Role of Clinical Parameters for Predicting the Cause of Upper Gastrointestinal Bleeding

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Background: *Upper gastrointestinal bleeding (UGIB) has been classified into portal and non-portal hypertension (PHT) bleeding causes. Differentiating the two major categories of UGIB is therefore important for selection of the appropriate empirical treatment.*

Objective: *To evaluate the potential of certain clinical parameters for predicting the cause of UGIB.*

Material and Method: *The records of patients with UGIB who underwent endoscopy within 72 hours of diagnosis were retrospectively examined for the clinical parameters. Potential predictive factors for categorizing the cause of UGIB were identified by univariate and multivariate analysis.*

Results: *One hundred forty six UGIB patients were enrolled in the present study. One hundred nine patients had non-PHT bleeding and 37 patients were PHT bleeding. Multivariate analysis identified three independent factors for predicting PHT bleeding, presence of signs of chronic liver disease or PHT (Odds ratio (OR) 51.1, $p < 0.05$), presence of underlying cirrhosis (OR 28.4, $p < 0.05$) and an initial hematocrit < 30% (OR 12.7, $p < 0.05$). A presentation with coffee ground vomitus was the only factor that indicated a reduced possibility of PHT bleeding (OR 0.1, $p < 0.05$).*

Conclusion: *The presence of underlying cirrhosis, signs of chronic liver disease or portal hypertension and an initial hematocrit < 30% were significantly correlated with PHT bleeding while the presentation of coffee ground vomitus indicated a less likely chance of PHT bleeding.*

Keywords: *Upper gastrointestinal bleeding, Portal hypertension bleeding, Non-portal hypertension bleeding, Clinical parameter, Predicting*

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Upper gastrointestinal bleeding (UGIB) is a common gastrointestinal emergency situation. The causes of UGIB have been classified into two major categories, PHT and non-PHT bleeding[1]. The most common cause of UGIB is peptic ulcer bleeding, responsible for 28 to 59% of cases[2], with a mortality rate of about 5 to 10%[3] and re-bleeding rate of about 20%[4]. The second most common cause is variceal bleeding, responsible for 12 to 31%, with a high mortality rate of 15 to 30% in six weeks and a high recurrent bleeding rate of 60% in one year[5].

Esophagogastroduodenoscopy (EGD) is the standard tool of choice for diagnosis and treatment of UGIB[2]. An early EGD performed within 24 hours after UGIB is widely accepted as the standard practice. However, Thailand, emergency EGD is seldom available in community hospitals due to a shortage of experienced personnel and budget constraints. This precludes providing for such emergency services after hours. Thus, most patients are usually resuscitated with blood transfusion as well as an empirical medical therapy until an endoscopic service is available.

According to several clinical practice guidelines regarding the pre-endoscopic medical therapy, an intravenous proton pump inhibitor (PPI) starting with 80 mg bolus dose followed with continuous infusion at 8 mg per hour is recommended for treatment of bleeding peptic ulcers[3,6] while a vasoactive agent (*e.g.* somatostatin, octreotide, terlipressin, etc) is recommended for PHT bleeding[5,7,8]. Intravenous PPI infusion before endoscopy has been shown to accelerate the resolution of signs of high-risk bleeding in bleeding ulcers as well as reducing the need for endoscopic therapy[9]. Vasoactive agents have been shown to control acute PHT bleeding in up to 70 to 80% of cases[10]. However, the benefit of these agents on non-PHT bleeding has not been proven[3].

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Ideally, an accurate clinical differentiation between PHT and non-PHT bleeding is required to guide the choice of appropriate empirical medical therapy.

The objective of this study was to assess clinical and laboratory parameters that may help to discriminate between the two categories of UGIB.

Material and Method

The present study was a retrospective study based on all patients over 18 years of age with UGIB in whom an EGD was performed within 72 hours of an index UGIB. All patients were treated as an in-patient basis at the Hat Yai Hospital between August 2008 and July 2009. All of the EGD procedures were performed by a single gastroenterologist (the author). The patients were excluded if there was one or more of the following features, prior gastric surgery, EGD within 1 year previously, current use of anticoagulants, or negative findings from the EGD. The present study protocol was approved by the Ethics Committee of the Hat Yai Hospital.

