의학석사 학위논문

Bedside endoscopy for gastrointestinal bleeding in critically ill patients

중환자실 환자에서 발생한

위장관 출혈에서 병상 내시경의 유용성

2016 년 2 월

서울대학교 대학원

의학과 내과학 전공

김 지 현

A thesis of the Master's degree

중환자실 환자에서 발생한

위장관 출혈에서 병상 내시경의 유용성

Bedside endoscopy for gastrointestinal bleeding in critically ill patients

February 2016

Seoul National University

College of Medicine

Internal Medicine

Jee Hyun Kim

Abstract

Bedside endoscopy for gastrointestinal bleeding in critically ill patients

Jee Hyun Kim

Department of Internal Medicine

Seoul National University College of Medicine

Background/Aim: Gastrointestinal (GI) bleeding is an important complication in critically ill patients. The aim of this study was to determine the efficacy of bedside endoscopy in ICU setting, especially to compare the outcomes of early endoscopy (within 24 h of GI bleeding) with late endoscopy (after 24 h of GI bleeding) in the intensive care unit (ICU) setting.

Methods: We retrospectively reviewed the medical records of patients who underwent bedside endoscopy for non-variceal GI bleeding following ICU admission at Seoul National University Hospital from January 2010 and May 2015.

Results: 253 patients underwent bedside esophagogastroduodenoscopy (EGD) for upper GI bleeding (early, 187; late, 66) and 69 bedside colonoscopy (CS) for lower GI bleeding (early, 36; late, 33). Common endoscopic findings were peptic ulcer (34%), and acute gastric mucosal lesion (17%) in EGD group, and ischemic colitis (24%) and acute hemorrhagic rectal ulcers (20%) in CS group. Early EGD significantly increased the rate of finding bleeding focus (82% vs. 73%, p = 0.003) and endoscopic hemostasis (60% vs. 8%, p = 0.002) as compared with late EGD. However, early CS significantly decreased the rate of identifying bleeding focus (58% vs. 82%,

p = 0.008) and hemostasis (7% vs. 16%, p = 0.011) because of its higher rate of poor bowel preparation and blood interference as compared with late CS. Risk factors of upper GI rebleeding were antiplatelets or anticoagulants therapy, coagulopathy, high level of BUN, and high blood transfusion requirements. In case of lower GI bleeding, coagulopathy was only significant factor associated with rebleeding.

Conclusions: Early bedside EGD is effective for diagnosis and endoscopic treatment in the ICU patients with GI bleeding. CS should be carefully performed after adequate bowel preparation.

Keywords: Bedside endoscopy; Gastrointestinal bleeding; Early endoscopy

Student Number: 2014-21108

List of Tables

Contents

Introduction

Critically ill patients often develop upper and lower gastrointestinal bleeding (UGIB; LGIB). Gastrointestinal (GI) bleeding is an important complication that causes increased morbidity and mortality in critically ill patients. Esophagogastroduodenoscopy (EGD) has been reported to play a significant role in the diagnosis and treatment of patients with UGIB. In particular, early endoscopy (within 24 h of GI bleeding) has been shown to improve clinical outcomes in both non-intensive care unit (ICU) and ICU.[1] Colonoscopy (CS) also has been shown to be valuable in diagnosing and managing LGIB patients in non-ICU setting.[2]

Various prognostic factors have been proposed to explain the poor outcome, such as older age, number of comorbid illnesses, recurrent bleeding, severe hemorrhage, shock, high risk endoscopic stigmata, and in-hospital bleeding.[3-5] Managing with GI bleeding in ICU patients remains difficult since most patients have multiple and complex poor prognostic features. Furthermore, limited data are available concerning the endoscopic findings and effectiveness of endoscopy therapy in reducing mortality in severely ill patients with GI bleeding that occurs during ICU stay.

Therefore we conducted this retrospective study to determine the clinical characteristics of GI bleeding and efficacy of bedside endoscopy for GI bleeding in ICU setting. Furthermore, impact of early endoscopy on clinical outcomes and the risk factors of recurrent GI bleeding were evaluated.

Materials and Methods

Patients

We retrospectively reviewed the medical records of patients undergoing bedside endoscopy for nonvariceal GI bleeding that developed while in their ICU stay at Seoul National University Hospital from January 2010 and May 2015.

In this study, GI bleeding was diagnosed when there were overt bleeding or clinically important bleeding. Clinically significant GI bleeding was diagnosed as overt bleeding (hematemesis, bloody nasogastric drainage, melena or hematochezia) complicated by one of the following features, in the absence of other causes: a) a spontaneous decrease in systolic or diastolic blood pressure of more than or equal to 20 mm Hg within 24 hours of upper gastrointestinal bleeding; b) an increase in pulse rate of 20 beats/min and a decrease in systolic blood pressure of 10 mmHg on orthostatic change; c) a decreased hemoglobin level of more than or equal to 2 g/dl (20 g/L) in 24 hours and transfusion of 2 units of packed red blood cells within 24 hours of bleeding; or d) failure of the hemoglobin level to increase by at least the number of units transfused minus 2 g/dl (20 g/L).[6]

Demographic data included patient characteristics (age, gender), admission diagnosis, APACHE II (Acute Physiology and Chronic Health Evaluation II) score, presence of multiple organ dysfunction, use of mechanical ventilation (MV), duration of ventilation, presence of shock (systolic blood pressure <90 mmHg and peripheral circulatory failure), in-hospital medical therapy (nonsteroidal anti-inflammatory drugs (NSAIDs), antiplatelets, anticoagulants or corticosteroids), stress ulcer prophylaxis (including use of histamine 2 receptor antagonists, proton-pump inhibitors, and sucralfate), enteral nutrition, mental status based on the Glasgow Coma Scale (GCS), decrease in hemoglobin (Hb), coagulopathy, and other laboratory data such

as Hb level, blood urea nitrogen (BUN), creatinine (Cr), and liver function (AST/ALT) when GI bleeding occurred. Coagulopathy was defined as a platelet count <50,000/mm3 or an international normalized ratio >1.5.

Endoscopic characteristics included early endoscopy, length between admission to the hospital and performance of endoscopy, operator of endoscopy (experts or fellows), identification of bleeding focus, endoscopic diagnosis, primary endoscopic hemostasis, mode of endoscopic hemostasis, and result of hemostasis. Early endoscopy was defined as endoscopy performed within 24 hours of onset of GI bleeding. Endoscopic hemostasis therapy included the use of hemoclipping, ligation, therapy with epinephrine, and the use of argon plasma coagulation (APC), or heater probe therapy for hemostasis. If a patient underwent multiple EGDs, only the initial EGD for GI bleeding was used in the analysis.

