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ORIGINAL ARTICLE

Predictors of Rebleeding and Mortality in Patients with High-Risk Bleeding Peptic Ulcers

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Abstract
Background and Aim Patients with bleeding ulcers can have recurrent bleeding and mortality after endoscopic therapy. Risk stratification is important in the management of the initial patient triage. The aim of this study is to identify the clinical and laboratory risk factors for recurrent bleeding and mortality.
Methods A prospective study was conducted in 390 consecutive patients with bleeding peptic ulcers and high-risk endoscopic stigmata, e.g., active bleeding, a non-bleeding visible vessel, adherent blood clot, and hemorrhagic dot. We tested 13 available variables for association with recurrent bleeding and 15 were tested for association with mortality. A logistic regression model was used to identify individual correlates associated with these adverse outcomes.
Results Bleeding recurred in 46 patients (11.8%) within 3 days and 21 patients (5.4%) had in-hospital mortality. In the full-factor analysis model, the incidence of recurrent bleeding was significantly higher in five of the 13 investigated variables and mortality was significantly higher in two of the 15 variables. In the final analysis model, significant risk factors for recurrent bleeding within 3 days, with adjusted odds ratios (OR), were in-hospital bleeding (OR 3.3), initial hemoglobin level <10 g/dl (OR 3.3) and ulcer ≥2 cm (OR 2.0). In-hospital bleeding was the only independent risk factor for mortality (OR 8.3).
Conclusion The study emphasizes the role of ulcer size, anemia and in-hospital bleeding as the determining high-risk predictors for adverse outcomes for bleeding peptic ulcers.

Keywords Risk factors · Logic regression model · Ulcer bleeding · In-hospital bleeder

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Introduction

Acute upper gastrointestinal (GI) bleeding from peptic ulcers is a major cause of morbidity and mortality. In recent years, treatment of bleeding ulcers has benefited from advances in endoscopic therapy, administration of high-dose intravenous proton pump inhibitors (PPIs), and management of these emergencies by specialized teams in intensive care units. However, rebleeding can occur in 10–20% of patients [1–4], and without early endoscopic re-intervention or definitive surgery, these patients are at high risk for mortality. In fact, mortality rates related to ulcer bleeding have remained essentially unchanged at 5–8% over the past 30 years [1, 5]. A number of specific risk factors, acting independently or in concert, have been proposed as predictors of recurrent bleeding and mortality after upper GI bleeding. These include both patient- and endoscopy-related factors [6–12]. Several risk stratification models have also been developed to help clinicians improve medical decision-making, particularly at the initial patient triage [4, 13–15]. However, the reported risk factors vary widely from study to study and most scoring systems examine a mixed population of acute upper GI bleeding,

i.e., variceal and non-variceal bleeding [4, 14, 15]. Only a few reports have focused on risk factors for adverse outcome after ulcer bleeding [7, 8, 11].

Recently we completed a prospective study evaluating the prevalence and outcomes of bleeding ulcers related to variable causes (nonsteroidal anti-inflammatory drug [NSAID]/antiplatelet use, *Helicobacter pylori* [*H. pylori*] infection, and non-*H. pylori* idiopathic causes) and different patient sources (outpatient bleeder, in-hospital bleeder) [16]. The present study was designed to use multivariate analysis and examine the published risk factors for their value in predicting the independent risk of rebleeding and mortality.

