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BRIEF ARTICLE

Somatostatin adjunctive therapy for non-variceal upper gastrointestinal rebleeding after endoscopic therapy

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Abstract

AIM: To evaluate the effect of pantoprazole with a somatostatin adjunct in patients with acute non-variceal upper gastrointestinal bleeding (NVUGIB).

METHODS: We performed a retrospective analysis of a prospective database in a tertiary care university hospital. From October 2006 to October 2008, we enrolled 101 patients with NVUGIB that had a high-risk stigma on endoscopy. Within 24 h of hospital admission, all patients underwent endoscopic therapy. After successful endoscopic hemostasis, all patients received an 80-mg bolus of pantoprazole followed by continuous intravenous infusion (8 mg/h for 72 h). The somatostatin adjunct group (n = 49) also received a 250-μg bolus of somatostatin, followed by continuous infusion (250 μg/h for 72 h). Early rebleeding rates, disappearance of endoscopic stigma and risk factors associated with early rebleeding were examined.

RESULTS: Early rebleeding rates were not significantly different between treatment groups (12.2% *vs* 14.3%, P = 0.766). Disappearance of endoscopic stigma on the second endoscopy was not significantly different between treatment groups (94.2% *vs* 95.9%, P = 0.696). Multivariate analysis showed that the complete Rockall score was a significant risk factor for early rebleeding (P = 0.044, OR: 9.080, 95% CI: 1.062-77.595).

CONCLUSION: The adjunctive use of somatostatin was not superior to pantoprazole monotherapy after successful endoscopic hemostasis in patients with NVUGIB.

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Key words: Somatostatin; Pantoprazole; Gastrointestinal bleeding; Rebleeding

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INTRODUCTION

The prevalence of non-variceal upper gastrointestinal bleeding (NVUGIB) is > 100 per 100 000 people yearly[1,2].

The number of NVUGIB cases has increased over recent years, due to the increasing use of non-steroidal anti-inflammatory drugs (NSAIDs) and antiplatelet drugs[3]. Among the conditions that lead to NVUGIB episodes, the most common is peptic ulcer disease[4]. Despite recent advances in endoscopic management of patients with NVUGIB, the overall mortality rate has remained at 5%-10% for several decades[4,5]. Therefore, there is a need to develop additional medical therapies that will improve the maintenance of hemostasis.

Data from *in vitro* studies have shown that platelet aggregation, the initial step of hemostasis, proceeds optimally at neutral pH. In a slightly acidic environment, platelet aggregation is impaired, and at pH < 6, it is virtually abolished. In acidic gastric juice, pepsinogen is processed to activated pepsin, which readily digests freshly formed blood clots within minutes. Furthermore, plasmin-mediated fibrinolysis impairs fibrin reinforcement of the initial platelet clot. It is important to understand these aspects, because ulcer rebleeding may be caused by early dissolution of the blood clot[6,7]. Thus, maintaining intragastric pH above 6 is important in the management of patients with NVUGIB. The use of a proton pump inhibitor (PPI), like omeprazole or pantoprazole, reduces the risk of rebleeding and death; thus, this has become the standard of care in patients with NVUGIB after endoscopic hemostasis[8-11].

Somatostatin and its analogs have been shown to induce hemostasis in variceal bleeding[12]. Somatostatin inhibits the release of vasodilator hormones, such as glucagon, indirectly causing splanchnic vasoconstriction and decreased portal inflow. It has a short half-life and disappears within minutes of bolus infusion[13]. Somatostatin exerts profound inhibitory effects in several gastrointestinal functions, including the secretion of gastric acid, gastrin, and pepsin[14]. The inhibition of pepsin secretion can stabilize clots or fibrin plugs that are readily digested by proteolytic activity[15,16]. also, it might offer an advantage over drugs that only inhibit gastric acid secretion, such as histamine 2 receptor antagonists and PPIs. In addition, without altering renal hemodynamics, somatostatin also induces reductions in portal venous volume, superior mesenteric blood flow, and gastric blood flow, which are positively correlated with rebleeding rates in patients with peptic ulcer bleeding[17,18]. Previously, Jenkins *et al*[19] have reported that somatostatin is an effective treatment for the control of NVUGIB in high-risk patients, i.e. those in whom hemorrhage does not cease spontaneously or is likely to recur. In a meta-analysis that compared somatostatin to histamine 2 receptor antagonists and placebo, somatostatin was more effective at reducing the risk for continued bleeding or rebleeding and at reducing peptic ulcer bleeding[20]. In addition, somatostatin has been suggested to be more effective than pantoprazole in maintaining high gastric pH during the first 12 h of infusion[21]. Rebleeding episodes often occur within 24 h in the majority of patients[22], therefore, we hypothesized that the use of somatostatin as an adjunct to pantoprazole potentiates hemostasis in patients at high risk for rebleeding.