Outcome variables included primary hemostasis rate, recurrent bleeding rate, rates of second endoscopy, angiography and surgery, length of hospitalization, length of ICU stay, in-hospital mortality, and the cause of death. Recurrent bleeding was defined as fresh hematemesis or hematochezia, instability of vital signs, bloody nasogastric drainage, and reduction in hemoglobin level in excess of 2 g/dl within 24 h after successful primary hemostasis.[7]

Statistical analysis

Quantitative data were summarized as mean ±standard deviation (SD) and categorical variables as number (%). The parameters were compared in two groups of patients using the Student's t test for continuous variables and the $\chi 2$ test for dichotomous variables. Furthermore, the impact of early endoscopy on clinical outcome of patients with GI bleeding and the risk factors for recurrent GI bleeding were examined by using multiple logistic regression analysis. The relative risk and 95% confidence interval of the significant factors were calculated. A P value<0.05 was considered

statistically significant. Statistical analyses were performed using SPSS 18.0 software (SPSS 18.0 version for Windows, SPSS, Chicago, Ill., USA).

Results

Characteristics of patients with GI bleeding that developed during ICU stay

A total of 314 ICU patients underwent bedside endoscopy for suspicious GI bleeding that developed while staying in the ICU at Seoul National University Hospital from January 1, 2010 to May 31, 2015. Of these, 253 patients received bedside EGD, and 69 patients received bedside CS for nonvariceal GI bleeding that occurred after admission to the ICU. Their characteristics are listed in Table 1.

In UGIB, the mean age was 68.8 ± 12.4 years and 63.2% of the patients were men. The average APACHE II score was 27.4, and GCS score was 10.8. The most common reasons for admission to the ICU was respiratory disease in 115 (45.5%). UGIB occurred a mean (±SD) of 17.9 (±25.6) days after admission to the hospital and 8.7 (±13.6) days after admission to the ICU. In 22 patients, GI bleeding resulted in transfer from a regular ward bed to an ICU. The mean days from admission to the hospital and occurrence of GI bleeding was shorter in the patients admitted to the ICU for GI bleeding than those whose bleeding developed after admission to the ICU for other reasons (6.7 vs.17.9 days; $p = 0.045$). Admission to the ICU for GI bleeding was related with a presence of shock, lower serum Hb, increased rate of angiography, and higher units of RBC transfused. Furthermore, the length of hospital stay and duration of ICU stay were shorter in patients admitted to the ICU for bleeding than in those patients admitted to the ICU for other causes. (mean, 25.3 vs. 53.4 days; $p < 0.001$, 12.8 vs. 24.2 days; $p < 0.001$, respectively). In the multivariate analysis, admission to the ICU for UGIB was an independent factor related with an increase in RBC transfusion, a shorter duration of hospitalization, and a shorter length of ICU stay. Bloody nasogastric tube drainage, hematemesis, and melena/hematochezia present in 109 (43.1%), 26 (10.3%) and 80 (31.6%) patients, were common indications for EGD. When GI bleeding

developed in the ICU, 231 patients (91.5%) had required MV for at least 24 hours, 155 (61.3%) had multiple organ dysfunction, 99 (39.1%) were in shock state, 226 (89.3%) received stress ulcer prophylaxis, and 139 (54.9%) received enteral feeding. Anemia (93.3%) and coagulopathy (40.3%) were common laboratory finding. In-hospital medical therapy in 253 patients with UGIB included NSAIDs in 13 (5.1%), antiplatelets in 73 (28.9%), anticoagulants in 60 (23.7%), and corticosteroids in 49 (19.4%).

The characteristics of the patients with LGIB were similar to those of the patients with UGIB regarding age, gender, APHACHE II score, use of MV, presence of multiple organ failure or shock, medication, and laboratory findings. Hematochezia/melena (89.9%) was the most common indication for CS. LGIB occurred a mean (±SD) of 24.6 (±24.7) days after admission to the hospital and 12.7 (±11.7) days after admission to the ICU. These durations were longer in LGIB than those in UGIB.

Endoscopic findings and outcomes of bedside endoscopy

Bedside EGD was performed a mean of 19.1 days after admission to the hospital and 9.8 days after admission to the ICU. On the other hand, CS was performed a mean of days 26.9 days after admission to the hospital and 15 days after admission to the ICU.

In UGIB, endoscopic findings were peptic ulcer, the most common bleeding source identified, in 88 (34.8%), acute gastric mucosal lesion (AGML) in 43 (17%), esophagitis in 32 (12.6%), angiodysplasia in 10 (4%), malignancy in 9 (3.6%), Dieulafoy's lesion in 6 (2.4%), Mallory-Weiss tear in 3(12%), others in 10 (4%), and bleeding focus could not be identified due to large hematoma in 8 (3.2%) patients. Stigmata of bleeding were observed in 77 (30.4%) patients with spurting artery of 9 (3.6%), oozing vessel of 43 (17%), and visible nonbleeding vessel of 25 (9.9%) (Table 2).

In LGIB, ischemic colitis in 16 (24.2%), acute hemorrhagic rectal ulcers in 14 (20.3%), pseudomembranous colitis in 5 (7.2%), and malignancy in 5 (7.2%) were common endoscopic diagnoses. Identifying bleeding site using CS failed in 13 (17.4%) patients due to poor bowel preparation (8.7%), blood interference (7.2%). In 2 (2.9%) patients, bleeding sources were thought to originate from the small bowel because fresh blood was still noted above the accessible area. Finally, among 314 patients with GI bleeding, 15 (4.8%) diagnosed as small bowel bleeding by angiography and surgery (Table 3).

Outcomes are shown in Table 2 (UGIB) and Table 3 (LGIB). In patients with UGIB, 68 (26.9%) received primary endoscopic hemostasis. Endoscopic hemostasis was usually attempted in patients with active bleeding, nonbleeding visible vessels, and visible vessels after clot removal. Among these patients, 45 (66.2%) patients underwent endoscopic hemostasis with argon plasma coagulation, 9 (13.2%) with hemoclipping, 9 (13.2%) with band ligation, and 1 (1.5%) with diluted epinephrine spray. Among 68 patients who received primary hemostasis, hemostasis was successful in 62 (91.2%). Recurrent GI bleeding occurred in 55 (21.7%) patients. Second endoscopy was performed in 48 (19%) patients to reidentify the source of bleeding or provide hemostasis. 37 (14.6%) patients received angiography, and 3 (1.2%) underwent surgery. Mean units of RBC transfused was 6.0 ± 5.3. Days of hospital stay was 53.4 ± 51.2, and days of ICU stay was 24.2 ± 29.4. The in-hospital mortality was 44.7%, but UGIB related death occurred in only 4 (1.6%) patients.