All data were collected by reviewing the in-patient charts for clinical history including age, gender, appearance of vomitus (red bloody, coffee ground), appearance of stool (red, melena), history of NSAIDs use, history of alcohol drinking and underlying cirrhosis. Physical signs at the time of the index UGIB including vital signs, signs of chronic liver disease (spider nevi, palmar erythema, gynecomastia, testicular atrophy or parotid gland enlargement), signs of portal hypertension (ascites, splenomegaly) as well as laboratory data of an initial hematocrit (Hct < 30% or > 30%), platelet count (< 100,000/mm^3 or > 100,000/mm^3) and prothrombin time (< 12.5 sec or > 12.5 sec) were all recorded.

The cause of UGIB based on the EGD findings was classified into PHT disease (esophageal varices, gastric varices and severe portal hypertensive gastropathy) and non-PHT disease including acid related disease (peptic ulcer disease, gastritis, duodenitis and esophagitis) and miscellaneous causes (Mallory-Weiss tear, esophageal ulcer and tumor, vascular abnormality, etc). The presence of high-risk stigmata of variceal bleeding (red wale sign, cherry red spot, hematin pigment, etc.) was noted. The stigmata of peptic ulcer bleeding were classified according to the Forrest classification.

Statistical analysis

Statistical analysis was performed with SPSS Program version 11.5. Univariate analysis for associations between clinical parameters and the type of UGIB was carried out using the odds ratio, 95% CI and $p < 0.05$ was considered statistically significant. Logistic regression analysis for multivariate analysis was performed to identify independent parameters associated between clinical parameters and the type of UGIB.

Results

Two hundred five patients with UGIB were identified, with 146 meeting the enrollment criteria. Fifty-nine patients were excluded for various reasons such as EGD performed over 72 hours after onset of UGIB (39 patients), prior EGD performed within one year (8 patients), prior gastric surgery (5 patients), normal EGD findings (5 patients) and use of anti-coagulants (2 patients).

Patient characteristics

One hundred forty six patients, 45 females and 101 males, with a mean age $\pm$ SD of 59.7 $\pm$ 15.8 were

Table 1. Demographic data of the 146 patients with upper GI bleeding

Clinical parameter	Number (%)
Age, mean $\pm$ SD (yrs)	59.6 $\pm$ 15.8
Sex	
Male	101 (69.2)
Clinical presentation	
Red blood vomitus	71 (48.6)
Coffee ground vomitus	33 (22.6)
Hematochezia	4 (2.7)
Melena	68 (46.6)
Clinical risk of UGIB	
History of NSAIDs use	61 (41.8)
History of alcohol drinking	37 (25.3)
Underlying cirrhosis	28 (19.2)
Physical examination	
BP < 90/60	14 (9.6)
PR > 100	54 (37.0)
Signs of chronic liver disease/PHT	53 (36.3)
Laboratory findings	
Initial Hct < 30%	117 (80.1)
Platelet count < 100,000	24 (16.4)
PT > 12.5 sec	50 (38.2)*
Empirical treatment	
Proton pump inhibitor (PPI) monotherapy	102 (69.9)
Combination of PPI and somatostatin	44 (30.1)

* n = 131 patients were tested for prothrombin time (PT)

included in the present study (Table 1). The most common clinical presentations were red blood vomitus (48.6%) and melena (46.6%). Twenty-two patients (15.1%) had more than one clinically significant symptom at the time of the index UGIB.

All patients were given empirical treatment with PPI and 44 patients (30.1%) were empirically treated with somatostatin analog as well. In patients treated with somatostatin analog (44 patients), 39 (88.6%) had signs of chronic liver disease, 36 (81.8%) presented with red blood vomitus, and 24 (54.5%) had been known for an underlying cirrhosis.

Endoscopic findings

One hundred fifteen patients (78.8%) were found to be non-PHT disease, 43 patients (29.5%) had PHT disease (Table 2) and 13 patients (8.9%) had both non-PHT and PHT diseases.

In the patients of PHT disease (43 patients), six patients (14.0%) had small esophageal varies without signs of recent PHT bleeding but had a sign of non PHT bleeding (5 patients had low risk of PU bleeding and one patient had high risk PU bleeding), 37 patients (86.0%) had signs of recent PHT bleeding that needed endoscopic treatments procedure (esophageal banding ligation). Thirty patients (81.1%) in PHT bleeding patients were given empirical treated with somatostatin and PPI before their EGD.

