Predictors of Variceal or Nonvariceal Source of Upper Gastrointestinal Bleeding. An Etiology Predictive Score Established and Validated in a Tertiary Referral Center

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ABSTRACT

Background & Aims. For upper gastrointestinal bleeding (UGIB), guidelines recommend pharmacological treatment before endoscopy. Therefore, it is important to establish an early diagnosis of the variceal or non-variceal source of bleeding. This study aims to analyze the clinical and laboratory parameters which are predictors of the UGIB etiology, and to develop a score for predicting variceal or non-variceal bleeding.

Methods. This study comprised patients presenting to the emergency department of a tertiary care center with UGIB, throughout a 1-year period. Clinical, ultrasound data and laboratory parameters were noted.

Results. Of the 517 patients with UGIB, 29.8% had variceal and 70.2% non-variceal bleeding. Six factors were associated with variceal hemorrhage: cirrhosis (OR=10.74, 95%CI: 3.50-32.94, p<0.001), history of variceal hemorrhage (OR=13.11, 95%CI: 3.09-55.57, p<0.001), ascites (OR=4.41, 95%CI: 1.74-11.16, p=0.002), thrombocytopenia (OR=2.77, 95%CI: 1.18-6.50, p=0.01), elevated INR (OR=4.77, 95%CI:1.47-15.42, p=0.009) and elevated bilirubin levels (OR=2.43, 95%CI:1.01-5.84, p=0.04). Two factors were associated with non-variceal bleeding: the use of NSAIDs (OR=0.32, 95%CI: 0.13-0.83, p=0.01) and of anticoagulants (OR=0.04, 95%CI: 0.00-0.89, p=0.04). A prediction score for UGIB etiology was designed based on this model. We calculated a cutoff value of 0.968, higher values being predictive of variceal bleeding. Positive predictive value (PPV) and negative predictive value (NPV) were: 82.7% and 97%, respectively. The score was validated prospectively in another group of 162 patients: PPV and NPV were 72.7% and 95.3%, respectively.

Conclusions. Several factors were identified as predictors for the etiology of UGIB. Due to its high PPV and NPV, our UGIB etiology score might be useful in predicting variceal bleeding and could assist in the selection of pharmacological therapy before endoscopy.

Key words: upper gastrointestinal bleeding (UGIB) – variceal bleeding – non-variceal bleeding – predictive factors – etiology – predictive score

INTRODUCTION

Upper gastrointestinal bleeding (UGIB) is the most common gastroenterological emergency. Despite major advances in diagnosis and treatment, UGIB remains a serious problem in clinical practice, with a mortality of 3%-14%, unchanged in the past 10 years [1, 2]. Causes of UGIB can be grouped into two categories: variceal (esophageal or gastric varices) and non-variceal (peptic ulcer, erosive gastritis, reflux esophagitis, Mallory-Weiss syndrome, tumors, etc). Emergency upper gastrointestinal endoscopy is the standard procedure recommended for both diagnosis and treatment of UGIB [3-9].

Since many hospitals do not have permanent emergency endoscopy call departments, most patients presenting with UGIB receive medical treatment before being sent to specialized centers that can perform endoscopy.

International guidelines recommend the administration of medication empirically, before undergoing endoscopy. If there is a suspicion of variceal hemorrhage, the treatment with vasoactive agents (e.g. Somatostatin, Octreotide, Terlipressin, etc) and antibiotics is recommended [7-11]. Administration of vasoactive agents can stop bleeding in up to 70-80% of cases, thereby reducing mortality [8, 12]. In cases of non-variceal
bleeding, treatment with proton pump inhibitors is indicated [3, 4]. Their administration reduces the endoscopic lesion stage and sometimes the requirement for endoscopic therapy [3, 4, 13-15].