In LGIB, primary endoscopic hemostasis rate (33.3%), its success rate (91.3%), and recurrent bleeding rate (26.1%) were similar to those in UGIB. Mean duration of hospital stay was 79.1 ± 62.5, and length of ICU stay was 38.9 ± 44.9. The length of hospital stay and ICU stay were longer in the patients with LGIB than those with UGIB. In-hospital mortality was 58%, but only 2 (5%) experienced in-hospital mortality related to LGIB, and there was no complication related to CS.

Associations of early endoscopy with the outcomes

Early endoscopies were performed in 187 (73.9%) patients with UGIB and 36 (52.2%) patients with LGIB. Comparisons of outcomes between patients with and without early endoscopy are listed in Table 2 and Table 3.

Early EGD was significantly associated with a higher rate of identifying bleeding focus, higher rate of endoscopic hemostasis, and lower units of RBC transfused. However no significant differences were found between patients who underwent early endoscopy and those who did not, regarding length of hospital or ICU stay, rate of recurrent bleeding, rate of second endoscopy, angiography or surgery, and mortality rate. Using these valid factors in the univariate analysis, clinical outcomes associated with early endoscopy were evaluated by multivariate analysis. Although length of ICU stay was not related with early endoscopy in the univariate analysis, we included it in multivariate analysis because early endoscopy reduced the length of hospital stay and length of ICU stay in the previous other studies.[8] In the multivariate analysis, early EGD showed better outcomes, such as a higher diagnostic rate, higher endoscopic hemostasis rate, and a decrease in RBC transfusion (Table 2). On the other hand, early CS was identified as an independent factor associated with a higher diagnostic rate, higher endoscopic hemostasis rate, and a reduction in units of RBC required (Table 3).

The risk factors for recurrent GI bleeding

The risk factors for recurrent GI bleeding are shown in Table 4 and Table 5. In univariate analysis, recurrent UGIB was associated with a lower GCS score, shorter duration between admission to hospital and occurrence of GI bleeding, presence of coagulopathy, medication with antiplatelets, anticoagulants or steroids, lower Hb level, higher BUN level, and higher units of RBC transfused.

Furthermore, in multivariate analysis, independent risk factors for recurrent UGIB were a shorter length between admission to the hospital and occurrence of GI bleeding, presence of coagulopathy, medication with antiplatelets or anticoagulants, higher level of serum BUN, and a higher RBC transfusion requirement during the first GI bleeding event. However, in LGIB, only coagulopathy was the risk factor of recurrent bleeding. In both UGIB and LGIB groups, there was no association between recurrent GI bleeding and a use of MV, serum Cr level, AST/ALT level, and an operator of endoscopy. A need of surgery, mortality rate, duration of hospitalization, and a length of ICU stay were not significantly different between the patients with rebleeding and those without.

Table 1. Characteristics of patients who received bedside endoscopy for GI bleeding that developed after admission to the ICU

	EGD (N=253)	CS (N=69)
Age (years)	68.8 ± 12.4	66.7 ± 11.8
Male gender	160 (63.2)	39 (56.5)
Reasons for ICU admission		
Respiratory disease	115 (45.5)	32 (46.4)
Sepsis	26 (10.3)	2 (2.9)
Major surgery	31 (12.3)	12 (17.4)
Cardiovascular disease	38 (15)	13 (18.8)
CNS disease	19 (7.5)	3 (4.3)
Hepatic failure	5 (2.0)	0 (0)
Acute renal failure	15 (5.9)	7 (10.1)
Others	4 (1.6)	0 (0)
Days between admission to the ICU and occurrence of GI bleeding	8.7 ± 14.3	12.7 ± 11.7
MV	231 (91.5)	63 (91.3)
Days of MV (days)	9.8 ± 15.0	13.4 ± 11.9
APACHE II score	27.4 ± 9.3	25.2 ± 9.2
GCS score	10.8 ± 4.0	11.8 ± 3.3
Multiple organ dysfunction	155 (61.3)	43 (62.3)
Presence of shock	99 (39.1)	29 (42.0)
Medication		
NSAIDs	13 (5.1)	2 (2.9)
Antiplatelets	73 (28.9)	13 (18.8)
Anticoagulants	60 (23.7)	22 (31.9)
Corticosteroids	49 (19.4)	25 (36.2)
Stress ulcer prophylaxis	226 (89.3)	66 (95.7)
Enteral nutrition	139 (54.9)	43 (62.3)
Anemia	236 (93.3)	64 (92.8)
Coagulopathy	102 (40.3)	27 (39.1)

Lab		
Hb (g/dL)	8.8 ± 2.2	9.1 ± 2.1
AST (IU/L)	313.9 ± 1056.2	109.9 ± 290.6
ALT (IU/L)	426.7 ± 2828.6	66.9 ± 167.2
BUN (mg/dL)	49.4 ± 31.6	46.8 ± 29.9
Cr (mg/dL)	2.3 ± 2.8	2.6 ± 4.6
Initial presentations		
Bloody NG drainage	109 (43.1)	0 (0)
Hematemesis	26 (10.3)	1 (1.4)
Hematochezia/Melena	80 (31.6)	62 (89.9)
Decreased in Hb (g/dl)	38 (15)	6 (8.7)
Early endoscopy	187 (73.9)	36 (52.2)

Values are presented as mean ± SD or number (%).

EGD, esophagogastroduodenoscopy; CS, colonoscopy; MV, mechanical ventilation; GCS, Glasgow Coma Scale; NG, nasogastric.

