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Original article

The Glasgow prognostic score as a significant predictor of clinical outcomes in patients with acute coronary syndrome

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ABSTRACT

Background: Many studies have reported both systemic inflammatory response and malnutrition provide valuable predictions of prognosis in patients with acute coronary syndrome (ACS). This study aims to assess the association between the Glasgow prognostic score (GPS) by combining C-reactive protein and serum albumin concentration, and clinical outcomes in patients with ACS.

Methods: This retrospective study included patients admitted for ACS between June 2010 and May 2013 in St. Vincent's Hospital, The Catholic University of Korea. In this study, high GPS was defined as a GPS ≥ 1. Primary outcomes were 12-month all-cause and cardiovascular mortality, stroke, stent thrombosis and target vessel revascularization. We used an inverse probability of treatment weighting (IPTW) analysis to adjust for potential confounding covariates and presented event rates with Kaplan–Meier curves.

Results: Total 593 patients were included and follow-up for a median 3.7 years. The patients were classified into two groups: GPS = 0 ($n = 424$, 71.5%) and GPS ≥ 1 ($n = 169$, 28.5%). The incidences of primary outcomes were 4% and 8.9% for the GPS = 0 and GPS ≥ 1, respectively. The primary outcomes and all-cause mortality difference between the two groups were significantly within 1 month in the Kaplan–Meier curve analysis (log rank $p < 0.001$, log rank $p < 0.001$, respectively). IPTW analysis showed high GPS was independently associated with higher incidence of primary outcomes (HR: 2.206; 95% CI: 1.085–4.486; $p = 0.029$), higher all-cause mortality (HR: 5.963; 95% CI: 2.068–17.190; $p < 0.001$) and higher cardiovascular mortality (HR: 6.122; 95% CI: 1.882–19.914; $p = 0.003$).

Conclusions: High GPS is independently associated with both total and cardiovascular mortality in patients with ACS. Hence, GPS could be helpful in predicting mortality in ACS patients.

Introduction

Acute coronary syndrome (ACS) including ST-segment elevation myocardial infarction (STEMI), non-STEMI, and unstable angina, is serious and potentially life-threatening coronary heart disease. The clinical course of ACS varies considerably. Therefore, early risk stratification may help in the optimization of therapy and outcomes [1,2]. Scoring systems such as the Global Registry of Acute Coronary Events (GRACE), clinical SYNTAX score are excellent risk-stratification tools for ACS patients and have good predictive ability for in-hospital mortality and long-term major adverse cardiac events (MACE) among ACS population [3–6].

Moreover, various inflammatory markers such as C-reactive protein (CRP), fibrinogen, and neutrophil count are related to long-term mortality in ACS patients [7–9]. Recent study has found that nutritional status calculated using ideal body weight and serum albumin, is a significant prognostic factor in clinical outcomes among patients with acute myocardial infarction [10]. Both systemic inflammatory response and malnutrition provide valuable predictions of prognosis in patients with ACS.

The Glasgow prognostic score (GPS) is an inflammation-based prognostic scoring system that is combined CRP and serum albumin levels. Many studies have shown that GPS provides prognostic values for patients with various malignancies [11–14]. The score is easy to measure and routinely available. Previous

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study has demonstrated that the GPS is also useful for predicting the prognosis in acute heart failure patients [15]. However, the prognostic value of GPS in ACS has not yet been reported. Consequently, the aim of the present study was to investigate the clinical significance of GPS in ACS patients requiring percutaneous coronary intervention (PCI).

Methods

Participants

From June 2010 to May 2013, 1071 patients undergoing PCI with dual antiplatelet therapy were enrolled in this retrospective, observational, single-center study. Patients with stable angina ($n = 221$), silent ischemia ($n = 10$), vasospastic angina ($n = 32$), subjects who did not have their albumin or CRP levels measured ($n = 167$) and those who received prasugrel ($n = 48$) instead of clopidogrel were excluded. A total of 593 patients were included in the study.

Definition

All patients were classified into three groups based on the GPS as follows: GPS of 2: elevated CRP (>1.0 mg/dL) and hypoalbuminemia (<3.5 g/dL); GPS of 1: elevated CRP or hypoalbuminemia; GPS of 0: neither elevated CRP nor hypoalbuminemia.[11,16] In this study, high GPS was defined as a GPS ≥ 1. The primary outcome was a composite of all-cause death at 12 months, cardiac death, stroke, stent thrombosis and target vessel revascularization (TVR).