Therefore, it is very important to guide the diagnosis towards a variceal or non-variceal bleeding before performing endoscopy. There are authors who suggest that the presence of clinical signs of cirrhosis, hematochezia, hematemesis of fresh blood, alcohol consumption are indicators of variceal hemorrhage [16-18]. On the other hand, the use of non-steroidal anti-inflammatory drugs (NSAIDs), of antiplatelet drugs, as well as the presence of ulcer dyspepsia indicate non-variceal sources [17-19]. Establishing predictive factors of variceal or non-variceal sources of UGIB is still a controversial issue.

The aim of this study was to analyze the clinical and laboratory parameters that might be predictive factors of variceal or non-variceal hemorrhage. It was also proposed to elaborate an UGIB etiology score for predicting variceal and non-variceal bleeding and to validate its accuracy.

METHODS

This is an observational, analytical, transversal, cohort study.

Patient selection criteria
All patients presenting to the emergency department of a tertiary care center (Regional Institute of Gastroenterology and Hepatology Prof. Dr. Octavian Fodor, Cluj-Napoca, Romania) with clinical diagnosis of UGIB were included in the study. The study was conducted over a 12-month period (August 2012 - July 2013). Inclusion criteria were predefined as follows: presence of hematemesis and/or melena, upper gastrointestinal endoscopy within the first 24 hours, age over 18 years. Patients who did not undergo endoscopy and those having another source of bleeding than the upper digestive tract were excluded. Patients presenting during the initial period (August 2012 - April 2013) were included in the group for establishment of an UGIB etiology score to predict variceal and non-variceal bleeding, while those presenting during the following period (May 2013 - July 2013) were included in the group for validation of the score.

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Hospital's Ethics Committee. All patients signed their informed consent.

Clinical and laboratory data
Data were acquired by medical specialists and residents in gastroenterology at the time of presentation in the emergency department. The following were noted: age, gender, past medical history, alcohol consumption, smoking and use of NSAIDs, antiplatelet drugs and anticoagulants. The presence of hematemesis and its appearance („coffee grounds” or fresh blood), of melena and hematochezia, the association of lipothyemia and sweating were also observed. Blood pressure was measured at presentation (hypotension was defined as BP <90/60mmHg), as well as heart rate (tachycardia was defined as heart rate >100 beats/min). The presence of clinical signs of liver cirrhosis was noted: angiomas, palmar erythema, gynecomastia, hepatomégaly with sharp anterior edge, splenomegaly, edema, ascites and hepatic encephalopathy.

Laboratory tests included: hemoglobin (normal value [NV] >11.5g/dl in women, >12.5g/dl in men), hematocrit (NV 35-47%), platelet count (NV 150,000-300,000/mm3), Quick time (NV <16 sec), international normalized ratio (INR, NV <1.2sec), AST (NV <37U/l), ALT (NV <42U/l), bilirubin (NV <1.2mg/dl), alkaline phosphatase (ALP) (NV 30-306U/L), gamma-glutamyl transpeptidase (GGT, NV 7-50U/L), urea (NV 10-50mg/dl) and creatinine levels (NV 0.5-1.1mg/dl).

All patients underwent ultrasound examination (performed recently or when presenting at the emergency department) (Siemens Acuson X300). The following were considered as diagnostic criteria for liver cirrhosis: abnormal hepatic contour, splenomegaly, ascites, recanalization of round ligament, pericholecystic, perigastric, or in the splenic hilum collateral circulation [20].

Esophagogastroduodenoscopy
Esophagogastroduodenoscopy (EGD) was performed in all patients within 24 hours since admission (Olympus Exera II CLE 165). Gastrointestinal bleeding causes were divided into two categories: variceal (esophageal or gastric varices, portal hypertensive gastropathy) and non-variceal (peptic ulcer, erosive gastritis, tumors, reflux esophagitis, Mallory-Weiss syndrome, Dieulafoy's lesion and angiodysplasia).

Statistical analysis
Statistical analysis was performed using the SPSS software, version 20, Chicago, IL, USA). Nominal variables were characterized using frequencies. Quantitative variables were described by mean and standard deviation or by median and percentiles (25-75%), when appropriate. The level of statistical significance was set at p<0.05.

Differences of frequencies between nominal variables were assessed with the chi-square test. Continuous variables were compared using the Student t or Mann–Whitney tests, when appropriate.