Table 2. Endoscopic findings and outcomes of bedside esophagogastroduodenoscopy

	EGD			
	Early (N=187)	Late (N=66)	P-value	Total (N=253)
Identification of bleeding focus	153 (81.8)	48 (72.7)	**0.003**	201 (79.4)
Operator of endoscopy (Expert)	25 (13.4)	14 (21.2)	0.129	39 (15.4)
Endoscopic diagnosis			**0.002**	
Ulcer with profusely bleeding stigma	42 (22.5)	4 (6.1)		46 (18.2)
Ulcer with clean base or firmly adherent blood clot	30 (16.0)	12 (18.2)		42 (16.6)
Mallory-Weiss syndrome	3 (1.6)	0 (0)		3 (1.2)
Dieulafoy's lesion	5 (2.7)	1 (1.5)		6 (2.4)
Angiodysplasia	9 (4.8)	1 (1.5)		10 (4.0)

Esophagitis	19 (10.2)	13 (19.7)		32 (12.6)
AGML	32 (17.1)	11 (16.7)		43 (17.0)
Malignancy	7 (3.7)	2 (3.0)		9 (3.6)
Others	6 (3.2)	4 (6.0)		10 (4.0)
Limited study*	7 (3.7)	1 (1.5)		8 (3.2)
Type of bleeding stigmata			0.464	77
Spurting artery	8 (12.1)	1 (9.1)		9 (3.6)
Oozing vessel	35 (53.0)	8 (72.7)		43 (17.0)
Visible non-bleeding vessel	23 (34.8)	2 (18.2)		25 (9.9)
Primary endoscopic hemostasis	60 (32.1)	8 (12.1)	**0.002**	68 (26.9)
Mode of endoscopic hemostasis			0.646	
APC	38 (63.3)	7 (87.5)		45 (66.2)
Ligation	8 (13.3)	1 (12.5)		9 (13.2)
Clipping	9 (15.0)	0 (0)		9 (13.2)
Epinephrine spray	1 (1.7)	0 (0)		1 (1.5)
Others	4 (6.7)	0 (0)		4 (5.9)
Recurrent bleeding	38 (20.3)	17 (25.6)	0.341	55 (21.7)
Second endoscopy	38 (20.3)	10 (15.2)	0.357	48 (19.0)
Angiography	27 (14.4)	10 (15.2)	0.888	37 (14.6)
Surgery	3 (1.6)	0 (0)	0.570	3 (1.2)
Units of RBC transfused	5.6 ± 4.9	7.3 ± 6.3	**0.027**	6.0 ± 5.3
Days of hospital stay (days)	52.0 ± 48.3	57.4 ± 58.8	0.463	53.4 ± 51.2
Days of ICU stay (days)	23.3 ± 27.4	26.7 ± 34.5	0.424	24.2 ± 29.4
Hospital mortality	87 (46.5)	26 (39.4)	0.316	113 (44.7)
Causes of death			0.538	
Underlying disease	70 (37.4)	22 (33.3)		92 (36.4)
GI bleeding	4 (2.1)	0 (0)		4 (1.6)
Others	13 (7.0)	4 (6.1)		17 (6.7)

Values are presented as mean ± SD or number (%).

EGD, esophagogastroduodenoscopy; AGML, acute gastric mucosal lesion; APC, argon plasma coagulation;

* Limited study means that bleeding focus could not be identified due to large hematoma.

Table 3. Endoscopic findings and outcomes of bedside colonoscopy

	CS			
	Early (N=36)	Late (N=33)	P-value	Total (N=69)
Identification of bleeding focus	21 (58.3)	27 (81.8)	**0.008**	48 (69.4)
Operator of endoscopy (Expert)	7 (19.4)	12 (36.4)	0.116	19 (27.5)
Endoscopic diagnosis			0.161	
Ischemic colitis	8 (22.2)	8 (24.2)		16 (24.2)
Acute hemorrhagic rectal ulcers	4 (11.1)	10 (30.3)		14 (20.3)
PMC	2 (5.6)	3 (9.1)		5 (7.2)
Malignancy	3 (8.3)	2 (6.1)		5 (7.2)
CMV colitis	2 (5.6)	1 (3.0)		3 (4.3)
Dieulafoy's lesion	0 (0)	2 (6.1)		2 (2.9)
Nonspecific colitis	1 (2.8)	0 (0)		1 (1.4)
Others	1 (2.8)	0 (0)		1 (1.4)
Limited study	8 (22.2)	4 (12.1)		13 (17.4)
Primary endoscopic hemostasis	7 (19.4)	16 (48.5)	**0.011**	23 (33.3)
Mode of endoscopic hemostasis			0.710	
APC	5 (71.4)	8 (50.0)		13 (56.5)
Ligation	0 (0)	1 (6.3)		1 (4.3)
Clipping	2 (28.6)	6 (37.5)		8 (34.8)
Epinephrine spray	0 (0)	0 (0)		0 (0)
Others	0 (0)	1 (6.3)		1 (4.3)
Recurrent bleeding	7 (19.4)	11 (33.3)	0.189	18 (26.1)
Second endoscopy	6 (16.7)	10 (30.3)	0.180	16 (23.2)
Angiography	8 (22.2)	10 (30.3)	0.445	18 (26.1)
Surgery	2 (5.6)	2 (6.1)	1.000	4 (5.8)
Units of RBC transfused	3.8 ± 3.0	8.1 ± 4.9	**0.002**	5.9 ± 4.5
Days of hospital stay (days)	70.0 ± 50.2	79.6 ± 60.9	0.474	79.1 ± 62.5
Days of ICU stay (days)	33.3 ± 41.3	42.8 ± 45.1	0.366	39.8 ± 44.9
Hospital mortality	23 (63.9)	17 (51.5)	0.298	40 (58.0)
Causes of death			0.322	
Underlying disease	18 (50.0)	16 (48.5)		34 (49.3)
GI bleeding	2 (5.6)	0 (0)		2 (2.9)

Others	3 (8.3)	1 (3.0)	4 (5.8)

Values are presented as mean ± SD or number (%).

CS, colonoscopy; PMC, pseudomembranous colitis; CMV, cytomegalovirus; APC, argon plasma coagulation.

Table 4. The risk factors for recurrent UGIB after esophagogastroduodenoscopy

Variables	Univariate analysis			Multivariate analysis		
	Without rebleeding (N=198)	With rebleeding (N=55)	P-value	OR	95% CI	P-value
Age (years)	68.2 ± 12.9	71.7 ± 9.5	0.090			
Male gender	127 (64.1)	33 (60)	0.530			
APACHE II score	27.7 ± 9.6	26.1 ± 7.9	0.291			
MV	178 (89.9)	53 (96.3)	0.323			
Days of MV (days)	10.1 ± 15.8	8.0 ± 8.9	0.472			
Multiple organ dysfunction	124 (62.6)	31 (56.8)	0.505			
GCS score	10.6 ± 4.2	11.9 ± 3.2	0.019	1.124	0.99-1.27	0.066
Presence of shock	78 (39.2)	21 (38.6)	0.941			
Medication						
NSAIDs	11 (5.3)	2 (3.8)	0.845			
Antiplatelets	51 (25.8)	22 (40)	0.052	**3.345**	**1.25-8.95**	**0.016**
Anticoagulants	39 (19.7)	21 (38.2)	0.010	**2.881**	**1.01-8.21**	**0.048**
Corticosteroids	33 (16.7)	16 (29.1)	0.021	2.779	0.93-8.22	0.065
Stress ulcer prophylaxis	177 (89.5)	49 (88.6)	0.870			
Enteral nutrition	115 (58.1)	24 (43.6)	0.085			