There have been no reports about the use of somatostatin as an adjunct to a PPI in patients with NVUGIB. This retrospective report of prospectively collected data investigated the effect of using a somatostatin adjunct in patients with NVUGIB under high-risk conditions. We also analyzed risk factors for early rebleeding.

MATERIALS AND METHODS

Patients

We reviewed the medical records of 205 patients who were admitted for NVUGIB to the emergency room at the Pusan National University Hospital in South Korea, from October 2006 to October 2008. We maintained a prospective database of patients investigated for NVUGIB. These data was analyzed retrospectively. This was not a blinded study.

The clinical Rockall score was calculated at the time of admission. Thereafter, the complete Rockall score was determined according to endoscopic findings[23]. A Forrest classification was also described according to endoscopic findings[24]. Patient demographic details, including symptoms of gastrointestinal hemorrhage, comorbidity, relevant drug history, initial biochemistry, and hematological profiles were recorded at admission (Table 1).

Patients who had endoscopic high-risk stigma (spurting, oozing and visible vessel) were included. Patients were excluded when they presented with an esophageal or gastric varix, pregnancy, < 18 years old, previous history of gastric surgery, a known allergy to somatostatin or pantoprazole, renal failure (creatinine > 2 mg/dL), bleeding from gastrointestinal cancer, or deficient hemostasis (platelet count < 50000/mL and international normalized ratio of the prothrombin time > 1.5). Finally, a total of 101 patients were enrolled.

All patients gave informed consent before the initiation of endoscopic procedures and somatostatin administration. The study was approved by the ethics committee of the Institutional Review Board.

Procedures

Any use of antiplatelet agents, NSAIDs, or anticoagulants was discontinued after admission. All endoscopy procedures, including thermal techniques and mechanical hemostasis with clipping devices, were performed by experts that had > 3 years experience in performing therapeutic endoscopy. Endoscopic procedures were performed within 24 h after hospital admission with an Olympus GIF Q260 endoscope. If adherent clots were observed, they were removed by endoscopic forceps. During endoscopy, when a stigma of a recent hemorrhage was observed, endoscopic injection therapy (epinephrine diluted 1:10000 in 0.9% saline) was performed with either hemoclips or monopolar coagulation with coagulation forceps, depending on the preference of the endoscopist.

Table 1 Clinical characteristics of treatment groups (mean ± SD) *n* (%)

	Pantoprazole group (*n* = 52)	Somatostatin group (*n* = 49)	Total cohort (*n* = 101)	*P* value
Sex (male)	19 (36.5)	13 (26.5)	32 (31.7)	0.280
Age (yr)	65.44 ± 19.46	64.24 ± 14.13	64.86 ± 17.01	0.735
Hemodynamic shock	26 (50.0)	27 (55.1)	53 (52.5)	0.608
Helicobacter pylori infection	14 (26.9)	8 (16.3)	22 (21.8)	0.197
Hemoglobin (g/dL)	8.56 ± 2.84	8.26 ± 2.61	8.41 ± 2.72	0.857
Hemoglobin < 7 g/dL	17 (32.7)	16 (32.7)	33 (32.7)	0.997
Blood urea nitrogen (mg/dL)	40.20 ± 27.06	39.47 ± 26.83	39.84 ± 26.82	0.920
Creatinine (mg/dL)	1.17 ± 0.80	1.29 ± 1.33	1.23 ± 1.09	0.187
Albumin (g/dL)	3.12 ± 0.54	2.79 ± 0.59	2.96 ± 0.59	0.173
Type 2 diabetes mellitus	12 (23.1)	16 (32.7)	28 (27.7)	0.283
Hypertension	22 (43.3)	19 (38.8)	41 (40.6)	0.718
Heart failure	7 (13.5)	4 (8.2)	11 (10.9)	0.393
Ischemic heart disease	15 (28.8)	11 (22.4)	26 (25.7)	0.462
Antiplatelet medication	24 (46.2)	20 (40.8)	44 (43.6)	0.589
NSAID	6 (11.5)	3 (6.1)	9 (8.9)	0.340
Multiple antiplatelet medications	5 (9.6)	2 (4.1)	7 (6.9)	0.274
Steroids	2 (3.8)	4 (8.2)	6 (5.9)	0.359
Melena	31 (59.6)	28 (57.1)	59 (58.4)	0.801
Hematemesis	28 (53.8)	32 (65.3)	60 (59.4)	0.241
Hematochezia	2 (3.8)	5 (10.2)	7 (6.9)	0.209
Complete Rockall score	6.84 ± 1.47	6.87 ± 1.31	6.86 ± 1.39	0.911
Rockall score > 6	26 (50.0)	29 (59.2)	55 (54.5)	0.354
Early rebleeding	6 (12.2)	7 (14.3)	13 (13.3)	0.766