Multivariate analysis was performed using logistic regression. We included the parameters that achieved the criterion of significance at p<0.2 in the univariate analysis. The UGIB etiology score was calculated using the following formula: score = exp(c + bixi) / 1 + exp(c + bixi); where exp is the base of natural logarithms; c is the constant of the equation; b is the coefficient of the predictor variables; x is value of the parameter (0 for absence; 1 for presence). The prediction value of the score was assessed by using the area under the ROC (AUROC). The cutoVer value was chosen where sensitivity and specificity were maximal. We calculated the sensitivity, specificity, positive predictive value, and negative predictive value for the score’s cutoVer value.

RESULTS

Patients' characteristics (demographic, historical, clinical and laboratory data)

The group in which the UGIB etiology score for predicting variceal and non-variceal bleeding was calculated comprised
533 patients. Of these, 16 patients were excluded because they did not have full laboratory investigation. Thus, only 517 remained in the study. After EGD, 154 of the patients (29.8%) had variceal hemorrhage and 363 (70.2%) had non-variceal bleeding. The causes of variceal bleeding were: esophageal varices (91.6%), gastric varices (5.2%) and portal hypertensive gastropathy (3.2%). The causes of non-variceal bleeding were: peptic ulcer (55.7%), erosive gastritis (15.2%), tumors (10.7%), reflex esophagitis (6.6%), Mallory-Weiss syndrome (6.3%), Dieulafoy’s lesion (4.4%) and angiodysplasia (1.1%).

Patient demographics and history of variceal and non-variceal bleeding are shown in Table I. Clinical data are shown as a comparison between the two categories of patients in Table II and the mean and median laboratory results are presented in Table III.

Table I. Demographic data and medical history of patients with variceal and non-variceal bleeding n (%)

<table>
<thead>
<tr>
<th>Variable</th>
<th>All patients (n=517)</th>
<th>UGIB (n=363)</th>
<th>Non-UGIB (n=154)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, mean ±SD (years)</td>
<td>61.9±15.1</td>
<td>57.7±12.6</td>
<td>63.6±15.7</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Gender</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>170 (32.9%)</td>
<td>50 (32.5%)</td>
<td>120 (33.1%)</td>
<td>0.97**</td>
</tr>
<tr>
<td>Men</td>
<td>347 (67.1%)</td>
<td>104 (67.5%)</td>
<td>243 (66.9%)</td>
<td></td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td>225 (43.5%)</td>
<td>97 (63%)</td>
<td>128 (35.3%)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Smoking</td>
<td>108 (20.9%)</td>
<td>30 (19.5%)</td>
<td>78 (21.5%)</td>
<td>0.69**</td>
</tr>
<tr>
<td>History of variceal UGIB</td>
<td>62 (12%)</td>
<td>58 (41.1%)</td>
<td>4 (1.1%)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>History of non-variceal UGIB</td>
<td>37 (7.2%)</td>
<td>35 (9.6%)</td>
<td>2 (1.3%)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>History of ulcer</td>
<td>85 (16.4%)</td>
<td>16 (19%)</td>
<td>69 (19%)</td>
<td>0.02**</td>
</tr>
<tr>
<td>Use of NSAIDs</td>
<td>118 (22.8%)</td>
<td>19 (12.3%)</td>
<td>99 (27.3%)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Use of antiplatelet</td>
<td>93 (18%)</td>
<td>9 (5.8%)</td>
<td>84 (23.1%)</td>
<td>&lt;0.001**</td>
</tr>
<tr>
<td>Use of anticoagulants</td>
<td>54 (10.4%)</td>
<td>2 (1.3%)</td>
<td>52 (14.3%)</td>
<td>&lt;0.001**</td>
</tr>
</tbody>
</table>

*Student test; **χ² test

Laboratory results were dichotomised based on the normal values for a highly accurate analysis. Thus, evaluating the laboratory data of the patients at the time of presentation to the emergency department, we found that patients with variceal hemorrhage presented the following conditions more commonly: thrombocytopenia (76.6% vs. 22.9%) (p<0.001), anemia (92.9% vs. 78.5%) (p<0.001), elevated INR (94.8% vs. 47.4%), AST >2x upper normal limit (UNL) (35.1% vs. 11.6%) (p<0.001), ALT >2x UNL (10.4% vs. 6.1%) (p=0.123), elevated bilirubin levels (85.1% vs 22.6%) (p<0.001) and gamma GT levels (60.4% vs. 28.1%) (p<0.001).

