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Is Glasgow prognostic score a predictor of mortality in infective endocarditis?

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ABSTRACT

Objectives: The inflammation-based Glasgow prognostic score (GPS), which comprises elevated serum C-reactive protein (CRP) and decreased albumin concentration, is the most valid inflammatory risk score in cancer. New prognostic markers are needed to predict high-risk infective endocarditis (IE) patients. In the present study, we investigated the in-hospital mortality estimation of GPS in infective endocarditis patients.

Methods: The retrospectively designed study included 53 IE patients diagnosed according to Duke criteria. Demographic and clinical data of the patients were recorded and GPS levels were measured. Patients were divided into two groups according to in-hospital mortality outcomes. Glasgow prognostic score was rated as 0, 1, or 2 points based on serum albumin and C-reactive protein levels.

Results: The nonsurvivor group was older and the number of patients with kidney failure or diabetes was higher in this group. Glasgow prognostic score was higher in the nonsurvivor group, while albumin levels were lower. Thirty-four patients died during intensive care unit follow-up, and the mean follow-up period was 24.1 ± 18.6 days. ROC analysis showed that the Glasgow prognostic score had a sensitivity of 82.4% and a specificity of 36.8% at a cut-off value of ≥ 1.5 in predicting in-hospital mortality. Chronic renal failure (OR: 6.720; 95% CI: 1.907-23.684; $p = 0.003$) and age (OR: 1.040; 95% CI: 1.001-1.081; $p = 0.044$) were the independent variables of the mortality prediction in univariate logistic regression analysis. In multivariate logistic regression analysis, only chronic renal failure (OR: 0.153; 95% CI: 0.036-0.653; $p = 0.011$) was found to be a significant predictor of mortality. Kaplan–Meier survival analysis revealed that long-term survival was reduced in patients with a high GPS (Log-rank: $p = 0.003$).

Conclusions: Glasgow prognostic score level is associated with increased in-hospital mortality in IE patients. Chronic renal failure and GPS are the independent predictors of mortality.

Keywords: Glasgow prognostic score, infective endocarditis, mortality

Infective endocarditis (IE) is a rare condition with an annual incidence rate of 3 to 10 cases per 100,000 population [1]. Although rare, IE is associated with increased morbidity and mortality and is the most common life-threatening infectious syndrome after sepsis, pneumonia, and intra-abdominal abscess [2]. Fever, embolic stroke and heart failure are the most common symptoms; however, nonspecificity of the symptoms makes the diagnosis of the disease difficult. Therefore, diagnostic criteria should include clinical signs, imaging and laboratory results, and the modified Duke criteria are the most frequently used diagnostic

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tool [3, 4]. Despite early diagnosis and intensive treatment modalities, the morbidity and mortality rates of IE remain high. IE responsible for 10% to 24% of in-hospital short-term mortality [5-8].

Clinical, microbiological and echocardiographic features such as white blood cell count, serum albumin level, left heart involvement, presence of comorbid disease, mental status, heart failure, causative microorganism were found to be predictors of mortality in IE patients [9-11]. C-reactive protein (CRP) level is a potent predictor of short-term adverse events and a useful marker of early risk identification. Besides that, a low serum albumin level (< 30 gLl) increases the mortality risk approximately five times [9]. New prognostic markers are needed to predict high-risk IE patients.

Studies have shown that inflammation plays a vital role in the etiopathogenesis of cardiovascular diseases [12-15]. Inflammation plays a crucial role in cancer pathogenesis [16, 17]. The Glasgow prognostic score (GPS), obtained using CRP and albumin values, is a practical tool for determining the prognosis of various cancer types. In the present study, we investigated the impact of GPS on in-hospital mortality in IE patients.

METHODS

Study Population

A total of 53 patients diagnosed with IE according to Duke criteria between 2012-2022 were included in this retrospective study. The study protocol was reviewed and approved by the institutional ethics committee under the principles of the Declaration of Helsinki. Patients younger than 18 years of age, whose serum albumin or CRP levels were not analyzed, who received systemic steroid therapy, who had malignancy or chronic inflammatory disease or who had chronic liver disease were excluded from the study.

Biochemical analysis

The GPS was calculated as follows: 0 points were given to patients with CRP (≤ 10 mg/L) and albumin (≥ 3.5 mg/dL) levels within the normal range. One point was given to patients with abnormal CRP or abnormal albumin levels. Patients with both high CRP (> 10 mg/L) and hypoalbuminemia (< 3.5 mg/dL) were given 2 points [18]. Body mass index was calculated using the formula: Body mass index = body weight / (body height)^2. Blood culture positivity and accompanying microorganism were recorded.