NSAID: Non-steroidal anti-inflammatory drug.

Enrolled patients were assigned to one of two groups. After the initial endoscopy, both groups received an 80-mg bolus of pantoprazole, followed by continuous intravenous (IV) infusion at 8 mg/h for a total of 72 h. The pantoprazole alone group received only pantoprazole for 72 h. The somatostatin adjunctive group, in addition to the pantoprazole for 72 h, received a 250-μg bolus of somatostatin, followed by continuous IV infusion of 250 μg/h for a total of 72 h. No other anti-ulcer medication was administered. At 48 h after initial endoscopy, repeat endoscopy was performed to investigate the presence of hemorrhagic stigma. When a remnant stigma was observed, an additional endoscopic procedure was performed, if deemed necessary clinically. After the 72-h infusion, patients were given one of the following, orally, each day, for 8 wk: 40 mg pantoprazole; 20 mg rabeprazole; 30 mg lansoprazole; or 40 mg esomeprazole.

Outcomes and measurement

The primary end point was the rate of clinically significant early rebleeding, as defined below. Secondary outcomes were the loss of endoscopic high-risk stigma on subsequent endoscopy and the associated risk factors for early rebleeding.

Definition

Hemodynamically, shock was defined as systolic pressure < 90 mm Hg or heart rate > 110 bpm. Stigma of recent hemorrhage or high-risk ulcer stigma was defined as spurting (Forrest classification I a), oozing (Forrest classification I b) and visible vessel (Forrest classification Ⅱ a)[24]. Rebleeding was defined as: (1) fresh hematemesis or fresh blood in the nasogastric tube; (2) passage of fresh melena or hematochezia with additional evidence of recurrent bleeding (a drop in hemoglobin of ⩾ 2 g/dL within 24 h after endoscopy); and (3) bleeding observed by endoscopy[25]. Early rebleeding was defined as rebleeding within 7 d of the endoscopic interventions.

Statistical analysis

Univariate analyses were performed with a χ^2 test or Fisher's exact test for categorical variables and Student's *t* test for continuous variables. Variables with $P < 0.25$ in the univariate analysis were included in a multiple logistic regression model to identify independent risk factors for early rebleeding. $P < 0.05$ indicated statistical significance. Statistical calculations were performed with SPSS for Windows version 12.0 (SPSS Inc., Chicago, IL, USA).

RESULTS

Among 205 patients who were admitted due to NVUGIB episodes, 104 were excludes as follows: endoscopic hemostasis not achieved successfully (*n* = 15); bleeding from gastrointestinal cancer (*n* = 15); and no high-risk bleeding stigma (*n* = 74). Finally, a total of 101 patients with NVUGIB were enrolled.

The treatment groups were not significantly different in the clinical characteristics including Rockall scores (Table 1). The mean patient age (SD) was 64.86 ± 17.01 years and 31.7% (32/101) were male. The mean complete Rockall score (SD) was 6.86 ± 1.39 (Table 1). Thirteen patients (13.3%) experienced rebleeding within 7 d after endoscopic intervention. Between treatment

Table 2 Endoscopic findings between treatment groups *n* (%)

	Pantoprazole group (*n* = 52)	Somatostatin group (*n* = 49)	Total cohort (*n* = 101)	*P* value
Cause of bleeding				0.177
Gastric ulcer	27 (51.9)	29 (59.2)	56 (55.4)	
Duodenal ulcer	11 (21.2)	8 (16.3)	19 (18.8)	
Dieulafoy lesion	13 (25.0)	7 (14.3)	20 (19.8)	
Mallory-Weiss syndrome	1 (1.9)	5 (10.2)	6 (5.9)	
Forrest type				0.894
Ⅰa	3 (5.8)	4 (8.2)	7 (6.9)	
Ⅰb	24 (46.2)	22 (44.9)	46 (45.5)	
Ⅱa	25 (48.1)	23 (46.9)	48 (47.5)	
Loss of stigma	49 (94.2)	47 (95.9)	96 (95.0)	0.696