### Multivariate analysis

Several models using binary logistic regression were designed for multivariate analysis. In the end, the most stable model included the following parameters: age, chronic alcohol abuse, history of variceal bleeding, use of NSAIDs, antiplatelet drugs and anticoagulants, the diagnosis of cirrhosis, hematemesis, hematochezia, lipothyria, tachycardia, ascites, encephalopathy, anemia, thrombocytopenia, elevated INR, AST >2xUNL, ALT >2xUNL, elevated bilirubin levels, elevated gamma GT levels, alkaline phosphatase, urea, creatinine. The Cox & Snell R Square model coefficient was 0.563. Six factors were found to be independently associated with variceal bleeding and other two with non-variceal bleeding (Table IV).

### UGIB etiology score for predicting variceal and non-variceal bleeding

UGIB etiology score for predicting variceal and non-variceal bleeding was calculated based on the eight factors that are independently influencing the cause of digestive bleeding, using the following formula:

\[
\text{SCORE} = \text{EXP}(-0.518 + (2.374 \times \text{dg cirrhosis}) + (2.574 \times \text{history of variceal hemorrhage}) - (1.127 \times \text{NSAIDs}) - (3.175 \times \text{anticoagulants}) + (1.483 \times \text{ascites}) + (1.019 \times \text{thrombocytopenia}) + (1.561 \times \text{elevated INR}) + (0.886 \times \text{elevated bilirubin levels})) / (1 + \text{EXP}(-0.518 + (2.374 \times \text{dg cirrhosis}) + (2.574 \times \text{history of variceal hemorrhage}) - (1.127 \times \text{NSAIDs}) - (3.175 \times \text{anticoagulants}) + (1.483 \times \text{ascites}) + (1.019 \times \text{thrombocytopenia}) + (1.561 \times \text{elevated INR}) + (0.886 \times \text{elevated bilirubin levels}))
\]

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Table III. Laboratory data of patients with variceal and non-variceal bleeding

<table>
<thead>
<tr>
<th>Variable</th>
<th>All patients (n=517)</th>
<th>UGIB (n=154)</th>
<th>Non-variceal (n=363)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>9.5±2.6</td>
<td>9.0±2.3</td>
<td>9.8±2.7</td>
<td>0.001*</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>29.4±7.8</td>
<td>27.7±7.2</td>
<td>30.1±7.9</td>
<td>0.001*</td>
</tr>
<tr>
<td>Thrombocytes (x10^9/L)</td>
<td>185,000 (113,000; 249,000)</td>
<td>104,500 (70,750; 135,750)</td>
<td>216,000 (153,000; 282,000)</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>INR (international normalized ratio)</td>
<td>1.32 (1.12; 1.69)</td>
<td>1.68 (1.49; 2.05)</td>
<td>1.2 (1.09; 1.4)</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Prothrombin time (seconds)</td>
<td>19.5 (17; 24.25)</td>
<td>24.05 (21.6; 28.63)</td>
<td>18 (16.6; 20.6)</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Urea (mg/dl)</td>
<td>71 (49; 103)</td>
<td>62 (45.75; 88.25)</td>
<td>73 (50; 111)</td>
<td>0.004**</td>
</tr>
<tr>
<td>Creatinine (mg/dl)</td>
<td>0.8 (0.64; 1.06)</td>
<td>0.78 (0.60; 1.06)</td>
<td>0.8 (0.68; 1.07)</td>
<td>0.006**</td>
</tr>
<tr>
<td>AST (U/l)</td>
<td>31 (21; 61.5)</td>
<td>60 (36.5; 100.5)</td>
<td>26 (19; 44)</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>ALT (U/l)</td>
<td>19 (13; 35)</td>
<td>27 (17.75; 43.25)</td>
<td>17 (11; 29)</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Total bilirubin (mg/dl)</td>
<td>0.9 (0.5; 1.9)</td>
<td>2.2 (1.38; 4.25)</td>
<td>0.6 (0.4; 1)</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Alkaline phosphatase (U/l)</td>
<td>168 (137.5; 257)</td>
<td>217.5 (161.8; 309.5)</td>
<td>158 (131; 218)</td>
<td>&lt; 0.001**</td>
</tr>
<tr>
<td>Gamma-glutamyl transpeptidase (U/l)</td>
<td>32 (20; 84.5)</td>
<td>72.5 (29; 149.75)</td>
<td>28 (17; 55)</td>
<td>&lt; 0.001**</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD, respective median (25; 75 percentile) *t Student test; **Mann-Whitney test. UGIB: upper gastrointestinal bleeding