Anemia	184 (92.9)	52 (94.6)	0.526			
Coagulopathy	65 (32.8)	37 (68.2)	0.000	**2.994**	**1.12-8.00**	**0.029**
Lab						
Hb (g/dL)	8.9 ± 2.3	8.2 ± 1.9	0.048	0.965	0.74-1.25	0.788
AST (IU/L)			0.700			
ALT (IU/L)	302.1 ± 1043.9	369.9 ± 1123.5	0.201			
BUN (mg/dL)	215.3 ± 1236.0	1430.6 ± 6184.8	0.005	**1.016**	**1.00-1.03**	**0.025**
Cr (mg/dL)			0.145			
	46.9 ± 30.8	61.6 ± 33.2				
	1.9 ± 1.3	2.6 ± 1.9				
Days between admission to the hospital and occurrence of GI bleeding	19.7 ± 27.5	9.9 ± 10.5	0.000	**0.948**	**0.92-0.98**	**0.002**
Days between admission to the ICU and occurrence of GI bleeding	9.1 ± 15.2	7.1 ± 8.6	0.393			
Identification of bleeding focus	130 (65.6)	34 (61.8)	0.597			
Units of RBC transfused	5.1 ± 4.5	10.7 ± 6.5	0.000	**1.253**	**1.15-1.36**	**0.000**

Values are presented as mean ± SD or number (%).

CI, confidence interval; MV, mechanical ventilation; GCS, Glasgow Coma Scale.

Table 5. The risk factors for recurrent LGIB after Colonoscopy

Variables	Univariate analysis			Multivariate analysis		
	Without rebleeding (N=51)	With rebleeding (N=18)	P-value	OR	95% CI	P-value
Age (years)	66.2 ± 11.5	68.2 ± 12.8	0.555			
Male gender	30 (58.8)	9 (50.0)	0.516			
APACHE II score	24.5 ± 9.3	27.0 ± 8.9	0.331			
MV	47 (92.2)	16 (88.8)	0.492			
Days of MV (days)	13.1 ± 12.3	14.8 ± 10.2	0.650			
Multiple organ dysfunction	29 (56.9)	14 (77.8)	0.115			
GCS score	12.4 ± 2.8	10.3 ± 4.2	0.068	0.818	0.64-1.05	0.108
Presence of shock	20 (39.2)	9 (50.0)	0.426			
Medication						
NSAIDs	2 (3.9)	0 (0)	1.000			
Antiplatelets	7 (13.7)	6 (33.3)	0.067	7.616	0.94-61.77	0.057
Anticoagulants	17 (33.3)	5 (27.8)	0.664			
Corticosteroids	18 (35.3)	7 (38.9)	0.785			
Stress ulcer prophylaxis	50 (98.0)	16 (88.9)	0.165			
Enteral nutrition	34 (66.7)	9 (50.0)	0.210			

Anemia	46 (90.2)	18 (100)	0.316			
Coagulopathy	15 (29.4)	12 (66.7)	0.005	**8.617**	**1.25-59.36**	**0.029**
Lab						
Hb (g/dL)	9.3 ± 2.3	8.7 ± 1.1	0.145			
AST (IU/L)	88.6 ± 301.8	170.3 ± 254.1	0.308			
ALT (IU/L)			0.395			
BUN (mg/dL)	56.6 ± 169.8	96.0 ± 160.7	0.006	1.028	0.99-1.06	0.090
Cr (mg/dL)	39.4 ± 23.5	67.7 ± 36.5	0.103			
	1.8 ± 1.4	5.2 ± 8.4				
Days between admission to the hospital and occurrence of GI bleeding	28.0 ± 27.4	14.8 ± 10.0	0.004	0.959	0.91-1.01	0.123
Days between admission to the ICU and occurrence of GI bleeding	13.5 ± 12.1	10.4 ± 10.5	0.338			
Operator (Expert)	12 (23.5)	7 (38.9)	0.210			
Identification of bleeding focus	30 (58.8)	11 (61.1)	0.865			
Units of RBC transfused	5.0 ± 4.2	8.3 ± 4.5	0.007	1.123	0.96-1.31	0.154

Values are presented as mean ± SD or number (%)

CI, confidence interval; MV, mechanical ventilation; GCS, Glasgow Coma Scale.

Discussion

GI bleeding is an important complication that causes increased mortality among critically ill patients. The incidence of UGIB is estimated to be 0.03-0.1%, and LGIB 0.02-0.027% in the general population.[9-11] Although our study does not evaluate the incidence of GI bleeding after ICU admission, previous studies of the patients admitted to ICU have identified overt UGIB in 0.6-2%, LGIB in 0.15-0.94% of patients.[7,12,13] High prevalence of GI bleeding must be associated with comorbid illness and critically ill status of ICU patients. We compared clinical characteristics and outcomes between patients admitted to ICU for UGIB and those whose bleeding developed after admission to ICU for other reasons. Although the patients admitted to the ICU for GI bleeding had more hemodynamic instability and lower Hb level when GI bleeding occurred, they showed better outcomes regarding shorter duration of hospital or ICU stay. Critically ill patients whose bleeding develops while in the ICU have a longer duration of hospital or ICU stay, which is most often thought to be related to the patient's underlying medical condition rather than a direct consequence of GI hemorrhage.