Table 3 Rebleeding according to endoscopic findings *n* (%)

	No rebleeding (*n* = 88)	Rebleeding (*n* = 13)	Total (*n* = 101)	*P* value
Forrest type				0.990
Ⅰa	6 (6.8)	1 (7.7)	7 (6.9)	
Ⅰb	40 (45.5)	6 (46.2)	46 (45.5)	
Ⅱa	42 (47.7)	6 (46.2)	48 (47.5)	
Loss of stigma	84 (95.5)	12 (92.3)	96 (95.0)	0.625

groups, the rebleeding rates were not significantly different ($P = 0.766$) (Table 1). A second endoscopic intervention was successful in most patients that experienced rebleeding (11/13, 84.6%). Two cases received an angiographic embolization because endoscopic intervention failed to stop bleeding. There was no bleeding-related death during the study period. The most common cause of NVUGIB was gastric ulcer (55.4%, 56/101) (Table 2). The treatment groups were not significantly different for endoscopic Forrest classification ($P = 0.894$) and loss of endoscopic high-risk stigma ($P = 0.696$) (Table 2). The early rebleeding rate according to endoscopic Forrest classification was not significantly different ($P = 0.990$) (Table 3).

For risk factor analysis for early rebleeding, univariate analysis showed that complete Rockall score > 6 was a significant indicator ($P = 0.003$) of early rebleeding (Table 4). Multivariate analysis showed that only the complete Rockall score was significantly associated with early rebleeding ($P = 0.044$, OR: 9.080, 95% CI: 1.062-77.595) (Table 5).

There were no serious adverse events related to the drugs used in this study, and no serious drug interactions were noted between pantoprazole and somatostatin during the infusion period.

DISCUSSION

NVUGIB is a serious medical disorder. Although endoscopic therapy is a highly effective treatment method, successful endoscopic treatment is largely dependent upon the expertise of the endoscopist[26,27]. After endoscopic hemostasis, the use of a PPI has become the standard of care in patients with NVUGIB[8-11]. However, a recent study has shown that a high-dose, continuous infusion of PPIs may not be sufficient to sustain an intragastric pH $\geqslant$ 6[28]. Somatostatin has been used in variceal bleeding[12], and it has been suggested to be more effective than pantoprazole in maintaining high gastric pH during the first 12 h of infusion[21]. If endoscopy is contraindicated or unavailable, somatostatin might be a reasonable alternative solution. In clinical practice, patients likely to have bleeding might be considered for somatostatin treatment before definitive endoscopy[29].

From a theoretical point of view, somatostatin has the advantage of reducing gastroduodenal blood flow and pepsin secretion, in addition to inhibiting gastric acid secretion[14,17,21]. These effects may be of value for patients with NVUGIB; particularly in patients with high-risk endoscopic findings. Therefore, we hypothesized that adjunctive use of somatostatin with pantoprazole could prove effective in reducing early rebleeding in patients treated for NVUGIB. The present study focused on the effects of infusing somatostatin as an adjunct to pantoprazole after a successful endoscopic procedure in patients with endoscopic high-risk stigma. Although this was not a randomized study, the clinical baseline characteristics were not significantly different between the treatment groups, including hemodynamic shock, endoscopic findings and Rockall scores (Tables 1 and 2). The results showed that the adjunctive use of somatostatin was not superior to pantoprazole infusion alone in preventing rebleeding ($P = 0.766$) (Table 1). We enrolled patients with endoscopic high-risk stigma who were treated with endoscopy; 48 h after initial endoscopy, a second endoscopy was performed to confirm the absence of the hemorrhagic stigma. The result was not significantly different between treatment groups ($P = 0.696$) (Table 3). A previous meta-analysis[30] has shown that all endoscopic therapies (including clips and thermal therapy) reduce the risk of rebleeding compared with pharmacotherapy alone. The present study enrolled patients in whom therapeutic interventions were successfully performed at initial endoscopy, therefore, it is not surprising that differences in endoscopic findings were not identified as important risk factors for rebleeding. When a high-risk hemorrhagic stigma could be eradicated by endoscopic intervention, gastric acid inhibition with a high dose of PPI alone appeared to be sufficient for maintaining hemostasis. Among patients that experienced rebleeding, two required angiographic embolization because endoscopic intervention was unsuccessful. Only a small number of cases required additional angiographic embolization, therefore, statistical analysis was limited.

