Predictive Risk Factors for Upper Gastrointestinal Bleeding with Simultaneous Myocardial Injury

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The aims of this study were to: (1) evaluate the epidemiology of simultaneous upper gastrointestinal bleeding (UGIB) and myocardial injury using parameters including troponin I (TnI); and (2) investigate the predictive risk factors of this syndrome. One hundred and fifty-five patients (101 men, 54 women; mean age, 64.7 ± 10.4 years; range, 38–94 years) at the emergency department (ED) with the major diagnosis of UGIB were included. They underwent serial electrocardiography (ECG) and cardiac enzyme follow-up. Emergent gastroendoscopy was performed within 24 hours in most patients except for those who refused or were contraindicated. Mild myocardial injury was defined as the presence of any of the following: typical ST-T change on ECG, elevated creatine kinase-MB (CK-MB) > 12 U/L, or TnI > 0.2 ng/dL. Moderate myocardial injury was defined as the presence of any two of the previously mentioned conditions. In total, 51 (32.9%) and 12 (7.74%) patients developed mild and moderate myocardial injuries, respectively. Myocardial injury was more common among patients with variceal bleeding (20/25 = 80.0%) than those with ulcer bleeding (23/112 = 20.5%). It could partially be attributed to a higher baseline TnI level in cirrhotic patients. After adjusting for significant risk factors revealed by the univariate analysis, UGIB patients with a history of liver cirrhosis and more than three cardiac risk factors comprised a high-risk group for simultaneously developing myocardial injury. Other factors including age, gender, the color of nasogastric tube irrigation fluid, history of nonsteroidal anti-inflammatory drug use, vasopressin or terlipressin administration, vital signs, and creatinine recorded at the ED were not significant predictors. Those who developed myocardial injury had a longer hospital stay (mean duration, 8.73 ± 6.94 *vs*. 6.34 ± 2.66 days; $p = 0.03$) and required transfusion of more units of packed erythrocytes.

Key Words: myocardial injury, troponin I, upper gastrointestinal bleeding
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Upper gastrointestinal bleeding (UGIB) and myocardial ischemia/infarction may occur simultaneously because massive UGIB compromises myocardial perfusion and reflex tachycardia increases myocardial oxygen consumption. Many studies were conducted to evaluate the causes and consequences of UGIB after acute myocardial infarction (AMI) and have found that decreased gastrointestinal blood flow and antiplatelet or anticoagulation therapy during AMI are two of the major causes of bleeding [1]. Myocardial injury after UGIB is frequently ignored because the

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signs and symptoms may be overshadowed by severe UGIB [2]. Several studies showed that this process is common, especially in critically ill patients [2–5]. Except for those with more cardiac risk factors and more severe anemia, there is no mutual agreement about other risk factors in this group. High-output heart failure has been noticed in cirrhotic patients and this syndrome is termed cirrhotic cardiomyopathy. Ventricular hyporesponsiveness is revealed when cirrhotic patients are challenged by pharmacologic or physiologic stress [6]. In addition, hemodynamic instability is often noticed in patients with liver cirrhosis and variceal bleeding [7]. This could potentiate (subclinical) heart failure and myocardial injury. Some medications used to treat bleeding varices, including terlipressin and vasopressin, have the potential for aggravating myocardial ischemia because of their coronary artery vasoconstricting effect [8,9]. However, their impact on myocardial injury during UGIB is generally not recognized.

Troponin I (TnI), which is more frequently used in recent years, is a more rapidly-rising and specific marker and outcome predictor during AMI [10]. It was shown to be a better biologic marker to detect occult myocardial injury during UGIB [5]. Pateron et al showed a frequent elevated TnI (32%) above the lower detection limit in patients with cirrhosis and indicated that it was associated with subclinical left ventricular myocardial injury [11]. The aims of this study were to: (1) evaluate the epidemiology of simultaneous UGIB and myocardial injury using parameters including TnI; and (2) determine the possible risk factors in patients who develop simultaneous myocardial injury.