Data

We reviewed the following data from medical records retrospectively: patient demographics; types of regular medications taken; smoking history; body mass index (BMI); laboratory data (including hemoglobin, platelet, fasting blood sugar, glycated hemoglobin, creatinine, albumin, low density lipoprotein cholesterol, triglyceride and platelet reactivity unit; coronary artery angiographic results. Risk stratification was performed by means of the GRACE score [17]. Follow-up data were obtained from outpatient clinic records by independent research nurses. The current study was exempted from the requirement for written informed consent because the medical data were reviewed retrospectively. All data records were anonymously identified and analyzed. This study was approved by the Institutional Review Board of St. Vincent's Hospital at the Catholic University of Korea (IRB Approval No: VC18RESI0101).

Statistical analysis

Continuous variables are expressed as the mean ± standard deviation using Student's test. Categorical variables are presented as total number and percentages and were compared using the chi-square or Fisher's exact test. Clinical outcomes were determined using the Kaplan–Meier method and compared using the log-rank test. Cox regression analyses were performed to analyze the impact of GPS on clinical outcomes. Inverse probability of treatment weighting (IPTW)-adjusted analysis was conducted with significant variables identified by univariate Cox regression analyses ($p < 0.05$). The hazard ratio (HR) and 95% confidence interval (CI) were also calculated. A p value <0.05 was considered statistically significant. All statistical analyses were performed using Statistical Analysis Software (SAS, version 9.2, SAS Institute, Cary, NC, USA).

Results

Baseline characteristics of the study population and procedural parameters

Table 1 shows the baseline demographic, clinical, and laboratory characteristics of patients classified according to the GPS: GPS = 0 ($n = 424$, 71.5%) and GPS ≥ 1 ($n = 169$, 28.5%). Total 593 patients were included and follow-up for a median 3.7 years. Patients with GPS ≥ 1 at baseline were older, more likely to have a history of renal failure, had higher concentrations of fasting blood sugar, creatinine, and platelet reactivity units, and had higher GRACE risk scores. The prevalence of non ST elevation myocardial infarction was significantly higher in high GPS group. They also had lower use of medications including beta blocker, aspirin and statins, lower concentrations of hemoglobin, low-density lipoprotein cholesterol, and triglyceride.

Clinical outcomes of the study population

The incidences of primary outcomes including all-cause mortality, cardiovascular mortality, stroke, stent thrombosis and TVR were significantly higher in high GPS group than GPS = 0 group. (17 [4%] versus 15 [8.9%], $p = 0.016$, Table 2). In hospital events of primary outcomes were 80% in high GPS group whereas in hospital events of primary outcomes were 29.4% in GPS = 0 group. Moreover, the incidence of all death and cardiac death was significantly higher in the ACS with high GPS ($p < 0.001$, $p = 0.001$, respectively, Table 2). IPTW analysis showed high GPS was independently associated with higher incidence of primary outcomes (HR: 2.206; 95% CI: 1.085–4.486; $p = 0.029$), higher all-cause mortality (HR: 5.963; 95% CI: 2.068–17.190; $p < 0.001$) and higher cardiovascular mortality (HR: 6.122; 95% CI: 1.882–19.914; $p = 0.003$) (Table 2). The primary outcomes and all-cause mortality in high GPS group were significantly higher than GPS = 0 group within 1 month in the Kaplan–Meier curve analysis (log rank $p < 0.001$, $p < 0.001$, respectively, Fig. 1). After 1 month, there was no difference in the rates of primary outcomes and all-cause mortality between two groups (log rank $p = 0.458$, $p = 0.183$, respectively, Fig. 1).

Subgroup analysis

Subgroup analysis showed the significant association between high GPS and primary outcome in male, diabetes mellitus, and BMI ≥ 23 (Table 3). There were no significant interactions observed in all subgroups.

Discussion

In the present study, we evaluated the clinical outcomes according to the GPS in ACS population requiring PCI. Approximately 29% of the study's ACS patients presented with high GPS at admission, which was associated with an increased risk for all cause mortality and cardiovascular mortality. Moreover, mortality within 1 month was significantly higher in patients with a greater GPS of 1 on admission relative to patients with a GPS of 0.

The GPS combines the serum CRP and albumin levels count into a single composite marker of inflammation and nutrition status. The GPS was initially designed to assess the systemic inflammatory response in patients with inoperable non-small-cell lung cancer and provides prognostic values for patients with cancer [12]. Many studies have shown an elevation of inflammatory markers in ACS patients. And various inflammatory markers are related to long-term mortality in ACS patients [7–9,18,19]. CRP is the most common used indicator for inflammation and recent data have

Table 1
Baseline characteristics.