Table IV. Predictors for etiology of UGIB in the multivariate analysis

<table>
<thead>
<tr>
<th>Variable</th>
<th>B</th>
<th>Odds ratio</th>
<th>95% CI</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Diagnosis of cirrhosis</td>
<td>2.374</td>
<td>10.74</td>
<td>3.50-32.94</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>History of variceal UGIB</td>
<td>2.574</td>
<td>13.11</td>
<td>3.09-55.57</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Use of NSAIDs</td>
<td>-1.127</td>
<td>0.32</td>
<td>0.13-0.83</td>
<td>0.01</td>
</tr>
<tr>
<td>Use of anticoagulant</td>
<td>-3.175</td>
<td>0.04</td>
<td>0.00-0.89</td>
<td>0.04</td>
</tr>
<tr>
<td>Ascites</td>
<td>1.483</td>
<td>4.41</td>
<td>1.74-11.16</td>
<td>0.002</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>1.019</td>
<td>2.77</td>
<td>1.18-6.50</td>
<td>0.01</td>
</tr>
<tr>
<td>High INR</td>
<td>1.561</td>
<td>4.77</td>
<td>1.47-15.42</td>
<td>0.009</td>
</tr>
<tr>
<td>High bilirubin</td>
<td>0.886</td>
<td>2.43</td>
<td>1.01-5.84</td>
<td>0.04</td>
</tr>
</tbody>
</table>

UGIB: upper gastrointestinal bleeding; NSAIDs: non-steroidal antiinflammatory drugs

DISCUSSION

This study analyzed the clinical and laboratory parameters predictive of variceal and non-variceal etiology of UGIB. Most studies in the literature have shown the role of these parameters as predictors of unfavorable evolution or of death [21-26]. In contrast, there are very few studies assessing their role in predicting the variceal or non-variceal source of hemorrhage [17, 18, 27]. It is necessary to validate the results of these studies predicting the cause of UGIB in the European population too (the cited studies addressing the Asian and North American population), as there are geographical differences regarding the causes of UGIB [1, 2]. Of the three studies, only one study calculated an UGIB etiology score for predicting variceal and non-variceal bleeding [17].

An accurate establishment of predictors of the cause of UGIB and especially the existence of a UGIB etiology score for predicting variceal and non-variceal bleeding are very useful in clinical practice because they enable choosing the most appropriate pharmacological treatment before performing EGD. Moreover, when there is a high suspicion of variceal hemorrhage, a Sengstaken–Blakemore tube can be inserted before sending the patient to specialized centers that can perform endoscopy.

This study found a relatively high prevalence of the variceal source of gastrointestinal bleeding (29.8% in the score calculation group and 27.8% in the score validation group) compared to literature data (4-14%) [1]. A study published in Turkey, which is a close geographical area to our country, reported similar prevalence of the variceal source of UGIB (30.5%) (28]. Recent data have also reported a similar prevalence of the variceal source of UGIB in the USA (33%) [29].