Patients and Methods

Patients

Patients at the emergency department (ED) of Kaohsiung Medical University Hospital during the period from July to December 2004 with the chief complaint of tarry stool passage or blood vomitus were diagnosed with UGIB. This hospital is a tertiary referral medical center in the Kaohsiung metropolitan area of southern Taiwan. All were admitted to the gastrointestinal (GI) ward after stabilization. All UGIB subjects older than 35 years were included. Because myocardial injury is less common in younger and previously healthy patients with minor bleeding, we excluded those with the presence of the following: (1) younger than 35 years; (2) having less than three cardiac risk factors; (3) initial systolic blood pressure (BP) >100 mmHg; and (4) initial hemoglobin (Hb) >12 g/dL. Subjects underwent standardized treatment according to current guidelines [7,12]. Most of them underwent emergent gastroendoscopy within 24 hours, except for those who refused or were contraindicated (irritable patients or with arrhythmia and severe shock). During gastroendoscopic examination, hemostatic strategies were employed if peptic ulcers with exposure vessels or varices with signs of recent bleeding were diagnosed.

Risk factors and outcome

Electrocardiography (ECG) was done on arrival at the ED and 4 hours later. Cardiac enzymes, including creatine kinase (CK), CK-MB, and TnI (Beckman Coulter Ireland, Inc., Fullerton, CA, USA) were checked every 12 hours for three times after admission. The presence of liver cirrhosis was previously diagnosed in our hospital when a typical change in abdominal ultrasound in addition to a long-standing hepatitis history or evidence of hepatic decompensation (elevated bilirubin level, prolonged prothrombin time, presence of varices in gastroendoscopic examination, encephalopathy) were identified. We used the five traditional cardiac risk factors (diabetes, hypertension, first-degree relatives with premature MI [male relatives <45 years, female relatives <55 years], hypercholesterolemia, smoking) and also included other commonly associated risk factors (age, male gender, obesity). Previous UGIB history, underlying malignancy and drug use including aspirin, coumadin, nonsteroidal anti-inflammatory drug (NSAID) and steroid, and history of smoking and alcoholic beverage drinking were evaluated using a standardized questionnaire.

In our study, mild myocardial injury was defined as the presence of any of the following: serial ST-T deviation in two consecutive leads on ECG; elevated CK-MB>12 U/L or TnI>0.2 ng/dL (normal, <0.02 ng/dL) [13,14]. Moderate myocardial injury was defined as the presence of any two of the previously mentioned conditions. Recurrent bleeding was diagnosed and recorded if patients had an increased amount of tarry stool with hemodynamic instability (tachycardia or hypotension) and decreased Hb measurement within 3 days of initial therapy. Such patients underwent

gastroendoscopy for revision and hemostasis. Intravenous medication used to control bleeding (omeprazole, vasopressin, terlipressin, or somatostatin), total amount of packed erythrocyte transfusion, length of hospital stay, intensive care unit (ICU) stay and complications including severe infection, respiratory failure, acute renal failure, were also recorded and analyzed.

Statistical analysis

The χ^2 test or Fisher's exact test was used to examine the presence of mild or moderate myocardial injury by the distribution of general characteristics. Logistic regression was used to investigate the relationship between mild or moderate myocardial injury and other potential predictors that were significant in the univariate analysis. Data were analyzed using the SAS statistical package (SAS Institute Inc., Cary, NC, USA), and the significant two-sided p value was 0.05.

RESULTS

Prevalence of simultaneous UGIB and myocardial injury

One hundred and eighty-six patients diagnosed with UGIB were admitted to the GI ward via the ED. Twenty-one of them were excluded according to the exclusion criteria and 10 refused to participate in the study. Of the 155 patients (101 men, 54 women; mean age, 64.7 ± 10.4 years; range, 38–94 years) enrolled in this study, 152 underwent emergent endoscopy. The bleeding sources were from peptic ulcer (esophageal, gastric, or duodenal ulcer) in 112 patients, from varices in 25 and from other lesions (esophagitis, acute gastric mucosal lesion, or gastric erosion) in 15 patients (Table 1). Sixteen (10.6%) had ST-T deviation on ECG. Thirty-nine (25.2%) and 13 (8.4%) had elevated CK-MB and TnI above the upper limit, respectively. In total, 51 (32.9%) had abnormality in any of the three and were defined as developing mild myocardial injury. Twelve (7.74%) patients had simultaneous moderate myocardial injury and two of them were transferred to the ICU due to overt MI. Catecholamines were not used in any of them.