	GPS = 0 (n = 424)	GPS ≥ 1 (n = 169)	p-Value
Age (years)	61.6 ± 12.7	66.7 ± 12.1	<0.001
Male, n (%)	305 (71.9)	122 (72.2)	0.950
BMI	24.1 ± 3.1	23.8 ± 3.3	0.303
Risk factor			
HTN, n (%)	256 (60.4)	108 (63.9)	0.426
DM, n (%)	117 (27.6)	59 (34.9)	0.078
Smoking, n (%)	265 (62.5)	98 (58.0)	0.309
Medical history			
Previous MI, n (%)	13 (3.1)	4 (2.4)	0.789
Previous CABG, n (%)	1 (0.2)	1 (0.6)	0.489
CVA, n (%)	34 (8.0)	21 (12.4)	0.095
Renal failure, n (%)	42 (9.9)	45 (26.6)	<0.001
Treatment			
Beta blocker, n (%)	273 (64.4)	88 (52.1)	0.006
ACEi or ARB, n (%)	308 (72.6)	113 (66.9)	0.162
Aspirin, n (%)	421 (99.3)	159 (94.1)	<0.001
Statin, n (%)	354 (83.5)	120 (71.0)	<0.001
Laboratory finding			
Hb	14.2 ± 5.0	13.2 ± 2.2	<0.001
Platelet ($\times 10^9 L^{-1}$)	242.6 ± 61.3	239.7 ± 81.7	0.672
FBS	160.7 ± 75.3	180.9 ± 110.0	0.038
HbA1C	6.8 ± 1.6	6.8 ± 1.8	0.761
Creatinine	1.0 ± 0.8	1.2 ± 1.0	0.007
LDL cholesterol	123.1 ± 38.6	111.7 ± 37.0	0.001
TG	119.5 ± 83.5	105.1 ± 66.3	0.029
PRU	222.8 ± 97.6	243.4 ± 101.5	0.022
GRACE risk score	132.0 ± 38.7	157.4 ± 51.9	<0.001
Diagnosis			
UA, n (%)	187 (44.1)	73 (43.2)	0.841
NSTEMI, n (%)	78 (18.4)	45 (26.6)	0.026
STEMI, n (%)	159 (37.5)	51 (30.2)	0.092
MVD, n (%)	253 (59.7)	111 (65.7)	0.175

GPS, Glasgow prognostic score; BMI, body mass index; HTN, hypertension; DM, diabetes mellitus; MI, myocardial infarction; CABG, coronary artery bypass grafting; CVA, cerebro-vascular attack; ACEi, angiotensin-converting-enzyme inhibitor; ARB, angiotensin II receptor blocker; Hb, hemoglobin; FBS, fasting blood sugar; HbA1C, glycated hemoglobin; LDL, low-density lipoprotein; TG, triglyceride, PRU, P_2Y_{12} reaction unit; UA, unstable angina; NSTEMI, non ST elevation myocardial infarction; STEMI, ST elevation myocardial infarction; MVD, multivessel disease; GRACE, Global Registry of Acute Coronary Events.

been shown the association of raised CRP concentrations with adverse outcomes in ACS patients [8,20–23]. Moreover, CRP release in patients with coronary artery disease is associated to severity of atherosclerosis, inflammation related to the extent of myocardial ischemia and necrosis, and the amount of circulating pro-inflammatory cytokines such as interlukin-6 (IL-6) [24].

In addition, malnutrition has been reported as an independent risk factor for mortality in patients with ACS [10,25,26]. Albumin is a widely used indicator for nutrition. Moreover, there is increasing evidence that the serum albumin level decreases as the severity of inflammation increases [27–29]. Hypoalbuminemia is the result of the combined effects of inflammatory response and inadequate protein and caloric intake in both acute and chronic disease [30,31].

In the present study, we found that GPS on admission was an independent predictor of mortality in ACS patients and high GPS was independently associated with higher incidence of primary outcomes and all-cause mortality after adjustment by GRACE risk

Table 2
Primary outcomes.