In the patients of non-PHT disease (115 patients), six patients (5.2%) had gastritis with large esophageal varix with signs of recent variceal bleeding and need to esophageal banding ligation. In the patients of non PHT bleeding patients (109 patients), 57 patients (52.3%) had low risk PU bleeding, 29 patients (26.6%) had a high risk PU bleeding, 13 patients (11.9%) had gastritis, duodenitis or esophagitis bleeding, and 10 patients (9.2%) had Mallory Weiss tear/esophageal ulcer bleeding

Table 2. Endoscopic findings in the 146 patients

Endoscopic finding	Number (%)
Portal hypertension disease (PHT)	43 (29.5)
PHT without signs of recent bleeding	6 (4.1)
PHT with signs of recent bleeding	37 (25.3)
Non-portal hypertension disease (non PHT)	115 (78.8)
Non PHT disease with PHT bleeding	6 (4.1)
Non PHT disease without PHT bleeding	109 (74.7)
Acid-related disease	100 (68.5)
Gastritis/duodenitis/esophagitis	13 (8.9)
Forrest class IIc or III (low risk bleeding PU)	57 (39.0)
Forrest grade Ia, Ib, IIa, IIb (high risk bleeding PU)	29 (19.9)
Other (Mallory Weiss tear/esophageal ulcer)	10 (6.8)
Both PHT and non PHT disease	13 (8.9)

Clinical parameters

Univariate analysis

Analysis of the PHT bleeding patients found that, over 90% were male, presented with signs of chronic liver disease/PHT, had initial Hct < 30% and prolonged PT. Furthermore, 86.5% had presence of red blood vomitus, 67.6% had a history of cirrhosis and only 5.4% had presence of coffee ground vomitus (Table 3). Univariate analysis showed that male gender, presence of red blood vomitus, history of alcohol drinking, history of cirrhosis or signs of chronic liver disease/PHT and laboratory findings of prolonged PT (>12.5-seconds), platelet count < 100,000 mm^3, or initial Hct < 30% were significantly associated with PHT bleeding. Additionally, presence of coffee ground vomitus and a history of NSAIDs use were significantly associated with non-PHT bleeding (Table 3).

Multivariate analysis

Multivariate analysis found that patients with signs of chronic liver disease or PHT, history of cirrhosis, or initial Hct < 30% were significantly associated with PHT bleeding (odds ratios 51.0, 28.4 and 12.7, respectively) and the only one factor, presence of coffee ground vomitus, was associated with decreased risk of PHT bleeding (odds ratio 0.07) (Table 4).

Patients with UGIB who presented with a history of cirrhosis, cirrhosis with signs of chronic liver disease/PHT, cirrhosis with initial Hct < 30% and cirrhosis with signs of chronic liver disease and an initial Hct < 30%) had a high specificity (ranged 97-98%) but a considerably low sensitivity (ranged 59-67%) for predicting PHT bleeding (Table 5). In contrast, the patients that had a only signs of chronic liver disease/PHT or initial Hct < 30% had a high sensitivity (94.6%) and negative predictive value (ranged 93-98%), but low specificity (ranged 25-83%) for predicting PHT bleeding. The high sensitivity and specificity was found in the patients who had signs of chronic liver disease/PHT with initial Hct < 30% had 86.5% and 89.0% respectively.

Table 3. Univariate analysis of clinical parameters of patients with Portal hypertension and non-portal hypertension bleeding

Clinical parameter	Non-PHT bleeding, n (%)	PHT bleeding, n (%)	Odds ratio	95% CI	p-value
Sex					
Male	67 (61.5)	34 (91.9)	7.1	2.1-24.6	0.001
Clinical presentation					
Red blood vomitus	39 (37.8)	32 (86.5)	11.5	4.1-31.9	0.000
Coffee ground vomitus	31 (28.4)	2 (5.4)	0.1	0.0-0.6	0.004
Hematochesia	4 (3.7)	0 (0)	-	-	-
Melena	55 (50.5)	13 (35.1)	0.5	0.3-1.2	0.106
Clinical risk of UGIB					
History of NSAIDs use	56 (51.4)	5 (13.5)	0.2	0.1-0.4	0.000
History of alcohol drinking	21 (19.3)	16 (43.2)	3.2	1.4-7.2	0.004
Underlying cirrhosis	3 (2.8)	25 (67.6)	73.6	19.3-290.6	0.000
Physical examination					
BP < 90/60	12 (11.0)	2 (5.4)	0.5	0.1-2.2	0.317
PR > 100	38 (34.9)	16 (43.2)	1.4	0.7-3.0	0.362
Chronic liver stigmata/PHT	18 (16.5)	35 (94.6)	88.5	19.5-401.3	0.000
Laboratory finding					
Hematocrit < 30%	82 (75.2)	35 (94.6)	5.8	1.3-25.6	0.011
Platelet count < 100,000	5 (4.6)	19 (51.4)	21.9	7.3-66.3	0.000
PT > 12.5 sec (n = 131)	18 (18.8)	32 (91.4)	46.2	12.7-167.9	0.000
Total	109	37			

