various incidence of hepatic encephalopathy in the present study, Wen J and Sharma P study was the difference of Child Pugh score in the population. Child Pugh score of the enrolled patients in the present study showed Child A, B, C 39.7%, 48.3%, and 12.1%, respectively. Wen J et al study Child Pugh score of the study population showed Child A, B, C 55.5%, 35.9%, and 8.6%, respectively. Finally, the study from Sharma P et al showed almost all the study population was diagnosed with cirrhosis Child. The high incidence of hepatic encephalopathy in Sharma P's study may be associated with cirrhosis Child C, which was similar to the incidence of development of hepatic encephalopathy in cirrhosis Child C in the present study (61.9%).

In the present study, cirrhosis Child C was the most significant clinical parameter for predicting

Table 5. Treatment outcome and complication of cirrhotic patients with acute variceal bleeding in develop and non-develop hepatic encephalopathy group

Treatment outcome	Hepatic encephalopathy patients (n = 25)	Non hepatic encephalopathy patients (n = 149)	*p*-value
Blood transfusion			
PRC (unit)	3.8±1.8	2.6±1.8	0.004*
Platelet (unit)	2.1±7.6	2.0±4.5	0.358
Fresfrozen plasma (cc)	1,000 (0-4,000)	500 (0-5,000)	0.021*
Complication			
Respiratory failure	5 (20%)	3 (2.0%)	<0.001*
Infection	10 (40%)	8 (5.4%)	<0.001*
Rebleeding	2 (8%)	8 (5.4%)	0.601
Death	2 (8%)	1 (0.7%)	0.009*
Length of stay (days)	9.0±3.5	5.6±1.8	<0.001*

PRC = peck red cells

the development of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding (odds ratio 36.7) and similar with the results from several previous studies[2,3,9-11]. Only the result from the study of Sharma P et al[5] showed that Child Pugh score was not a significant clinical parameter in prediction of the development of hepatic encephalopathy in cirrhotic patients with acute variceal bleeding. This is because the study population of Sharma P et al were cirrhosis Child C in both groups. Cirrhosis Child C may be considered as the major clinical parameter for predicting the development of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding.

The present study demonstrated that hemoglobin level below 8 gm/dL was the significant predictor of the development of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding. This is similar with the result from Wen J et al study[9], which found that hemoglobin level in hepatic encephalopathy group was significantly lower than the non-hepatic encephalopathy group (7.2 gm/dL vs. 8.7 gm/dL). The present study also showed that serum potassium <3.5 mmole/L and WBC in complete blood count >10,000 cells/mm^3 were significant predictors of the development of hepatic encephalopathy in cirrhotic patients with acute variceal bleeding, which were also considered as the well-established precipitating factors of hepatic encephalopathy[2,4,11,12].

The present study demonstrated that cirrhotic patients presented with acute variceal bleeding and developed hepatic encephalopathy during hospitalization had significantly increased rate of infection, respiratory failure, unit of red blood cell transfusion, volume of fresh frozen plasma transfusion, length of stay and mortality rate. The results of the present study were in agreement with several previous studies[1,3,4,13,14]. Early detection and recognition of precipitating factors for hepatic encephalopathy in cirrhotic patients who presented with acute variceal bleeding could possibly prevent the development of hepatic encephalopathy, which will result in improvement of the clinical outcome and lowering morbidity and mortality rates[5,9].

All of the existing clinical practice guidelines and recommendations regarding management and prevention of encephalopathy in patients with acute variceal bleeding were still not clearly documented[1,6-8]. There was small number of studies about efficacy of prevention of hepatic encephalopathy in cirrhotic patients with acute variceal bleeding[5,9]. In the future, when there is more clinical evidence of the efficacious prevention method for hepatic encephalopathy in cirrhotic patients with acute variceal bleeding, the recommendation for prevention of the hepatic encephalopathy in cirrhotic patients with acute variceal bleeding may be documented in the clinical practice guidelines. The present study has shown that the majority of patients with cirrhosis Child C or with a precipitating factor of hepatic encephalopathy, such as hypokalemia (serum K <3.5 mmole/L), severe anemia (hemoglobin level below 8 gm/dL), and leukocytosis (WBC in complete blood count >10,000 cells/mm^3) on the day of admission were the significant predictors for development of hepatic encephalopathy.

The limitation of the present study was a single center retrospective study and small sample size.

The data collection by reviewing IPD records, the majority of hepatic encephalopathy in the present study were overt hepatic encephalopathy, the minimal hepatic encephalopathy may be enrolled in non-hepatic encephalopathy group. In general practice, the overt hepatic encephalopathy was more important than minimal hepatic encephalopathy.

Conclusion

The clinical parameters including cirrhosis Child C, serum potassium <3.5 mmole/L, WBC in complete blood count >10,000 cells/mm^3 and hemoglobin below 8 gm/dL on the day of admission were significant predictors indicating the development of hepatic encephalopathy in cirrhotic patients presented with acute variceal bleeding. Cirrhotic patients with acute variceal bleeding with the development of hepatic encephalopathy had significant increase in rate of infection, respiratory failure, unit of red blood cell transfusion, length of hospital stay, and mortality rate.

What is already known on this topic?

Only two studies, Sharma P et al and Wen J et al study showed lactulose was effective for primary prophylaxis of hepatic encephalopathy in patients with cirrhosis and acute variceal bleeding. However, of all the clinical practice guidelines, the recommendations regarding to prevention of encephalopathy in patients with acute variceal bleeding were still not clear.

What this study adds?