Risk factors for UGIB patients who developed mild or moderate myocardial injury

Thirty patients (19.4%) had a history of NSAID exposure and six of them were taking aspirin. Among the 155 patients, 31 (20.0%) had underlying liver cirrhosis. Fourteen of the 22 (66.5%) subjects diagnosed with variceal bleeding received intravenous omeprazole and terlipressin or vasopressin simultaneously in the first 2 days. Somatostatin was used in the remaining eight patients. Only one of the 130 cases with non-variceal bleeding received such vasoactive agents in addition to intravenous omeprazole. Myocardial injury was more common among patients with variceal bleeding (20/25 = 80.0%) than those with ulcer bleeding (23/112 = 20.5%). However, it was not related to vasopressin or terlipressin use. Mean TnI level was slightly higher among the 112 ulcer bleeding cases (0.11 ± 0.41 ng/dL) than the 25 variceal bleeding cases (0.80 ± 3.73 ng/dL; $p = 0.054$). In the ulcer bleeding group, seven patients (6.25%) had underlying cirrhosis. After adjusting for age, gender, and other potential confounders revealed by univariate analysis, the presence of underlying cirrhosis ($p = 0.01$) and more cardiac risk factors ($p = 0.007$) were associated with the development of mild myocardial injury. The mean age of the 104 UGIB patients without myocardial injury was 65.0 ± 12.2 years (range, 36–94 years), while that of the 51 patients with myocardial injury was 64.4 ± 13.2 years (range, 39–85 years; $p = 0.79$). The mean lowest hematocrit (Hct) level was 26.52 ± 7.58% in the 104 UGIB controls and 24.28 ± 5.57% in the 51 patients with simultaneous myocardial injury.

Univariate analysis revealed that liver cirrhosis, pitressin or terlipressin use, fresh color during nasogastric (NG) tube irrigation, more cardiac risk factors (> three items), and a lowest Hct ≤ 25% are risk factors in UGIB patients for developing mild myocardial injury (Table 1). After adjusting for those potential confounders, liver cirrhosis (adjusted odds ratio [aOR] = 7.68, 95% confidence interval [CI] = 2.78–21.20, $p < 0.0001$) and the presence of more than three cardiac risk factors (aOR = 3.38, 95% CI = 1.35–8.46, $p = 0.009$) were still significant predictors (Table 2). The significant predictors in UGIB patients who developed moderate myocardial injury were the same as the ones for mild myocardial injury by using univariate analysis (data not shown). After adjusting for age, sex, and other significant predictors in univariate analysis, liver cirrhosis and more than three cardiac risk factors remained significant risk factors (Table 2). Together, the mean BP and pulse rate on arrival, initial Hb and blood urea nitrogen (BUN)/creatinine (Cr) ratio were not significant predictors of the presence

Table 1. Distribution of general characteristics among 155 upper gastrointestinal bleeding cases dichotomized by the presence of mild myocardial injury

Variables	Case no.	Mild myocardial injury		*p*
		Present (*n* = 51), *n* (%)	Absent (*n* = 104), *n* (%)	
Gender				0.32
Male	101	36 (70.6)	65 (62.5)	
Female	54	15 (29.4)	39 (37.5)	
Age, yr				0.27
≤ 55	40	16 (31.4)	24 (23.1)	
> 55	115	35 (68.6)	80 (76.9)	
Mean BP on arrival (mmHg)				0.14
≤ 70	80	22 (43.1)	58 (55.8)	
> 70	75	29 (56.2)	46 (44.2)	
Cardiac risk factors				0.02
≤ 3	94	24 (47.1)	70 (67.3)	
> 3	61	27 (52.9)	34 (32.7)	
Liver cirrhosis				< 0.0001
Absent	124	30 (58.8)	94 (90.4)	
Present	31	21 (41.2)	10 (9.6)	
Vasopressin or terlipressin use				0.015
Absent	141	42 (82.4)	99 (95.2)	
Present	14	9 (17.6)	5 (4.8)	
NG color*				0.008
Fresh	44	20 (39.2)	24 (23.1)	
Not fresh	91	21 (41.2)	70 (67.3)	
Missing	20	10 (19.6)	10 (9.6)	
Initial Cr (mg/dL)				0.73
≤ 1.6	127	41 (80.4)	86 (82.7)	
> 1.6	28	10 (19.6)	18 (17.3)	
Lowest Hct (%)				0.09
≤ 25	73	29 (56.9)	44 (42.3)	
> 25	82	22 (43.1)	60 (56.7)	
History of NSAID use				0.34
Absent	125	39 (76.5)	86 (82.7)	
Present	30	12 (23.5)	18 (17.3)	
Cause of bleeding				< 0.0001
Peptic ulcer†	112	23 (45.1)	89 (85.6)	
Varices	25	20 (39.2)	5 (4.8)	
Others‡	15	5 (9.8)	10 (9.6)	
Missing	3	3 (5.9)	0	

*The color of the fluid during nasogastric tube irrigation; †including esophageal, gastric, and duodenal ulcers; ‡including esophagitis, acute gastric mucosal lesion, and gastric erosion. BP = blood pressure; NG = nasogastric; Cr = creatinine; Hct = hematocrit; NSAID = nonsteroidal anti-inflammatory drug.

of subsequent myocardial injury. Age, gender, diabetes mellitus, hypertension, renal function abnormality (Cr > 1.6 mg/dL), history of malignancy and use of painkillers were not found to be significant risk factors.