Table 4. Multivariate analysis showing independent factors associated with portal hypertension bleeding

Clinical parameter	Odds ratio	95% CI	p-value
Coffee ground vomitus	0.068	0.006-0.734	0.010
Underlying cirrhosis	28.410	4.187-192.788	0.000
Signs of chronic liver disease/PHT	51.046	9.222-282.538	0.000
Hematocrit < 30%	12.714	1.072-150.784	0.021

Table 5. Sensitivity, specificity, positive predictive value (PPV) and negative predictive value (NPV) of clinical parameters for predicting PHT bleeding

Clinical parameter	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
One clinical parameter				
History of cirrhosis	67.6	97.2	89.3	89.8
Presence of signs of liver disease/PHT	94.6	83.5	66.0	97.8
Initial Hct < 30%	94.6	24.8	66.0	93.1
Two clinical parameters				
History of cirrhosis and sign of liver disease/PHT	64.9	97.2	88.9	89.1
History of cirrhosis and initial Hct < 30%	62.2	98.2	92.0	88.4
Presence of sign of liver disease/PHT and Hct < 30%	86.5	89.0	72.7	95.1
Three clinical parameters				
Cirrhosis with sign of liver disease/PHT and Hct < 30%	59.5	98.2	91.7	87.7

Discussion

Earlier studies have aimed to determine the clinical parameters for prediction of the risk of worst outcome or mortality from UGIB[11,12] using multiple scoring systems e.g. the Rockall score[11] and Blatchford score[12]. Chalasani N et al[13] and Zaman A et al [14] performed studies looking for the predictors of large varies and variceal bleeding in cirrhotic patients. To the best of the authors' knowledge, only one published study aimed to determine if certain clinical parameters could accurately predict the type of UGIB (PHT bleeding vs. non-PHT bleeding) in general practice when dealing with patients who presented with upper GI bleeding.

Pongprasobchai S et al[15] performed a prospective study in 261 patients that presented with UGIB, and found that patients with underlying cirrhosis, those with signs of chronic liver disease/PHT, and those who presented with red blood vomitus/ red NG aspirate were significant to predict PHT bleeding. They proposed the UGIB score (calculated by 3.1 x previous diagnosis of cirrhosis or the presence of signs of chronic liver disease (score 1)) + (1.5 x presence of red vomitus (score1)) + (1.2 x presence of red NG aspirate (score1)) of $\geq$ 3.1, be used to predict PHT bleeding (with a sensitivity of 85% and specificity of 81%). Based up on the proposed formula score, the known diagnosis of cirrhosis or the presence of signs of chronic liver disease was the strongest predictor (weighted score x 3.1) of PHT bleeding.

The present study confirmed the use of two parameters of the presence of underlying cirrhosis and signs of portal hypertension/chronic liver disease for prediction of PHT bleeding, with a specificity of 83.5% and 97.2% respectively. However, the present study also found that an initial Hct < 30% was also significantly associated with portal hypertension bleeding while the presence of coffee ground vomitus was less likely to indicate PHT bleeding. Understandably, the retrospective study design is the major limitation in the present study. Since NG aspirate test was not performed on every patient, the significance of the presence of red blood vomitus or red blood NG aspirate, therefore, could not be determined in the present study.

In the present study, an initial Hct < 30% was significant for predicting PHT bleeding by multivariate analysis with a high sensitivity (94.6%) and NPV (97.8%) but a low specificity (24.8%). Using this cut-off Hct value may be useful as an exclusion of PHT bleeding.

The last study, Douglas et al study[16] showed that an initial Hct < 30%, presence of red blood in the NG aspitate, and history of vomiting red blood were significantly associated with adverse outcome in UGIB patients, but not associated for predict PHT bleeding as the present study.

The limitation of the present study is a single-center retrospective study and small sample size. Although Hat Yai Hospital has tertiary care, there is only one gastroenterologist and an emergency endoscopic team is unavailable. Therefore, 39 patients were excluded from the present study because the EGD was performed over 72 hours after the onset of UGIB.