Cirrhotic patients with acute variceal bleeding who were cirrhosis Child C or had the other precipitating causes of hepatic encephalopathy such as, serum potassium <3.5 mmole/L, WBC in complete blood count >10,000 cells/mm^3 and hemoglobin below 8 gm/dL on the day of admission were significant predictors indicating the development of hepatic encephalopathy. Therefore, in the cirrhotic Child C patients with acute variceal bleeding or the cirrhotic patients who had the other precipitating causes of hepatic encephalopathy, the lactulose or other management to prevent hepatic encephalopathy should be recommended.

Potential conflicts of interest

None.

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ปัจจัยทางคลินิกที่ใช้ทำนายการเกิดภาวะโรคสมองที่เกิดจากตับในผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตก

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ภูมิหลัง: *ผู้ป่วยโรคตับแข็งที่มารักษาในโรงพยาบาลเนื่องจากมีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตก หากมีภาวะโรคสมองที่เกิดจากตับเกิดขึ้นร่วมด้วยจะมีผลการรักษาที่ไม่ดีและมีอัตราการเสียชีวิตสูง แต่แนวทางในการดูแลรักษาและการป้องกันภาวะนี้ในผู้ป่วยกลุ่มดังกล่าวยังไม่มีความชัดเจน หากสามารถทราบปัจจัยเสี่ยงที่ทำให้เกิดภาวะโรคสมองที่เกิดจากตับในผู้ป่วยกลุ่มนี้ได้ ก็น่าจะนำมาใช้เป็นแนวทางในการดูแลรักษาและป้องกันภาวะโรคสมองที่เกิดจากตับในผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตกได้*

วัตถุประสงค์: *เพื่อหาปัจจัยที่มีความสำคัญทางคลินิกเพื่อใช้ทำนายการเกิดภาวะโรคสมองที่เกิดจากตับในผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตก*

วัสดุและวิธีการ: *เป็นการศึกษาแบบย้อนหลังจากการทบทวนเวชระเบียนของผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นและได้รับการส่องกล้องยืนยันแล้วว่ามีภาวะหลอดเลือดโป่งพองแตก โดยเก็บข้อมูลเกี่ยวกับปัจจัยทางคลินิกต่าง ๆ เช่น ประวัติ การตรวจร่างกาย การตรวจทางห้องปฏิบัติการต่าง ๆ แล้วนำมาวิเคราะห์ข้อมูลทางสถิติตามวัตถุประสงค์ของการศึกษา*

ผลการศึกษา: *มีผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตกที่ตรงตามข้อบ่งชี้ในการศึกษาทั้งหมด 174 ราย พบผู้ป่วยที่มีภาวะโรคสมองจากตับ 25 ราย (ร้อยละ 14.4) เมื่อทำการวิเคราะห์ข้อมูลแบบพหุตัวแปร พบว่า ระดับ child pugh score มากกว่า 9 คะแนน ระดับโพแทสเซียมในเลือดต่ำกว่า 3.5 มิลลิโมลต่อลิตร จำนวนเม็ดเลือดขาวมากกว่า 1,000 เซลล์ต่อมิลลิลิตร และค่าปริมาณฮีโมโกลบินน้อยกว่า 8 กรัมต่อเดซิลิตร ในวันที่รับไว้นอนในโรงพยาบาลจะมีความสัมพันธ์ชัดเจนต่อการเกิดโรคสมองที่เกิดจากตับในผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตก (อัตราส่วน odds เท่ากับ 36.7, 9.25, 4.91 และ 4.52 เท่า ตามลำดับ) และหากเกิดภาวะโรคสมองที่เกิดจากตับในผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตกจะทำให้มีภาวะแทรกซ้อนต่าง ๆ เพิ่มขึ้นอย่างมีนัยสำคัญทางสถิติ เมื่อเทียบกับกลุ่มที่ไม่เกิดโรคสมองที่เกิดจากตับ คือ อัตราการติดเชื้อ (ร้อยละ 40 และร้อยละ 5.4) การเกิดภาวะการหายใจล้มเหลว (ร้อยละ 20 และร้อยละ 2) จำนวนยูนิตของเม็ดเลือด (3.8±1.8 ถุง และ 2.6±1.8 ถุง) และปริมาณพลาสมาแช่แข็ง (1,000 มิลลิลิตร และ 500 มิลลิลิตร) ที่ต้องให้แก่ผู้ป่วย ระยะเวลาการนอนโรงพยาบาล (9.0±3.5 วันและ 5.6±1.8 วัน) และอัตราการเสียชีวิต (ร้อยละ 8 และ 0.7)*

สรุป: *ผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตกหากมีระดับ child pugh score มากกว่า 9 คะแนน ระดับโพแทสเซียมในเลือดต่ำกว่า 3.5 มิลลิโมลต่อลิตร จำนวนเม็ดเลือดขาวมากกว่า 1,000 เซลล์ต่อมิลลิลิตร และค่าปริมาณฮีโมโกลบินน้อยกว่า 8 กรัมต่อเดซิลิตร จะเป็นปัจจัยสำคัญที่จะทำให้เกิดภาวะโรคสมองที่เกิดจากตับ และหากผู้ป่วยโรคตับแข็งที่มีอาการเลือดออกในทางเดินอาหารส่วนต้นจากภาวะหลอดเลือดโป่งพองแตกเกิดภาวะโรคสมองที่เกิดจากตับจะทำให้มีภาวะแทรกซ้อนและอัตราการเสียชีวิตเพิ่มมากขึ้น*