Outcome

One patient was discharged against advice on the 1st day and was excluded when analyzing the adverse outcome. As shown in Table 3, it was found that those who developed mild myocardial injury had a

Table 2. Distribution of variables and potential confounders among the cases with mild or moderate myocardial injury and 104 upper gastrointestinal bleeding controls without myocardial injury

Variables	Control ($n=104$), n (%)	Mild myocardial ischemia ($n=51$)			Moderate myocardial injury ($n=12$)		
		n (%)	aOR* (95% CI)	p	n (%)	aOR* (95% CI)	p
Gender				0.93			0.97
Male	65 (62.5)	36 (70.6)	1.00		8 (75.0)	1.00	
Female	39 (37.5)	15 (29.4)	0.92 (0.34–2.50)		4 (25.0)	0.96 (0.16–5.80)	
Age, yr				0.93			0.18
≤55	24 (23.1)	16 (31.4)	1.00		3 (25.0)	1.00	
>55	80 (76.9)	35 (68.6)	1.00 (0.97–1.03)		9 (75.0)	1.05 (0.98–1.12)	
CAD risk factors				0.009			0.02
≤3	72 (69.2)	24 (47.1)	1.00		4 (25.0)	1.00	
>3	34 (30.8)	27 (52.9)	3.38 (1.35–8.46)		8 (75.0)	8.52 (1.37–53.09)	
Liver cirrhosis				<0.0001			0.01
Absent	94 (90.4)	30 (58.8)	1.00		6 (50.0)	1.00	
Present	10 (9.6)	21 (41.2)	7.68 (2.78–21.20)		6 (50.0)	11.67 (1.66–82.69)	
Vasopressin or terlipressin use				0.35			0.21
Absent	99 (95.2)	42 (82.4)	1.00		8 (66.7)	1.00	
Present	5 (4.8)	9 (17.6)	1.96 (0.48–8.07)		4 (33.3)	5.74 (0.49–28.74)	
NG color†							
Fresh	24 (22.1)	20 (39.2)	1.00		7 (58.3)	1.00	
Not fresh	70 (67.3)	21 (41.2)	0.65 (0.26–1.66)	0.37	3 (25.0)	0.36 (0.06–2.14)	0.26
Missing	10 (9.6)	10 (19.6)	2.32 (0.68–7.89)	0.18	2 (16.7)	3.05 (0.34–27.82)	0.32
Lowest Hct (%)				0.15			0.86
≤25	44 (42.3)	29 (56.9)	1.00		7 (58.3)	1.00	
>25	60 (57.7)	22 (43.1)	0.56 (0.25–1.24)		5 (41.7)	0.87 (0.20–3.89)	

*aOR, after adjusting for age (≤55 *vs.* >55 yr), gender (male *vs.* female), CAD risk factors (≤3 *vs.* >3 items), NG color (fresh or not), lowest Hct (≤25% *vs.* >25%), the presence of liver cirrhosis or vasopressin use; †the color of the fluid during nasogastric tube irrigation. aOR = adjusted odds ratio; CI = confidence interval; CAD = coronary artery disease; NG = nasogastric; Hct = hematocrit.

longer hospital stay (mean duration = 8.9 ± 6.91 *vs.* 6.31 ± 2.65 days, $p=0.013$) and required more packed erythrocyte transfusion (5.94 ± 4.07 *vs.* 4.07 ± 3.94, $p=0.007$) than those who did not. A higher rebleeding rate (eight cases, 5.19%) was also seen in those with moderate myocardial injury than those without (25% *vs.* 3.5%, $p=0.01$). In those who had recurrent UGIB, five were from benign gastric ulcers, two were from duodenal ulcers, and one was from esophageal varices. None of them had a history of NSAID use. Four patients died; one and two of them had mild and moderate myocardial injury, respectively. One of them had rebleeding.