Methods

Patients

This was a prospective, single-center study conducted at the Department of Gastroenterology and Hepatology at Chang Gung Memorial Hospital, a university hospital and tertiary referral center in northern Taiwan. We screened consecutive adult patients with a clinical diagnosis of upper GI bleeding. The study was approved by the Institutional Review Board of Chang Gung Memorial Hospital. All patients diagnosed with upper GI bleeding underwent therapeutic endoscopy within 24 h of arrival. Only patients with active bleeding ulcers and ulcers with stigmata of a recent hemorrhage were enrolled for final analysis. We excluded patients if they were younger than 18 years or pregnant, were bleeding from a non-ulcer etiology (varices, hemorrhagic erosive gastritis, Mallory-Weiss tears, Dieulafoy's lesions, vascular ectasia, malignancies), or had a clean ulcer base in endoscopic examination, coagulopathy, or a history of a gastrectomy. A standardized questionnaire was completed for each patient. We prospectively documented patient data (age, gender, date of endoscopy); historical data (presenting symptoms, comorbid illnesses, health status on presentation, previous uncomplicated or complicated peptic ulcer history, smoking and drinking history, medications); physical findings (initial hemodynamic status including systolic blood pressure and heart rate); and, initial laboratory data (complete blood counts, prothrombin time, and renal function). Hemodynamic instability was defined as systolic blood pressure <100 mmHg or heart rate >100 beats per minute. Two investigators (CL Cheng and HT Cheng) scrutinized patients for recent use of NSAIDs and antiplatelet agents (aspirin, clopidogrel, dipyridamole, ticlopidine). Exposure to these medications was defined as at least one dose of the drug taken within 1 week before bleeding. Grading of overall health and comorbidity was performed according to the American Society of Anesthesiology (ASA) classification as follows: grade 1, healthy patients; grade 2, mild systemic illness; grade 3, severe but not incapacitating systemic illness; and grade 4, life-threatening illness. Risk assessment of acute peptic ulcer bleeding was evaluated according to the Rockall classification [4]. All information was collected from direct interview of the patients and from medical records while they were hospitalized.

Endoscopic Evaluation

All patients gave written informed consent before the endoscopy, which was carried out by attending physicians with more than 4 years of experience performing the procedure. An ulcer was defined as a lesion with loss of mucosal integrity and continuity of ≥ 5 mm and an apparent depth of ≥ 1 mm. The diameter of the ulcer was measured using gastric biopsy forceps (Olympus FG-25K) as the standard. These forceps measure 5 mm between the tips when open. An apparent depth of ≥ 1 mm was based on the endoscopist's discretion. Bleeding activity was classified according to the modified Forrest criteria [17]. The commonly used hemostatic procedures were epinephrine injection therapy (1:10,000) in combination with heater probe thermocoagulation, argon plasma coagulation, or hemoclipping. The mode of therapy was chosen at the discretion of the endoscopists.

H. pylori Status Evaluation

After successful hemostasis, gastric biopsies were obtained from the majority of the patients during index endoscopy for an *H. pylori* diagnosis. Biopsy specimens were taken from the antrum (two biopsies), corpus (two biopsies), and gastric ulcer edge (two biopsies) for a rapid urease test (Pronto dry test; Medical Instruments Corp., Z.I. Nord, Brignais, France) and histology examination using hematoxylin and eosin stain. Two biopsy specimens from the antrum were used for the rapid urease test, and the other four specimens were sent for histological examination. If neither of the endoscopic diagnostic tests could be performed because the patient could not tolerate the prolonged procedure, a serology test was then used to detect *H. pylori* infection in a previously untreated patient. *H. pylori* infection was considered present if a patient had a positive result in any of the endoscopic tests or, if previously untreated, in the serological examination.

Definitions

We defined an *H. pylori* ulcer by a positive test for *H. pylori* and no exposure to NSAIDs and/or antiplatelet agents within 1 week before the bleeding episodes. Patients who used one of these drugs within 1 week before the ulcer

bleeding were classified as having NSAID ulcers regardless of their *H. pylori* status. Non-*H. pylori* idiopathic ulcers were defined as ulcers without prior exposure to NSAIDs or antiplatelet agents in the week before bleeding began, and a negative diagnostic test for *H. pylori*. Patients who were admitted for ulcer bleeding were defined as outpatient bleeders and those who bled while hospitalized as in-hospital bleeders.

Outcome Evaluation

Rebleeding was defined as a new episode of bleeding after the initial bleeding had stopped, and was based on clinical suspicion defined by recurrent hematemesis or hematochezia, fresh blood in the nasogastric aspirate, or circulatory instability. After successful endoscopic hemostasis, we recorded the clinical outcomes based on the rebleeding episodes (up to 30 days), number of units of packed red blood cells transfused, the need for surgical intervention, the number of days hospitalized, and the in-hospital mortality rate for all patients.

Statistical Analysis

Data were submitted for statistical testing using the Strategic Applications System (SAS, version 9.1; SAS Institute Inc., Cary, NC). The patients' baseline characteristics were presented as descriptive data. Student's *t*-test and Pearson χ^2 test were used as appropriate. The logistic regression model was used to identify individual correlates associated with the risk of recurrent bleeding and mortality. Using the backward stepwise method, variables with a *P*-value less than 0.10 were included in a final multiple logistic regression model. Goodness-of-fit for the final logistic regression model was assessed by the negative log likelihood criterion, multiplied by 2. Odds ratios (OR) and 95% confidence intervals (CI) were estimated where appropriate. A two-sided *P*-value less than .05 was considered statistically significant.