Most of the data on GI bleeding has collected from patients admitted to hospital with bleeding.[9,14-21] Peptic ulcers and erosive diseases accounted for nearly 50% and 20-35% of UGIB, respectively, in these patients.[9,19] However, few studies have been reported the etiology of GI bleeding in patients whose bleeding develops after hospitalization or admission to ICU.[9,14,16,20] In ICU patients undergoing EGD, peptic ulcers and erosive syndromes accounted for 56% and 17% of bleeding, respectively.[22] In our study, common causes of UGIB were peptic ulcer (34.8%) and AGML (17.0%). These are comparable to the results identified in previous studies in critically ill patients.[9,14,16,19,22] Furthermore, the present study demonstrates that the source of UGIB in ICU patients does not differ significantly from the source in patients admitted to the hospital for UGIB. The focuses of LGIB in the general population are mostly angiodysplasia, diverticular disease, polyp, and malignancy accounting for more than 50% of cases of

LGIB.[23,24] In contrast, main causes of LGIB in ICU patients were different substantially. Ischemic colitis, acute hemorrhagic rectal ulcer, and pseudomembranous colitis were predominant causes of LGIB in ICU patients according to data from this study and study reported by Kim *et al*.[13] This could be related with comorbid illness, old age, hemodynamic instability, and prolonged use of antibiotics.[12] Although uncommon in the general population, ischemic colitis occurs with greater frequency in the elderly and the critically ill patients. Ischemic colitis in critically ill patients is usually due to compromised blood flow by changes in the systemic circulation including sepsis, congestive heart failure, hypovolemia, and burns.[25] Acute hemorrhagic rectal ulcer is an important etiology of LGIB in ICU patients and elderly bedridden patients. With increasing elderly populations, and improved survival in critically ill patients, the incidence of acute hemorrhagic rectal ulcer has increased.[26]

There are several diagnostic approaches of GI bleeding, such as endoscopy, angiography, dynamic enhanced computed tomography (CT), and radionuclide scintigraphy, with a widely variable diagnostic rate (45-95%).[27] However angiography and CT could induce acute renal problems to patients with renal failure, and it is difficult to perform these procedures in ICU patients. Endoscopy can identify the site of bleeding with more than 95% of cases of UGIB and 74~90% of LGIB.[28-31] However, we demonstrated that a diagnostic rate of endoscopy was 79.4% for UGIB and 69.4% for LGIB. This result suggests that diagnostic yield of endoscopy is lower among the ICU patients than in general population. In the previous studies for GI bleeding in ICU patients, diagnostic rates of bedside endoscopy were 85% for UGIB and 65- 67% for LGIB.[7,12,13] These rates are similar to the results of the present study. Moreover, low diagnostic rates in our study could be explained by higher rate of small bowel bleeding of 4.8% than that of 1.4-3% in previous studies.[32]

Several studies of UGIB in the general population reported rates of endoscopic hemostasis for peptic ulcer disease with above 90%, and most of recurrent bleeding rates were below 20%. [33-37] In the previous studies of LGIB, primary endoscopic hemostasis rate is about 62% , and rebleeding rate ranged from 3.5% to 19% in a non-ICU setting.[2,27] Our study reported low endoscopic hemostasis rate of 26.9% in

UGIB, 33.3% in LGIB, which suggests either a lower therapeutic role of endoscopy or a decreased need of hemostasis in ICU patients. However the rate of recurrent GI bleeding was high (21.7% in UGIB, 26.1% in LGIB) in this study. These findings suggest that severely ill patients with GI bleeding have a higher frequency of rebleeding than non-critically ill patients. And this could be explained by the higher incidence of poor hemodynamics and coagulopathy of these severely ill patients, in addition to substantial differences in responsible lesions, especially in LGIB. Lee *et al*. also reported a primary hemostasis rate of UGIB in the ICU patients of 32.4% and rebleeding rate of 30.4% in prospective study.[7]

Early endoscopy was performed in 74% of the patients with UGIB, whereas in 53% of those with LGIB. Lower rate of early endoscopy in LGIB than in UGIB could be due to the time required for bowel preparation. Receiving early EGD has been shown to decrease the morbidity, mortality, and rebleeding rate in both non-ICU and ICU setting.[1] A Chak *et al*. reported early EGD was associated with approximately a 20% reduction in length of ICU stay and a 33% reduction in length of hospital stay.[8] Also, early CS can improve diagnostic and therapeutic yields and prevent the need for surgical treatment.[24,38] In this study, in both UGIB and LGIB, receiving early endoscopy reduced RBC transfusion requirements, but did not decrease mortality rate or rebleeding rate. Furthermore, there were different effects of early endoscopy on UGIB and LGIB in this study. Early EGD was independently associated with an increased diagnostic rate or primary hemostasis rate, whereas early CS was related with a reduced diagnostic rate or primary hemostasis rate. It could be explained by higher possibility of poor bowel preparation and blood interference in early CS group than those in late CS group (100% vs. 60%, p=0.076). Therefore early EGD should be considered in the ICU patients with GI bleeding, but CS should be carefully conducted after bowel preparation.

Several reports have suggested that the risk factors for UGIB in ICU patients include use of MV, coagulopathy, and renal failure.[1,6,22,39] Lin *et al*. also reported that coagulopathy, anticoagulation drugs, use of MV, and presence of shock were associated with LGIB in critically ill patients.[12] However there

were few studies reported risk factors for recurrent GI bleeding in ICU patients. The study reported by Koh *et al*. demonstrated recurrent UGIB was associated with anemia, hypoxemia, hypoalbuminemia, and higher units of blood transfused.[40] Our independent risk factors for recurrent UGIB in ICU patients are similar to risk factors of UGIB and recurrent UGIB in the previous studies, which are coagulopathy, medication with antiplatelets or anticoagulants, and higher RBC transfusion requirements. An increase in transfusion is considered as the index that reflects the anemia and hypoxemia due to large quantities of blood loss. In previous prospective study, blood transfusion appeared to reverse the hypercoagulable response to hemorrhage thereby encouraging rebleeding and hence the need for an operation.[41] What's important to recognize in this study is that a shorter period between admission to the hospital and arise of GI bleeding, and a higher level of BUN which means impaired renal function are found to be other independent risk factors for recurrent UGIB. In ICU patients, GI mucosa tends to be more friable and susceptible to injury from various causes including low perfusion, and rate of mucosal recovery is slower than those in non-ICU patients. A shorter period between admission to the hospital and occurrence of GI bleeding could mean a rapid progression of mucosal injury, and a more vulnerable mucosa. So the patients whose bleeding occurred shortly after admission to the hospital could show higher rate of rebleeding. GI mucosal abnormalities ranging from edema to ulceration can occur in two-thirds of patients dying of uremia,[42] which also supports the fact that a high level of BUN is likely to be associated with GI bleeding. However, high BUN levels also directly reflect the high load of nitrogen due to significant GI blood loss. Recurrent GI bleeding is not independently associated with the clinical outcomes such as in-hospital death, duration of hospitalization, or length of ICU stay.