Discussion

In our study, the prevalence of simultaneous UGIB and moderate myocardial injury (7.74%) is similar to previous studies (0.94–14%) [2–4]. One of the explanations

Table 3. Comparison of the outcome among the 154 upper gastrointestinal bleeding cases according to presence of clue to myocardial injury*

	Mild myocardial injury			Moderate myocardial injury		
	Present (n = 50), n (%)	Absent (n = 104), n (%)	p	Present (n = 12), n (%)	Absent (n = 142), n (%)	p
Hospital stay (d)†						
Mean ± SD	8.9 ± 6.91	6.31 ± 2.65	0.013	12.33 ± 10.82	6.72 ± 3.42	0.10
PRBC transfusion (units)†						
Mean ± SD	5.94 ± 4.07	4.07 ± 3.94	0.007	8.25 ± 5.32	4.39 ± 3.81	0.001
In-hospital complications‡§						
Present	20 (40.0)	39 (37.5)	0.77	6 (50)	53 (37.32)	0.39
Absent	30 (60.0)	65 (62.5)		6 (50)	89 (62.68)	
Rebleeding¶						
Present	4 (8.0)	4 (3.85)	0.28	3 (25)	5 (3.52)	0.001
Absent	46 (92.0)	100 (96.15)		9 (75)	137 (96.48)	

*One patient was discharged against advice on the 1st day and was excluded; †Student's t test; ‡in-hospital complications included acute renal failure, respiratory failure, or severe infection; §χ^2 test; ¶Fisher's exact test. SD = standard deviation; PRBC = packed red blood cells.

for the variety in prevalence is that different parameters were used to define myocardial injury in those without chest complaints. In earlier studies, ECG, CK, and CK-MB but not TnI were used to define myocardial injury, and some of them were retrospective studies in which the prevalence might be underestimated. Recent reports showed that TnI is a more sensitive and specific marker for myocardial necrosis or strain [15]. However, elevation of TnI has been reported in various conditions including pulmonary embolism [16], major trauma [17], septic shock [18,19], and chronic renal insufficiency [20]. It is not always synonymous with infarction or ischemia. The level of significance of TnI elevation depends on the assay cut-off value. In a study of 227 UGIB patients, 12% were found to develop myocardial injury defined as an elevation of TnI (RxL Dimension, Dade Behring, Milan, Italy) above 0.15 ng/dL. The authors also showed that among the potential risk factors such as ECG change, initial BP, and history of coronary artery disease (CAD), only minimum Hb was found to be a significant predictor. Liver cirrhosis and mild renal dysfunction (Cr, 1.5–2.0 mg/dL) were not significantly associated with an elevated TnI [5]. Our study differed in that an integration of biomarkers (TnI and CK-MB) and ECG were used to define myocardial injury. The identified significant predictors were consistent using either one or two abnormalities as an indicator of myocardial injury. A recent report suggested that such an integration will help identify false-positive acute coronary syndrome patients in the ED [21].

Our study found that cirrhotic patients were probably among the high-risk groups to develop myocardial injury during UGIB. Vasopressin and terlipressin are commonly used to treat bleeding varices in cirrhotic patients and might cause coronary vasocontracting side effects [8,9]. In contrast, there is no evidence to suggest that somatostatin has coronary side effects. After adjusting for confounders including the use of those two drugs, cirrhosis was still shown to be a significant risk factor. However, vasopressin or terlipressin use was not an independent risk factor for developing myocardial injury. A study investigating 32 patients with liver cirrhosis showed that 10 (32%) had slightly elevated TnI (range, 0.06–0.25 ng/dL) [11]. Echocardiography was done and the authors suggested that elevated TnI was associated with subclinical left ventricular myocardial damage. One of our study's limitations is that we did not check the baseline CK-MB and TnI levels. The finding of a higher prevalence of myocardial injury among the variceal bleeding group (80.0%) than among the ulcer bleeding group (20.5%) could partially be attributed to a higher baseline CK-MB or TnI level in patients with liver cirrhosis. However, in the ulcer bleeding group, fewer patients (6.25%) had underlying cirrhosis, and we still found that those with more CAD risk factors and liver cirrhosis were in the high-risk group to develop

myocardial injury. In addition, we used a higher TnI level (0.2 ng/dL) to define the development of myocardial injury. Thus, other factors exist in addition to a higher baseline TnI to explain why cirrhosis is a risk factor of this syndrome. Although the effect of liver cirrhosis on coronary microcirculation is not clear, the cirrhotic heart develops a series of structural and functional abnormalities consisting of diastolic dysfunction and reduced myocardial reactivity during exercise [22,23]. These provide an explanation as to why cirrhotic patients are more susceptible to myocardial injury during UGIB and physicians should pay more attention to such patients.