Results

A total of 390 patients (263 men, 127 women; mean age 63 ± 16) were enrolled in the study (Table 1). According to our definition, NSAID ulcers were noted in 223 patients (57.2%), *H. pylori* ulcers in 102 (26.2%), and non-*H. pylori* idiopathic ulcers in 65 (16.7%). A total of 298 patients (76.4%) were outpatient bleeders and 92 (23.6%) were in-hospital bleeders.

The outcomes have been reported previously [16]. Overall, 46 patients (11.8%) developed recurrent bleeding within 3 days after initial successful endoscopic hemostasis and 66 patients (16.9%) had rebleeding within 30 days. In-hospital mortality occurred in 21 patients (5.4%). There was no statistically significant difference between patients with NSAID ulcers, *H. pylori* ulcers, and *H. pylori*-negative idiopathic ulcers in terms of the 3-day recurrent bleeding rate (10.8% vs. 9.8% vs. 18.5%) or 30-day recurrent bleeding rate (13.9% vs. 19.6% vs. 23.1%). However, patients with non-*H. pylori* idiopathic ulcers had a significantly higher mortality rate than patients with NSAID ulcers and *H. pylori* ulcers (12.3% vs. 4.5% vs. 2.9%, respectively, $P = 0.02$). The mortality rate increased to 16.3% in patients with in-hospital bleeding and was elevated to 28.3% if the in-hospital bleeding was caused by non-*H. pylori* idiopathic ulcers.

Table 1 Patient characteristics

Number of patients	390
Male	263 (67.4%)
Age older than 65 years	210 (53.9%)
History of prior ulcer bleeding	128 (32.8%)
NSAID and/or antiplatelet exposure	223 (57.2%)
Active *H. pylori* infection	217 (55.6%)
Current smokers	120 (30.8%)
Unstable hemodynamics at presentation	223 (57.2%)
Initial hemoglobin level <10 g/dl	212 (54.4%)
ASA grades ≥3	237 (60.8%)
Rockall scores ≥5	228 (58.5%)
In-hospital bleeders	92 (23.6%)

Independent Risk Factors for Recurrent Ulcer Bleeding

Full-Factor Analysis Model

A total of 46 patients developed recurrent bleeding within 3 days and 66 patients had rebleeding within 30 days. The following 13 factors were evaluated in the full logistic analysis for recurrent ulcer bleeding: age <65 versus ≥65 years, gender, smoking habits, initial hemoglobin level <10 versus ≥10 g/dl, presence of unstable hemodynamics at presentation, ratio of blood urea nitrogen (BUN) to creatinine (Cr) <20 versus ≥20, ASA grade ≤2 versus ≥3, presence of any comorbidity, cause of ulcers (NSAID ulcer vs. non-*H. pylori* idiopathic ulcer and, *H. pylori* ulcer vs. non-*H. pylori* idiopathic ulcer), outpatient bleeder versus in-hospital bleeder, ulcer size ≥2 versus <2 cm, Rockall grade ≥5 versus <5, and Forrest severity ≥IIc versus <IIb. The five factors that significantly influenced the risk of 3-day recurrent bleeding by full logistic model were initial hemoglobin level <10 g/dl, in-hospital bleeding, male sex, ulcer size ≥2 cm and Forrest severity ≥IIc (Table 2). The four significant risk factors for 30-day recurrent bleeding

Table 2 Full model of risk factors for 3-day rebleeding

Variable	Odds ratio	95% confidence intervals	*P*-value
Initial Hb level <10 g/dl	2.477	1.067, 5.750	0.0347
In-hospital bleeders	2.136	1.024, 4.453	0.0430
Male sex	2.189	0.955, 5.021	0.0642
Ulcer size ≥2 cm	1.857	0.918, 3.759	0.0853
Forrest severity ≥IIc	0.506	0.232, 1.102	0.0862
ASA grades ≥3	3.047	0.593, 15.663	0.1823
Idiopathic ulcers	1.696	0.737, 3.901	0.2139
Age <65 years	1.431	0.685, 2.990	0.3400
BUN/Cr ratio >20	0.720	0.360, 1.442	0.3541
Any comorbidity	1.805	0.178, 18.322	0.6177
Current smokers	0.863	0.382, 1.951	0.7232
Unstable hemodynamics	0.951	0.441, 2.055	0.8991
Rockall grade ≥5	1.080	0.263, 4.431	0.9153

by full logistic model included Forrest severity ≥IIc, initial hemoglobin level <10 g/dl, male gender, and in-hospital bleeding (Table 3).