It has been demonstrated that the mortality of patients with GI bleeding was significantly higher than that of those without GI bleeding. As in the study by the Schuster *et al*.,[39] critically ill patients who bled in ICU were more severely ill than those who did not, as judged by a higher incidence of MV support, a longer duration of such support in those who required it, a longer duration of ICU stay, and a higher mortality. Our observed in-hospital mortality rates of 44.7% in UGIB and 58% in LGIB are comparable

to the reported mortality rates of the ICU patients who underwent endoscopy for GI bleeding (48.5-77.1%).[6,7] GI bleeding related mortality rates in our study were low at 1.6% of UGIB and 2.9% of LGIB, similar to rates of range from 0% to 6.2% in the previous study.[7,22] This finding suggests that it is important to control of underlying diseases in the ICU patients with GI bleeding, because the critically ill patients with GI bleeding usually died due to decompensation of underlying disease rather than bleeding.

In conclusion, critically ill patients with GI bleeding that occurred during their ICU stay have similar causes of UGIB as patients admitted to hospital with bleeding, whereas ICU patients with LGIB have distinct sources of bleeding. Although bedside endoscopy shows an acceptable diagnostic rate without procedure-related complications, rates of endoscopic diagnosis and hemostasis in ICU patients are lower than those in general population. Early EGD increases the rate of diagnosis or primary hemostasis, but early CS reduces the rate of diagnosis or primary hemostasis. Receiving early endoscopy is effective for reducing the need of transfusion in both UGIB and LGIB, but it is not associated with a reduced risk of rebleeding or death, and length of ICU or hospital stay. The common risk factor for recurrent upper and lower GI bleeding is coagulopathy, and recurrent GI bleeding is not associated with the clinical outcomes such as in-hospital death, duration of hospitalization, or length of ICU stay. The mortality rate is high in patients with GI bleeding that develops in the hospital. However GI bleeding related mortality rate is low. These results suggest that patients' underlying conditions are important for both recurrent bleeding and hospital death in the ICU setting.

References

1. LOREN AL. Endoscopic therapy for acute nonvariceal upper gastrointestinal hemorrhage: a meta-analysis. Gastroenterology 1992;102:139-148.

2. Jensen DM, Machicado GA. Diagnosis and treatment of severe hematochezia. Gastroenterology 1988;95:1569-1574.

3. Rockall T, Devlin H, Logan R, Northfield T. Variation in outcome after acute upper gastrointestinal haemorrhage. The Lancet 1995;346:346-350.

4. Kupfer Y, Cappell MS, Tessler S. Acute gastrointestinal bleeding in the intensive care unit: the intensivist's perspective. Gastroenterology Clinics of North America 2000;29:275-307.

5. Beejay U, Wolfe MM. Acute gastrointestinal bleeding in the intensive care unit: the gastroenterologist's perspective. Gastroenterology Clinics of North America 2000;29:309-336.

6. Cook DJ, Fuller HD, Guyatt GH, et al. Risk factors for gastrointestinal bleeding in critically ill patients. New England journal of medicine 1994;330:377-381.

7. Lee Y-C, Wang H-P, Wu M-S, Yang C-S, Chang Y-T, Lin J-T. Urgent bedside endoscopy for clinically significant upper gastrointestinal hemorrhage after admission to the intensive care unit. Intensive care medicine 2003;29:1723-1728.

8. Chak A, Cooper GS, Lloyd LE, Kolz CS, Barnhart BA, Wong RC. Effectiveness of endoscopy in patients admitted to the intensive care unit with upper GI hemorrhage. Gastrointestinal endoscopy 2001;53:6-13.

9. Rockall T, Logan R, Devlin H, Northfield T. Incidence of and mortality from acute upper gastrointestinal haemorrhage in the United Kingdom. Bmj 1995;311:222-226.

10. Yavorski RT, Wong R, Maydonovitch C, Battin LS, Furnia A, Amundson DE. Analysis of 3,294 cases of upper gastrointestinal bleeding in military medical facilities. The American journal of gastroenterology 1995;90:568-573.

11. Longstreth GF. Epidemiology and outcome of patients hospitalized with acute lower gastrointestinal hemorrhage: a population-based study. The American journal of

gastroenterology 1997;92:419-424.

12. Lin C-C, Lee Y-C, Lee H, et al. Bedside colonoscopy for critically ill patients with acute lower gastrointestinal bleeding. Intensive care medicine 2005;31:743-746.

13. Kim BC, Cheon JH, Kim TI, Kim WH. Risk factors and the role of bedside colonoscopy for lower gastrointestinal hemorrhage in critically ill patients. Hepatogastroenterology 2008;55:2108-2111.

14. Blatchford O, Davidson LA, Murray WR, Blatchford M, Pell J. Acute upper gastrointestinal haemorrhage in west of Scotland: case ascertainment study. BMj 1997;315:510-514.

15. Katschinski B, Logan R, Davies J, Langman M. Audit of mortality in upper gastrointestinal bleeding. Postgraduate medical journal 1989;65:913-917.

16. Johnston SJ, Jones PF, Kyle J, Needham CD. Epidemiology and course of gastrointestinal haemorrhage in north-east Scotland. BMJ 1973;3:655-660.

17. Longstreth GF. Epidemiology of hospitalization for acute upper gastrointestinal hemorrhage: a population-based study. The American journal of gastroenterology 1995;90:206-210.

18. Logan R, Finlayson N. Death in acute upper gastrointestinal bleeding: Can endoscopy reduce mortality? The Lancet 1976;307:1173-1175.

19. Silverstein F, Gilbert D, Tedesco F, Buenger N, Persing J. THE NATIONAL ASGE SURVEY ON UPPER GASTROINTESTINAL-BLEEDING. 1. STUDY DESIGN AND BASELINE DATA: MOSBY-YEAR BOOK INC 11830 WESTLINE INDUSTRIAL DR, ST LOUIS, MO 63146-3318, 1981.

20. Schlup M, Barbezat G, Maclaurin B. Prospective evaluation of patients with upper gastrointestinal haemorrhage. The New Zealand medical journal 1984;97:511-515.

21. Mayberry J, Counsell B, Penny W, Rhodes J. Mortality in acute upper gastrointestinal haemorrhage: a six-year survey from the University Hospital of Wales. Postgraduate medical journal 1981;57:627-632.

22. Lewis JD, Shin EJ, Metz DC. Characterization of gastrointestinal bleeding in severely ill hospitalized patients. Critical care medicine 2000;28:46-50.

23. Leitman IM, Paull DE, Shires 3rd G. Evaluation and management of massive lower gastrointestinal hemorrhage. Annals of surgery 1989;209:175.

24. Jensen DM, Machicado GA, Jutabha R, Kovacs TO. Urgent colonoscopy for the diagnosis and treatment of severe diverticular hemorrhage. New England Journal of Medicine 2000;342:78-82.