In summary, the known underlying cirrhosis or presence signs of chronic liver disease/PHT in patients who presented with UGI bleeding were probably the strongest predictors of PHT bleeding. Other clinical parameters such as an initial Hct < 30%, presence of red blood vomitus, and presence of red blood NG aspirate for predicting PHT bleeding appeared to be inconclusive based on available data. Further studies are required to define the clinical utility of these clinical factors in predicting the two major categories of upper GI bleeding.

Conclusion

In UGIB patients, known underlying cirrhosis, presence of signs of chronic liver disease/PHT and initial HCT < 30% were significantly related with PHT bleeding. In contrary, UGIB patients who presented with coffee ground vomitus were unlikely to be PHT bleeding.

Potential conflicts of interest

None.

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บทบาทการใช้ปัจจัยทางคลินิกเพื่อทำนายสาเหตุของภาวะเลือดออกในทางเดินอาหารส่วนบน

อรรถพล รัตนสุภา

ภูมิหลัง: *ภาวะเลือดออกในทางเดินอาหารส่วนบนเป็นภาวะฉุกเฉินที่พบบ่อย ซึ่งสาเหตุของภาวะเลือดออกในทางเดินอาหารส่วนบนแบ่งเป็นสองสาเหตุหลัก คือ ภาวะที่มีและไม่มีความสัมพันธ์กับความดันหลอดเลือดดำในตับสูง ซึ่งทั้งสองภาวะจะให้การรักษาที่แตกต่างกัน ดังนั้นจากใช้ปัจจัยทางคลินิกเพื่อทำนายสาเหตุของภาวะเลือดออกในทางเดินอาหารส่วนบนก่อนที่จะได้รับการส่องกล้องรักษาจึงมีความสำคัญมาก*

วัตถุประสงค์: *ศึกษาถึงปัจจัยทางคลินิกที่สามารถทำนายถึงสาเหตุของภาวะเลือดออกในทางเดินอาหารส่วนบน*

วัสดุและวิธีการ: *เป็นการศึกษาแบบย้อนหลังจากเวชระเบียนของผู้ป่วยที่มีอาการเลือดออกในทางเดินอาหารส่วนบนที่ได้รับการส่องกล้องทางเดินอาหารส่วนบนภายในเวลา 72 ชั่วโมง โดยเก็บข้อมูลเกี่ยวกับประวัติตรวจร่างกาย การตรวจปฏิบัติการเบื้องต้น การรักษาเบื้องต้น และผลการส่องกล้อง เพื่อนำมาวิเคราะห์ข้อมูล*

ผลการศึกษา: *ผู้ป่วย 146 คน ที่ทำการศึกษา พบว่ามี 37 และ 109 คน เลือดออกในทางเดินอาหารเกิดจากภาวะที่มีและไม่มีความสัมพันธ์กับความดันหลอดเลือดดำในตับสูงตามลำดับ การวิเคราะห์ข้อมูลแบบ multivariate พบว่าผู้ป่วยที่ตรวจร่างกายพบอาการแสดงของโรคตับหรือภาวะความดันในหลอดเลือดดำในตับสูง, มีโรคประจำตัวเป็นโรคตับแข็ง และระดับความเข้มข้นเลือดที่ต่ำกว่า 30% จะมีโอกาสที่เลือดออกในทางเดินอาหารส่วนบนเกิดจากภาวะความดันหลอดเลือดดำในตับสูงเพิ่มมากขึ้น 51.1, 28.4 และ 12.7 เท่า ตามลำดับอย่างมีนัยสำคัญทางสถิติ แต่หากผู้ป่วยมีอาการอาเจียนออกมาเป็นเลือดสีดำ ๆ จะมีโอกาสที่เลือดจะออกจากภาวะความดันหลอดเลือดดำในตับสูงลดลงเหลือ 0.1 เท่าอย่างมีนัยสำคัญทางสถิติ*

สรุป: *ผู้ป่วยที่เป็นโรคตับแข็ง, ตรวจร่างกายพบอาการแสดงของโรคตับหรือภาวะความดันในหลอดเลือดดำในตับสูงและระดับความเข้มข้นเลือดที่ต่ำกว่า 30% จะมีโอกาสเป็นอย่างมากที่เลือดออกในทางเดินอาหารส่วนบนเกิดจากภาวะความดันหลอดเลือดดำในตับสูงเพิ่มมากขึ้นอย่างมีนัยสำคัญทางสถิติ แต่หากผู้ป่วยมีอาการอาเจียนออกมาเป็นเลือดสีดำ ๆ จะมีโอกาสที่เลือดจะออกจากภาวะนี้น้อย*