Final Analysis Model

In addition to the causes of ulcers, only those risk factors that reached a *P*-value cut-off of 0.10 or less were used to fit the final logistic regression model. Table 4 shows the results of stepwise multiple logistic regression analysis from the pool of five potential risk factors for 3-day recurrent bleeding in the full logistic model. Three risk factors were significant according to the final logistic model: ulcer size ≥2 cm (OR 2.0), initial hemoglobin level <10 g/dl (OR 3.3), and in-hospital bleeding (OR 3.3). Table 5 shows the results of the final logistic regression analysis from the pool of four potential risk factors for 30-day recurrent bleeding in the full logistic model. Two risk factors were significant according to the final logistic model, initial hemoglobin level <10 g/dl (OR 3.5), and in-hospital bleeding (OR 3.4).

Table 3 Full model of risk factors for 30-day rebleeding

Variable	Odds ratio	95% confidence intervals	*P*-value
Forrest severity ≥IIc	0.397	0.200, 0.785	0.0079
Initial Hb level <10 g/dl	2.455	1.220, 4.942	0.0118
Male sex	2.491	1.208, 5.137	0.0135
In-hospital bleeders	2.251	1.154, 4.394	0.0174
Ulcer size ≥2 cm	1.637	0.872, 3.074	0.1254
Age <65 years	1.457	0.759, 2.796	0.2578
Rockall grade ≥5	1.651	0.488, 5.593	0.4202
Idiopathic ulcers	1.346	0.639, 2.837	0.4339
ASA grades ≥3	1.661	0.458, 6.032	0.4403
Current smokers	0.794	0.393, 1.605	0.5210
BUN/Cr ratio >20	0.842	0.456, 1.555	0.5824
Any comorbidity	1.197	0.276, 5.204	0.8101
Unstable hemodynamics	0.973	0.501, 1.893	0.9369

Table 4 Reduced model for 3-day rebleeding

Risk factors	Odds ratio	95% confidence intervals	*P*-value
In-hospital bleeders	3.289	1.674, 6.459	0.0005
Initial Hb level <10 g/dl	3.307	1.501, 7.288	0.0030
Ulcer size ≥2 cm	2.029	1.033, 3.986	0.0400
Forrest severity ≥IIc	0.492	0.239, 1.014	0.0546
Male sex	1.849	0.887, 3.854	0.1011

Table 5 Reduced model for 30-day rebleeding

Risk factors	Odds ratio	95% confidence intervals	*P*-value
In-hospital bleeders	3.421	1.751, 6.685	0.0003
Initial Hb level <10 g/dl	3.528	1.613, 7.719	0.0016
Forrest severity ≥IIc	0.493	0.241, 1.013	0.0541
Male sex	1.783	0.861, 3.692	0.1196

Independent Risk Factors for Mortality

Full-Factor Analysis Model

Twenty-one patients died during their stay in the hospital. The following 15 factors were evaluated in the full logistic analysis model for in-hospital mortality; age <65 versus ≥65 years, gender, smoking habits, initial hemoglobin level <10 versus ≥10 g/dl, presence of unstable hemodynamics at presentation, ratio of BUN to Cr < 20 versus ≥20, ASA grade ≤2 versus ≥3, presence of any comorbidity, cause of ulcers (NSAID ulcer versus non-*H. pylori* idiopathic ulcer and, *H. pylori* ulcer versus non-*H. pylori* idiopathic ulcer), outpatient bleeder versus in-hospital bleeder, need for surgical intervention, ulcer size, Forrest severity, Rockall score, and recurrence of bleeding within 30 days. In-hospital bleeding and idiopathic ulcers were found to be significant risk factors by full-analysis model for mortality (Table 6).