25. Cappell MS. Intestinal (mesenteric) vasculopathy. II. Ischemic colitis and chronic mesenteric ischemia. Gastroenterology Clinics of North America 1998;27:827-860, vi.

26. Lin C-K, Liang C-C, Chang H-T, Hung F-M, Lee T-H. Acute hemorrhagic rectal ulcer: an important cause of lower gastrointestinal bleeding in the critically ill patients. Digestive diseases and sciences 2011;56:3631-3637.

27. Chaudhry V, Hyser MJ, Gracias VH, Gau FC. Colonoscopy: The initial test for acute lower gastrointestinal bleeding/Discussion. The American Surgeon 1998;64:723.

28. Peterson WL, Barnett CC, Smith HJ, Allen MH, Corbett DB. Routine early endoscopy in upper-gastrointestinal-tract bleeding: a randomized, controlled trial. New England Journal of Medicine 1981;304:925-929.

29. Jensen D, Machicado G. Colonoscopy for diagnosis and treatment of severe lower gastrointestinal bleeding. Routine outcomes and cost analysis. Gastrointestinal endoscopy clinics of North America 1997;7:477-498.

30. García SM, González GA, López VP, et al. [Role of early colonoscopy in severe acute lower gastrointestinal bleeding]. Gastroenterología y hepatología 2000;24:327-332.

31. Richter JM, Christensen MR, Kaplan LM, Nishioka NS. Effectiveness of current technology in the diagnosis and management of lower gastrointestinal hemorrhage. Gastrointestinal endoscopy 1995;41:93-98.

32. Bogoch A. Bleeding from the alimentary tract. Bockus gastroenterology. 5th ed. Philadelphia: WB Saunders Company 1995;1:62-84.

33. Sanowski R. Endoscopic injection therapy for nonvariceal bleeding lesions of upper GI tract. J Clin Gastroenterol 1989:249-257.

34. Jensen D. Heater probe for endoscopic hemostasis of bleeding peptic ulcers. Gastrointest Clin N Am 1991;1:319-339.

35. Choudari C, Rajgopal C, Palmer K. Comparison of endoscopic injection therapy versus the heater probe in major peptic ulcer haemorrhage. Gut 1992;33:1159-1161.

36. Lin H, Lee F, Kang W, Tsai Y, Lee S, Lee C. Heat probe thermocoagulation and pure alcohol injection in massive peptic ulcer haemorrhage: a prospective, randomised controlled trial. Gut 1990;31:753-757.

37. Cipolletta L, Bianco MA, Marmo R, et al. Endoclips versus heater probe in preventing early recurrent bleeding from peptic ulcer: a prospective and randomized trial. Gastrointestinal endoscopy 2001;53:147-151.

38. Ohyama T, Sakurai Y, Ito M, Daito K, Sezai S, Sato Y. Analysis of urgent colonoscopy for lower gastrointestinal tract bleeding. Digestion 2000;61:189-192.

39. Schuster DP, Rowley H, Feinstein S, McGue MK, Zuckerman GR. Prospective evaluation of the risk of upper gastrointestinal bleeding after admission to a medical intensive care unit. The American journal of medicine 1984;76:623-630.

40. Koh SJ, Cheon JH, Kim JS, et al. Risk factors for upper gastrointestinal rebleeding in critically ill patients. Korean Journal of Gastrointestinal Endoscopy 2006;32:320-325.

41. Blair S, Janvrin S, McCollum C, Greenhalgh R. Effect of early blood transfusion on gastrointestinal haemorrhage. British Journal of Surgery 1986;73:783-785.

42. Kang J. The gastrointestinal tract in uremia. Digestive diseases and sciences 1993;38:257-268.

요약 (국문 초록)

목적: 위장관 출혈은 중환자실 환자에서 나타나는 중요한 합병증 중 하나로, 매우 다양한 원인을 가지고 있으며 일반 병실이나 외래의 환자와는 다른 특성을 지니고 있다. 이번 연구에서는 임상적으로 의미 있는 위장관 출혈로 병상 내시경술을 시행 받은 중환자실 환자들을 대상으로 임상적 특징, 내시경 검사의 유효성 및 예후에 대해 알아보고자 하였다.

방법: 본 연구는 후향적 코호트 연구로서 2010년 1월부터 2015년 5월까지 서울대학교 병원에서 중환자실 입실 후 발생한 위장관 출혈로 병상 내시경술을 시행한 환자 (상부위장관내시경 253명, 하부장관내시경 69명)를 대상으로 환자기본정보, 내시경 정보, 재출혈과 사망 여부 등을 조사하여 분석하였다.

결과: 중환자에서 발생한 상부위장관 출혈의 원인으로 소화성궤양 (34%), 급성 위점막병변 (17%)이 많았고, 이는 일반인에서 발생한 상부위장관 출혈의 원인과 비슷하였다. 그러나 중환자에서 발생한 하부장관 출혈의 흔한 원인으로는 허혈성 장염 (24%)과 급성 출혈성 직장 궤양 (20%)으로 이는 일반인에서의 원인과는 차이가 있었다. 조기 내시경 검사는 상부위장관내시경 187명, 하부장관내시경 36명에서 시행되었다. 내시경을 조기에 시행함으로써 상부위장관 출혈에서 출혈 병소 발견율 (82% vs. 73%, p = 0.003)과 지혈술 시행 비율 (60% vs. 8%, p = 0.002)을 유의하게 높일 수 있었으나, 하부장관 출혈에서는 병소 발견율 (58% vs. 82%, p = 0.008)과 지혈술 시행 비율 (7% vs. 16%, p = 0.011)이 모두 감소하였다. 그러나 조기에 시행한 내시경은 재출혈과 사망률에는 영향이 없었다. 재출혈의 위험을 높이는 요인으로서 항혈소판제/항응고제 투여, 응

고장애, 높은 혈액요소질소 수치, 초기 출혈 발생시 높은 적혈구 수혈량이 있었다.

결론: 중환자실 환자에서 발생한 상부위장관 출혈에 대해 조기 내시경 검사는 진단 및 내시경적 지혈술에 있어서 효과적이었다. 하지만 하부장관 출혈에서 조기 대장내시경 검사는 적절한 장정결에 주의하여 신중히 시행해야 하겠다. 또 위장관 출혈이 있었던 중환자실 환자 중 응고장애, 항응고제 치료, 높은 혈액요소질소 수치, 초기 출혈 발생시 높은 적혈구 수혈이 필요했던 환자들은 재출혈에 대해 주의 깊은 관찰이 필요하겠다.

주요어: 병상 내시경, 위장관 출혈, 조기 내시경 검사

학번: 2014-21108