The analysis of patient demographics and history revealed that patients with variceal bleeding were younger and showed more often chronic alcohol abuse and a history of variceal hemorrhage (Table I). The group of patients with non-variceal bleeding presented the following common conditions: history of ulcer, use of NSAIDs, use of antiplatelet drugs and use of...
anticoagulants. From a clinical point of view, patients with variceal bleeding experienced hematemesis more frequently (Table 1). The assessment for the appearance of haematemesis noted that vomiting of fresh blood was more frequent in variceal bleeding. A percentage of 93.5% of cirrhotic patients had variceal bleeding. Patients with variceal bleeding also exhibited more frequently ascites and hepatic encephalopathy. Analysis of laboratory data showed that patients with variceal bleeding manifested more frequently thrombocytopenia, anemia, elevated INR, AST >2xUNL, ALT >2xUNL, elevated bilirubin and GGT levels.

Multivariate logistic regression showed that only six parameters are independently associated with variceal bleeding (diagnosis of cirrhosis, history of variceal hemorrhage, ascites, thrombocytopenia, elevated INR and elevated bilirubin levels) and two are independently associated with non-variceal hemorrhage (the use of NSAIDs and the use of anticoagulants). Data reported in Thailand showed that the independent predictors of variceal hemorrhage are the previous diagnosis of cirrhosis or the clinical signs of chronic liver disease, hematemesis of fresh blood vomiting and fresh blood on nasogastric aspirate [17], previous diagnosis of cirrhosis or clinical signs of chronic liver disease and hematocrit levels <30% [27]. Limitations of these studies derive from the relatively small number of patients with variceal bleeding (47 and 37, respectively). The Canadian study on a larger number of patients (215 patients with variceal hemorrhage) found that there are a few conditions important in predicting the variceal source: the history of liver disease, stigmata of chronic liver disease, excessive alcohol use, hematemesis and hematocrit 18. In all three studies, there is a constant element predicting variceal bleeding, namely the previous diagnosis of cirrhosis and the clinical signs of chronic liver disease. Our study also found the diagnosis of cirrhosis as a strong predictor for variceal bleeding (OR 10.74). Moreover, 93.5% of patients with variceal bleeding were diagnosed with cirrhosis. In the study of Pongprasobchai et al, only 36% of patients with variceal bleeding had been previously diagnosed with cirrhosis and 64% had clinical signs of chronic liver disease [17]. In the study of Alharbi et al, 77% of cases of variceal hemorrhage had a history of liver disease 18. The higher percentage of patients with cirrhosis in our group with variceal bleeding could be due to the fact that the diagnosis of cirrhosis was made using clinical signs in combination with ultrasound findings, this increasing the accuracy of diagnostic tests. Other previous studies reported the use of anticoagulants 18 and the hematemesis aspect similar to coffee grounds as the only independent risk factors for non-variceal bleeding [27].

UGIB etiology score for predicting variceal and non-variceal bleeding was established based on the eight factors that are independently influencing the cause of UGIB. Although our formula is more complex than that already published 17, which at first sight might seem a disadvantage, we found that a cutoff value of 0.968 indicated a PPV of 82.7% and a NPV of 97%. This means that a score <0.968 would indicate an extremely low likelihood of variceal bleeding and this might have major implications in clinical practice. The study of Pongprasobchai et al found a much lower positive predictive value (50%), which could be due to the smaller number of factors taken into account, and a similar negative predictive value (96%) [17].

The results of the present study and the accuracy of the UGIB etiology score for predicting variceal and non-variceal bleeding need to be validated in larger clinical trials and in more geographical regions.

CONCLUSION

The predictive factors of variceal bleeding are the diagnosis of liver cirrhosis, the history of variceal hemorrhage, ascites, thrombocytopenia, elevated INR and elevated bilirubin levels, while the predictive factors of non-variceal bleeding are the use of NSAIDs and the use of anticoagulants. The high PPV and NPV of the proposed UGIB etiology score might be useful in predicting variceal bleeding and could assist in the selection of pharmacological therapy before endoscopy.

Conflicts of interest. No conflict to declare.

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