Optimal acid suppression facilitates clot formation over arteries in bleeding peptic ulcers. A previous study has reported that infusion of high-dose omeprazole before endoscopy accelerated the resolution of signs of bleeding in ulcers and reduces the need for endoscopic therapy[31]. If infusion of high-dose omeprazole after hemostasis had been administered, the rates of recurrent

Table 4 Clinical characteristics according to occurrence of early rebleeding event (mean ± SD) *n* (%)

	No rebleeding (*n* = 88)	Rebleeding (*n* = 13)	Total (*n* = 101)	*P* value
Sex (male)	27 (30.7)	5 (38.5)	32 (31.7)	0.574
Age (yr)	64.25 ± 17.40	69.0 ± 13.97	64.86 ± 17.01	0.350
Hemodynamic shock	46 (52.3)	7 (53.8)	53 (52.5)	0.916
Helicobacter infection	19 (21.6)	3 (23.1)	22 (21.8)	0.904
Hemoglobin (g/dL)	8.61 ± 2.77	7.09 ± 2.02	8.41 ± 2.72	0.060
Hemoglobin < 7 g/dL	26 (29.5)	7 (53.8)	33 (32.7)	0.081
Blood urea nitrogen (mg/dL)	39.81 ± 25.92	40.09 ± 33.50	39.84 ± 26.82	0.972
Creatinine (mg/dL)	1.26 ± 1.12	1.03 ± 0.83	1.23 ± 1.09	0.473
Albumin (g/dL)	2.97 ± 0.60	2.86 ± 0.47	2.96 ± 0.59	0.532
Type 2 diabetes mellitus	21 (23.9)	3 (23.1)	24 (23.8)	0.950
Hypertension	38 (43.2)	3 (23.1)	41 (40.6)	0.168
Heart failure	9 (10.2)	2 (15.4)	11 (10.9)	0.577
Ischemic heart disease	17 (19.3)	1 (7.7)	18 (17.8)	0.454
Antiplatelet medication	37 (42.0)	7 (53.8)	44 (43.6)	0.423
NSAID	7 (8.0)	2 (15.4)	9 (8.9)	0.380
Multiple antiplatelet medications	5 (5.7)	2 (15.4)	7 (6.9)	0.221
Steroid	5 (5.7)	1 (7.7)	6 (5.9)	0.572
Melena	50 (56.8)	9 (69.2)	59 (58.4)	0.397
Hematemesis	51 (58.0)	4 (30.8)	55 (54.5)	0.066
Hematochezia	5 (5.7)	2 (15.4)	7 (6.9)	0.199
Complete Rockall score	6.73 ± 1.40	7.69 ± 1.03	6.86 ± 1.39	0.020
Rockall score > 6	43 (48.9)	12 (92.3)	55 (54.5)	0.003
Somatostatin use	41 (46.6)	8 (61.5)	49 (48.5)	0.314

NSAID: Non-steroidal anti-inflammatory drug.

Table 5 Predictors of early rebleeding on multivariate analysis

	P value	Exp (B)	95% CI
Adjunct somatostatin use	0.374	0.527	0.128-2.164
Hypertension	0.175	2.864	0.627-13.086
Multiple antiplatelet medication	0.421	2.351	0.239-18.879
Hemoglobin < 7 g/dL	0.402	1.768	0.466-6.704
Hematemesis	0.072	3.672	0.889-15.179
Hematochezia	0.614	0.593	0.078-4.517
Complete Rockall score > 6	0.044	9.080	1.062-77.595

bleeding did not differ between the groups[31]. In high-risk patients, early endoscopy involving therapy stops bleeding and potentially saves lives. Early endoscopy also permits low-risk patients to be discharged early from hospital. The use of high-dose PPIs cannot replace the need for early endoscopy. Our study group had stigmata of recent hemorrhage and most of the endoscopy was performed within 4 h. The effect of preemptive PPI on rebleeding might be negligible. It may be different between arterial and venous bleeding (such as varix or telangiectasia). In this study, most of the lesions were arterial bleeding. Variceal bleeding was excluded and no telangiectatic lesions were included.