	GPS = 0 (n = 424)	GPS ≥ 1 (n = 169)	Log-rank p-value	Univariate HR	p-Value	Adjusted HR by IPTW[a]	p-Value
Primary outcome	17 (4.0)	15 (8.9)	0.013	2.349 (1.173–4.705)	0.016	2.206 (1.085–4.486)	0.029
-In hospital events	5 (29.4)	12 (80.0)					
All Death	5 (1.2)	12 (7.1)	<0.001	6.477 (2.282–18.387)	<0.001	5.963 (2.068–17.190)	<0.001
-In hospital events	3 (60.0)	10 (83.3)					
Cardiac Death	4 (0.9)	10 (5.9)	<0.001	6.784 (2.127–21.632)	0.001	6.122 (1.882–19.914)	0.003
-In hospital events	2 (50.0)	8 (80.0)					
Stroke	7 (1.7)	2 (1.2)	0.769	0.790 (0.164–3.805)	0.769	0.861 (0.177–4.187)	0.853
-In hospital events	1 (14.3)	1 (50.0)					
Stent thrombosis	3 (0.7)	2 (1.2)	0.501	1.834 (0.306–10.973)	0.507	1.889 (0.316–11.304)	0.486
-In hospital events	1 (33.3)	2 (100.0)					
TVR	3 (0.7)	1 (0.6)	0.955	0.937 (0.097–9.008)	0.955	1.000 (0.103–9.669)	0.999
-In hospital events	1 (33.3)	1 (100.0)					

GPS, Glasgow prognostic score; HR, hazard ratio; IPTW, inverse probability of treatment weighting; TVR, target vessel revascularization; Hb, hemoglobin; FBS, fasting blood sugar; Cr, creatinine; LDL-C, low-density lipoprotein cholesterol; TG, triglyceride; PRU, platelet reactivity unit; GRACE, Global Registry of Acute Coronary Events; NSTEMI, non-ST elevation myocardial infarction.
[a] Adjusted by Age, Sex, Renal failure, Beta blocker, Aspirin, Statin, Hb, FBS, Cr, LDL-C, TG, PRU, GRACE risk score, NSTEMI

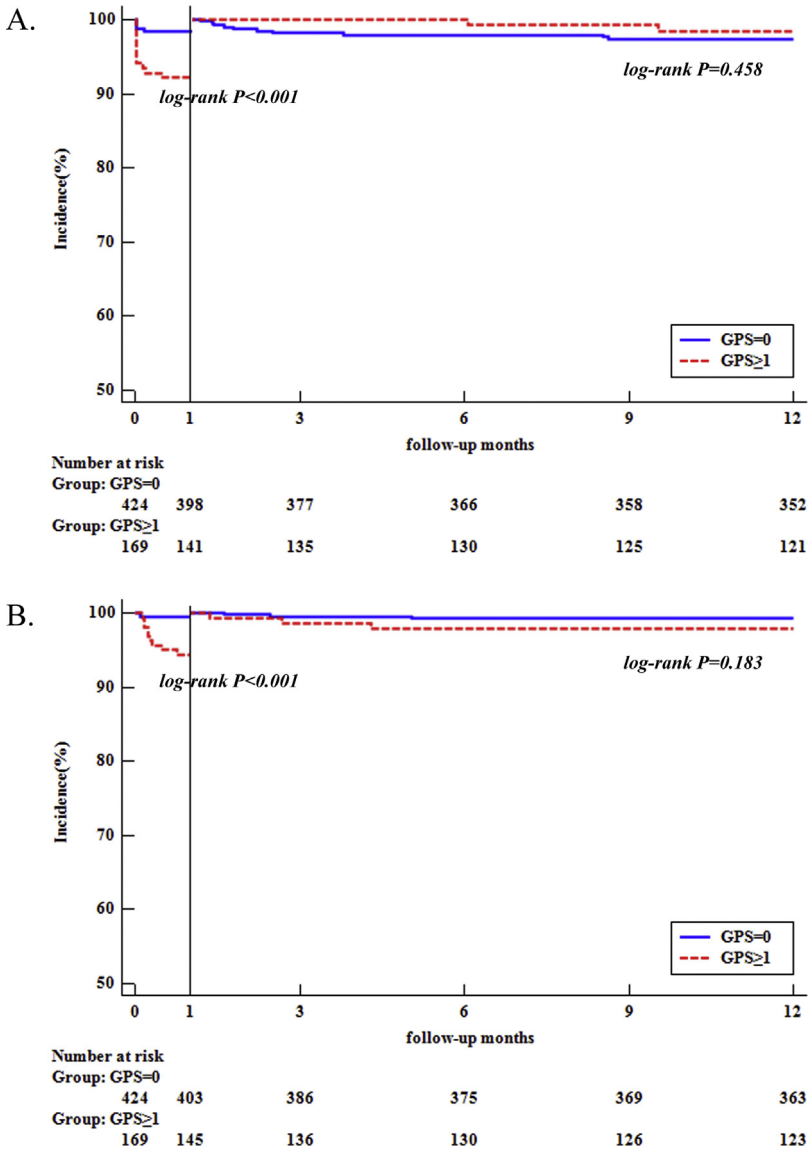

Fig. 1. Event-free Kaplan–Meier curves and 1 month landmark analysis of Clinical outcomes according to the Glasgow prognostic score. (A) Primary outcomes, (B) All-cause mortality.