Final Analysis Model

Only those risk factors that reached a *P*-value cut-off of 0.10 or less were used to create the final logistic regression

Table 6 Full model of risk factors for in-hospital mortality

Variable	Odds ratio	95% confidence intervals	*P*-value
In-hospital bleeders	4.247	1.395, 12.929	0.0109
Idiopathic ulcers	2.903	0.960, 8.780	0.0591
Current smokers	2.024	0.639, 6.415	0.2307
Male sex	1.781	0.523, 6.068	0.3561
Rebleeding with 30 days	1.566	0.552, 4.439	0.3988
BUN/Cr ratio >20	0.692	0.253, 1.891	0.4725
Unstable hemodynamics	1.392	0.436, 4.444	0.5766
Initial Hb level <10 g/dl	0.780	0.252, 2.417	0.6671
Age <65 years	0.806	0.264, 2.459	0.7045
Ulcer size ≥2 cm	1.100	0.398, 3.046	0.8538
Rockall grade ≥5	>999	<0.001, >999	0.9212
ASA grades ≥3	>999	<0.001, >999	0.9443
Forrest severity ≥IIc	1.019	0.347, 2.993	0.9726
Need for surgery	>999	<0.001, >999	0.9840
Any comorbidity	0.143	<0.001, >999	0.9926

Table 7 Reduced model for in-hospital mortality

Variable	Odds ratio	95% confidence intervals	*P*-value
In-hospital bleeders	8.353	3.089, 22.589	<0.0001
Idiopathic ulcers	2.257	0.850, 5.994	0.1024

model. The result of final logistic regression from the pool of two potential risk factors for mortality in the full analysis model showed that in-hospital bleeding was the only independent risk factor for mortality (OR 8.3; 95% CI, 3.0–22.6; $P < 0.0001$) (Table 7).

Discussion

In this report, we described factors that were independently associated with the risk for an adverse outcome in a cohort of 390 patients presenting with bleeding peptic ulcers. We demonstrated that an initial low hemoglobin level and in-hospital bleeding were associated with rebleeding within 30 days and in-hospital bleeding was the only independent determinant for mortality. These clinical and laboratory factors are identifiable during the initial emergency evaluation of the bleeding ulcer, are useful for predicting outcomes, and should influence decisions about when an endoscopy should be performed and what level of care is appropriate for these patients.

Approximately 80% of ulcers will stop bleeding spontaneously without recurrence. Most morbidity and mortality occur among the remaining 20% which have continued or recurrent bleeding [18]. Identifying patients at high risk for recurrent bleeding on the basis of clinical, laboratory, and endoscopic variables is critically important in the management of bleeding ulcers. Many risk stratification schemes use both clinical and endoscopic criteria [4, 13, 15]. Rockall and his colleagues showed that low-risk patients (score ≤2, defined as under age 60, with no symptoms of shock, no concurrent disease, no endoscopic stigmata from a recent hemorrhage apart from Mallory-Weiss lesions) had a rebleeding rate of 4% and a mortality rate of 0.1%. On the other hand, high-risk patients (score ≥ 8) had a rebleeding rate of 48% and a mortality rate of 39% [4]. However, scores based on clinical and endoscopic findings are of limited practical value because patients must often be triaged before endoscopy. Reports disagree on the additional prognostic power provided by endoscopic findings [9, 11]. Another study claimed that nonendoscopic risk stratification was better than the endoscopy-based Rockall score in predicting the need for clinical intervention and had a higher correlation with the proxy markers of clinical severity, such as length of hospital stay and units of blood transfused [14]. A retrospective observational study involving 335 outpatients with ulcers or variceal bleeding found five clinical variables (an initial hematocrit <30%, red blood in the nasogastric lavage, hypotension, hematemesis, and a history of portal hypertension) that were independent predictors for an adverse outcome. A decision rule based on these clinical risk factors allows accurate initial risk stratification [19]. Our primary aim was to evaluate the prevalence and outcome of bleeding ulcers related to NSAID/antiplatelet use, *H. pylori* infection, and non-*H. pylori* idiopathic causes. As a secondary goal, logistic regression was used to identify any independent risks for adverse outcomes.

There is a large body of evidence on risk stratification using clinical and laboratory criteria, with variations in patient samples, standards of care, and analytic approaches. In our study, we used multivariate analysis to evaluate risk in patients with bleeding ulcers and high-risk endoscopic stigmata. The rebleeding rates of 11.8% within 3 days and 16.9% within 30 days in our study are comparable to those reported in other studies which included a more general population of patients [1, 7]. Our study demonstrated that factors in sicker patients (initial hemoglobin level <10 g/dl and in-hospital bleeding) are independent risks for rebleeding. These findings are broadly compatible with other studies. In studies that used multivariate analysis to evaluate risk factors among patients with nonvariceal upper GI bleeding, the clinical predictors of increased risk for persistent or recurrent bleeding included age older than 65 years [7], hypotension [7, 8], poor overall health [1, 13], comorbid illness [8, 13], low initial hemoglobin level [8], and fresh blood in the emesis [7]. Other significant clinical predictors of persistent or recurrent bleeding reported in a recent review study included melena, fresh red blood on

digital rectal exam, and fresh blood in the nasogastric lavage [6].