In a large epidemiological study, increased risk of gastrointestinal bleeding was significantly associated with low-dose aspirin use (< 100 mg/d)[32]. In the present study, 43.6% (44/101) of patients were using antiplatelet agents (including aspirin). However, we found that the risk of early rebleeding was not significantly associated with antiplatelet agents (P = 0.423) (Table 4). The risk of gastrointestinal bleeding due to antiplatelet drugs persists as long as therapy continues, but declines within 7 d of withdrawal; a time comparable to the life of the platelet[32]. Thus, although the use of antiplatelet drugs is a principal risk factor for gastrointestinal bleeding, the risk of rebleeding might be associated with the time after antiplatelet withdrawal.

Several scoring systems have been developed to assess the risk of recurrent bleeding and death in patients with upper gastrointestinal bleeding. Although endoscopic findings may identify individuals who are at high risk for rebleeding, other factors such as age and comorbidity may affect overall mortality. Of the scoring systems that include endoscopic findings, the Rockall scoring system[23] is most commonly used. The Rockall scoring system takes into account age, presence of shock, comorbidity, source of bleeding, and major stigmata from recent hemorrhage. We found that the complete Rockall score was a significant predictor of early rebleeding (P = 0.044, OR: 9.080, 95% CI: 1.062-77.595).

There were some limitations to the current study. Although the study data were collected prospectively, it was not a randomized study, and the doctor responsible for ordering medication was not blinded to the patient's condition. Although the mean Rockall score, an extensively validated measure of the risk for morbidity, was not significantly different between the two groups, it may not have been sufficiently comprehensive. It is possible that somatostatin treatment was associated with other, unmeasured clinical and demographic variable, and these may have confounded our results. The rebleeding rate might have been affected (somatostatin group was higher than control group, 14.3% *vs* 12.2%, respectively, P = 0.766).

Moreover, we did not measure intragastric pH; therefore, we could not precisely determine the efficacy of adjunctive somatostatin for maintaining intragastric pH. Finally, because all enrolled patients were treated with endoscopy and therapeutic interventions, a definitive comparison between the medications might not have been possible.

In conclusion, we believe that this is the first study to focus on the adjunctive effect of somatostatin with PPI in acute NVUGIB patients with high-risk endoscopic lesions. Adjunctive somatostatin for management of NVUGIB did not show an additive effect in reducing early rebleeding. Complete Rockall score can predict early rebleeding for patients who have high-risk endoscopic stigma after successful endoscopic management.

COMMENTS

Background

Proton pump inhibitors (PPIs) and somatostatin are suggested to be effective treatments for non-variceal upper gastrointestinal bleeding (NVUGIB). However, the clinical effect of a PPI with a somatostatin adjunct has not been established. We hypothesized that the use of somatostatin as an adjunct to pantoprazole may potentiate hemostasis in patients at high risk for rebleeding.

Research frontiers

NVUGIB is a serious medical disorder. After endoscopic hemostasis, the use of a PPI has become the standard of care in patients with NVUGIB. However, a recent study has shown that high-dose, continuous infusion of PPIs may not be sufficient to sustain an intragastric pH $\geqslant$ 6. Somatostatin has been suggested to be more effective than pantoprazole in maintaining high gastric pH during the first 12 h of infusion. From a theoretical point of view, somatostatin has the advantage of reducing gastroduodenal blood flow and pepsin secretion in addition to inhibiting gastric acid secretion. These effects may be of value for patients with NVUGIB, particularly in patients with high-risk endoscopic findings.

Innovations and breakthroughs

Adjunctive use of somatostatin was not superior to pantoprazole monotherapy after successful endoscopic hemostasis in patients with NVUGIB.

Applications

Adjunctive use of somatostatin was not superior to pantoprazole monotherapy after successful endoscopic hemostasis in patients with NVUGIB. Complete Rockall score can predict early rebleeding for patients who have high-risk endoscopic stigmata after successful endoscopic management.

Terminology

NVUGIB means bleeding from non-variceal origins such as peptic ulcer, Dieulafoy lesion and Mallory-Weiss syndrome. High-risk ulcer stigma is defined as spurting (Forrest classification Ⅰa), oozing (Forrest classification Ⅰb) and visible vessels (Forrest classification Ⅱa).

Peer review

Choi *et al* have performed a study to establish the effect of adjunctive somatostatin for prevention of NVUGIB after endoscopic therapy. This paper is interesting and it could be valuable for other researchers.

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