score. Although it is well known that scoring systems such as the GRACE and SYNTAX score are excellent risk-stratification tools for ACS patients, GPS is easier to measure than GRACE and SYNTAX score. The mechanism of the link between the higher GPS and the poor clinical outcome of patients with ACS has not been completely understood. Because GPS is calculated based on the serum CRP and albumin levels in the peripheral blood, GPS may characterize both the inflammatory and nutritional status, which could affect the survival rate of cancer patients [11–14]. Several mechanisms may be responsible. CRP, an acute phase protein that is mainly produced in the liver in response to inflammatory cytokine such as IL-6, is a marker of inflammatory processes that play an important role in atherosclerosis, plaque disruption, and thrombosis. In ACS, thrombotic products such as thrombin and platelet derived growth factor, induce vascular smooth muscle cells within the ruptured plaque to increase production of IL-6, amplify CRP release, and complete a vicious cycle of thrombosis and inflammation [21]. In addition, serum albumin increases the production of the antiaggregatory prostaglandin (PGD2) from cyclic endoperoxides as a platelet aggregation inhibitor [32,33]. Furthermore, decreased albumin may increase blood viscosity [34]. Therefore, in theory, combining CRP and serum albumin count to create the GPS may be able to estimate the nutritional and inflammatory status of ACS patients.

To the best of our knowledge, this is the first study to investigate the prognostic value of GPS, a combined score based on CRP and serum albumin levels, in patients with ACS. However, the present study has several limitations. First, this retrospective study was conducted at a single center, and the findings in the present study need to be confirmed and validated with further multicenter studies in a larger population. Second, only baseline CRP and serum albumin levels were checked, and serial measurements of them may provide an additional prognostic value. Third, we compared two groups (GPS = 0 group and GPS $\geq$ 1). Because the GPS suggests incrementally worsening prognosis with increasing score originally for numerous different cancer, it is ideal dividing into three groups. However, only 30 patients (5.1%) among this study population were calculated the GPS = 2. Therefore, we compared two groups because the difference between three groups is large. In addition, there are some previous studies that increased GPS is

Table 3
Subgroup analysis.

	GPS = 0 (n = 424)		GPS ≥ 1 (n = 169)		HR	95% CI		p-Value	Interaction p-value
	No. of Event/ No. of patients	Event Rate per 1 year, %	No. of Event/ No. of patients	Event Rate per 1 year, %		Lower	Upper		
Age (years)									
<65	4/248	1.61	3/70	4.29	2.68	0.60	11.97	0.197	0.64
≥65	13/176	7.39	12/99	12.12	1.79	0.82	3.93	0.15	
Sex									
Male	11/305	3.61	10/122	8.20	2.38	1.01	5.61	0.05	0.97
Female	6/119	5.04	5/47	10.64	2.26	0.69	7.42	0.18	
DM									
No	12/307	3.91	7/110	6.36	1.69	0.67	4.30	0.27	0.35
Yes	5/117	4.27	8/59	13.56	3.50	1.14	10.71	0.03	
HTN									
No	0/168	0.00	3/61	4.92	–	–	–	–	–
Yes	17/256	6.64	12/108	11.11	1.80	0.86	3.76	0.12	
Renal failure									
No	9/382	2.36	4/124	3.23	1.41	0.43	4.58	0.56	0.97
Yes	8/42	19.05	11/45	24.44	1.46	0.59	3.63	0.42	
BMI									
<23	9/164	5.49	7/71	9.86	1.89	0.70	5.08	0.21	0.61
≥23	8/260	3.08	8/98	8.16	2.82	1.06	7.50	0.04	

GPS, Glasgow prognostic score; HR, hazard ratio; CI, confidence interval; DM, diabetes mellitus; HTN, hypertension; BMI, body mass index.

associated with reduced survival for various malignancies [14,35]. Finally, future prospective studies are warranted to clarify the pathophysiologic roles of the GPS components.

Conclusions

High GPS was identified as prognostic factor for poor clinical outcome in patients with ACS. Therefore, appropriate assessments of the nutritional-inflammatory status of ACS patients at the time of hospital admission can help to predict prognosis in ACS patients.

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