In this study, in-hospital mortality occurred in 21 patients (5.4%). Reported mortality rates related to bleeding ulcers range from 5 to 14%. Most of these studies included a more general population of patients [1–3]. By contrast, we evaluated patients with high-risk ulcers, i.e., ulcers with active bleeding or stigmata from a recent hemorrhage. Most of our patients died of their underlying comorbid illness (18 out of 21 patients), and this finding was in agreement with a previous report [10]. The reported relationships between *H. pylori* infection and bleeding ulcer outcomes using multivariate analysis have been controversial in the literature. One study concluded that *H. pylori* negativity is an independent predictor (OR 3.2) of unfavorable outcome (surgery or mortality) in duodenal ulcer bleeding [20]. Another study found that *H. pylori* infection is an independent risk factor (OR 3.5) of mortality in peptic ulcer bleeding [3]. In our study, we found that *H. pylori* infection and the use of NSAIDs did not increase the risk of bleeding mortality. The only independent predictor of mortality in our study was in-hospital bleeding (OR 8.3), which is in concordance with a previous report [1, 3]. The mortality rate was 16.3% in patients with in-hospital bleeding in this study, and this high mortality rate is in line with data from Europe and North America [1, 3]. Other reported independent clinical predictors for increased risk of mortality include age >60 years, hemodynamic instability, ASA grade ≥3, comorbid illness, continued or recurrent bleeding, hematemesis, fresh blood on rectal examination, bright red blood in the nasogastric tube aspirate, elevated urea level, serum Cr level >150 μmol/l, elevated serum aminotransferase levels, and sepsis [1, 6].

A large ulcer is also an important predictor of rebleeding [11, 21]. In our present study, an ulcer larger than 2 cm in diameter was a significant risk factor for recurrent bleeding within 3 days, but not within 30 days. This implies that rebleeding of large ulcers usually occurs within 3 days. The relationship between ulcer location and rebleeding risk is well known. Ulcers located on the posterior wall of the duodenal bulb and over the lesser curvature side of the high body have a higher risk of rebleeding [21]. In our present study, the numbers of patients with ulcers in these "difficult" locations were too small and further statistical analysis was difficult. This was a potential limitation of our study.

Endoscopic hemostasis and proton pump inhibitor use have been shown to reduce recurrent bleeding and mortality after nonvariceal upper GI bleeding. Decreased rebleeding was associated with PPI use in all patients regardless of endoscopic stigmata (OR 0.53), and with endoscopic hemostasis in patients with high-risk stigmata (OR 0.39). PPI use (OR 0.18) and endoscopic therapy (OR 0.31) were also each independently associated with decreased mortality in patients with high-risk stigmata [1]. High-dose proton pump inhibitor therapy has been demonstrated to significantly reduce rebleeding in patients with high-risk stigmata following endoscopic therapy [22–24]. The nonendoscopic risk stratification of patients into low- and high-risk categories for adverse outcomes is undoubtedly important for proper initial patient triage. However, it is well known that physicians are unwilling to accept these risk stratification schemes and this explains why many patients who are at low risk for adverse outcome often are inappropriately hospitalized for prolonged periods [25]. A lack of clear objective evidence that risk stratification is a better prognostic method than clinical judgment may be partly to blame. Direct evidence showing that this risk score is beneficial to resource use is also limited. Further studies to validate the usefulness of initial risk stratification on patient management and resource utilization are needed.

In conclusion, multivariate analysis indicates that an initial hemoglobin level <10 g/dl and in-hospital bleeding carry an increased risk of adverse outcome after ulcer bleeding. The causes of bleeding were not relevant to the prognosis. Risk stratification will allow physicians to better identify patients who are at significant risk, provide emergency endoscopic hemostasis, and order intensive pharmacological therapy. Additional studies are required to validate our findings and document the impact of initial risk stratification on actual patient management.

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