The development of concurrent myocardial injury is associated with the amount and acuteness of blood loss, which is difficult to estimate precisely. It was reported that severe postpartum hemorrhage with hypovolemic shock is highly related to concurrent myocardial injury indicated by elevated TnI and ECG change, especially in those treated with catecholamine [24]. The amount of blood loss could be estimated by the presence of hypotension, tachycardia, orthostatic hypotension, and azotemia (BUN > 40 mg/dL or BUN/Cr > 20). Because it takes hours to achieve full compensation, the initial Hct or Hb measurement is not considered to be a good indicator of severity during acute blood loss. Frequent monitoring and careful fluid resuscitation are recommended. There is still controversy regarding the lowest Hb concentration that can safely be tolerated. One report showed that reversible AMI after blood transfusion was found in elderly patients with massive GIB (mean Hct elevated from 28.9% to 47.7%) [25]. In another study introducing isovolemic reduction of Hb concentration in 55 healthy adults, transient, reversible ST-segment depression occurred in three asymptomatic subjects when it reduced to 5–7 g/dL [26]. In our study, initial and minimum Hct levels (≤ 25% *vs.* > 25%) were not significant predictors for UGIB patients to develop myocardial injury. However, it is still important to consider the necessity of adequate blood transfusion before performing gastroendoscopy, which further increases heart load.

Only eight of the 154 cases (5.19%) had recurrent UGIB and none of them had a history of NSAID use. Recurrent bleeding occurred more often in patients who developed moderate myocardial injury (25% *vs.* 3.52%, $p = 0.01$), in whom antiplatelet or anticoagulation drugs were contraindicated. Three of the four cases of mortality were from the group who developed myocardial injury and one of them had recurrent UGIB. It follows that major complications often occur simultaneously, and treatment of rebleeding or MI should be coupled together. Those who developed moderate myocardial injury ($n = 12$) tended to have a longer hospital stay than those without. However, the difference was not significant and could be attributed to a smaller case number in this group. Another limitation of this study is that we did not exclude patients who had CK-MB or TnI above normal range on arrival, so it is difficult to tell if myocardial injury develops before, concurrent with, or after UGIB.

In conclusion, UGIB patients with a history of liver cirrhosis and more than three cardiac risk factors had a high risk of simultaneously developing myocardial injury. More adverse outcomes were found in those patients. Monitoring ECG and cardiac enzymes, including TnI, are recommended in high-risk patients, even if no chest complaints are present. Further studies are necessary to investigate the exact mechanisms by which liver cirrhosis precipitates myocardial injury during UGIB.

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急性上消化道出血患者併發心肌受損之危險因子探討

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本研究的目的在評估急性上消化道的病患，併發心肌受損的發生率並找出可預測的危險因子。共有 155 名至高醫急診求診且主診斷為上消化道出血的病患同意參與本研究 (男性 101 人，女性 54 人；平均年齡 64.7 ± 10.4 歲，分佈區間 38－94 歲)，他們均接受一系列心電圖及心肌酵素 (CPK、CK-MB、TnI) 的追踪；除了拒絕或不適合者，絕大多數病患均在 24 小時內接受緊急胃鏡。輕度心肌受損定義為出現典型心電圖 ST-T 變化、CK-MB > 12 U/L 或 TnI > 0.2 ng/dL 三項中任一項；中度心肌受損定義為上述三項中曾出現任二項異常者。依此定義，共有 51 人 (32.9%) 發生輕度心肌受損，12 (7.74%) 發生中度心肌受損。心肌受損在食道靜脈瘤出血的患者 (20/25 = 80.0%) 較潰瘍出血的患者 (23/112 = 20.5%) 常見；此現象無法排除與肝硬化患者可能有較高的基準 TnI 值有關。在校正過單變項分析有意義的因子後發現，潰瘍出血患者若有肝硬化病史或有超過三項冠心症危險因子者，是併發心肌受損的高危險群。其他因子包括年齡、性別、鼻胃管沖洗液的顏色、是否曾使用非類固醇止痛劑、 vasopressin 或 terlipressin、到急診時的生命徵象及肌肝酸值均非有意義的危險因子。而併發心肌受損者比單純出血者平均住院時數較長 (8.73 ± 6.94 *vs.* 6.34 ± 2.66 天；$p = 0.03$)，且需要接受較多的濃縮紅血球輸注。

關鍵詞：心肌受損，心肌酵素，上消化道出血

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