Clinical Follow-up

Patients underwent transthoracic and (or) transesophageal echocardiography (TEE) (Epiq 7, Koninklijke Philips N.V., Amsterdam, Netherlands); intracardiac complications such as vegetation, perivalvular abscess, leaflet perforation, and paravalvular regurgitation were evaluated. Clinical complications such as acute heart failure, acute renal failure, peripheral embolism, need for surgery, and death were recorded. In the present study, IE patients were divided into two groups as non-survivor (group 1) and survivor (group 2) to determine the in-hospital mortality rates.

Statistical Analysis

Statistical analyses were performed using the SPSS 22.0 statistics package (SPSS Inc, Chicago, Ill, USA). Categorical variables were expressed as percentages. The Chi-square test and Fisher's exact tests were used for categorical variables. Normally distributed data were reported as mean ± standard deviation after being analyzed with Kolmogorov-Smirnov test, while non-normally distributed continuous variables were presented as median. Student's t-test was used for comparing normally distributed data, Mann-Whitney U test was used for comparing non-normally distributed data. Univariate and multivariate logistic regression analyses were used to determine the independent predictors of in-hospital mortality. Receiver operating characteristics (ROC) analysis was performed to determine the optimal cut-off value of Glasgow's prognostic score to predict mortality. Kaplan-Meier survival curve constructed for low and high Glasgow prognostic score groups. A p - value < 0.05 was considered statistically significant.

RESULTS

Fifty-three patients diagnosed with IE, according to Duke's criteria, were included in our study. Of these 53 patients, 20 (37.7%) patients were female and the mean age was 66.2 ± 16 years. The main demographic, laboratory and clinical variables of the groups are summarized in Table 1 and Table 2. When we exam-

Table 1. Demographic and laboratory variables of the patients

	All patients (n = 53)	Non-survivors (Group 1) (n = 34)	Survivors (Group 2) (n = 19)	*p value*
Age (years)	66.2 ± 16	69.6 ± 15.1	60.1 ± 16.1	0.03
Gender (female), n (%)	20 (37.7)	15 (44.1)	5 (26.3)	0.2
Height (cm)	167.5 ± 7.5	168 ± 8.21	166.6 ± 6.13	0.52
Weight(kg)	75.9 ± 13.4	77 ± 14.2	74.1 ± 12.1	0.46
Hospitalization time (days)	26.9 ± 18.8	24.1 ± 18.6	32 ± 18.6	0.14
Diabetes mellitus, n (%)	18 (34)	15 (44.1)	3 (15.7)	0.03
Hypertension, n (%)	39 (73.6)	28 (82.4)	11 (57.9)	**0.05**
Coronary artery disease, n (%)	22 (42.3)	14 (41.2)	8 (44.4)	0.82
Congestive heart failure, n (%)	8 (15.1)	6 (17.6)	2 (10.5)	0.48
Atrial fibrillation, n (%)	5 (9.4)	4 (11.1)	1 (9.4)	0.43
Chronic renal failure, n (%)	29 (54.7)	24 (70.6)	5 (26.3)	**0.002**
COPD, n (%)	3 (5.6)	1 (2.9)	2 (10.5)	0.25
Angina, n (%)	12 (22.6)	8 (23.5)	4 (21)	0.83
Dyspnea, n (%)	25 (47.2)	17 (50)	8 (42.1)	0.58
Syncope, n (%)	4 (7.54)	2 (5.8)	2 (10.5)	0.53
Fever, n (%)	50 (94.3)	32 (94.1)	18 (94.7)	0.92
Cerebrovascular disease, n (%)	16 (30.2)	13 (38.2)	3 (15.7)	0.15
Electrocardiographic evaluation, n (%)				
Atrioventricular block	1 (1.88)	1 (2.94)	0 (0)	0.45
Atrial fibrillation	5 (9.4)	4 (11.7)	1 (5.2)	0.43
Hemoglobin (g/dL)	8.5 ± 2	8.36 ± 1.8	9.85 ± 2.27	**0.01**
White blood cell×10^3/mm^3	18.2 ± 9.5	21.3 ± 9.4	12.7 ± 6.7	**0.001**
Neutrophil (10^9/L)	15.4 ± 9	18.1 ± 9.6	10.5 ± 5	**< 0.001**
Bazophil (10^9/L)	0.7 ± 0.1	0.8 ± 0.1	0.5 ± 0.02	0.26
Lymphocyte 10^9/L	2 ± 1	1.8 ± 0.7	1.74 ± 1.2	0.07
Monocyte (10^9/L)	0.9 ± 0.6	0.8 ± 0.46	0.99 ± 0.82	0.58
Thrombocyte (×10^3/mm^3)	157.7 ± 500	158 ± 118	157 ± 168	0.96
Mean platelet volume (fL)	10.8 ± 1.2	11.1 ± 1.9	10.3 ± 1.9	0.16
C-reactive protein (mg/L)	44.1 ± 30	47.8 ± 71.6	37.4 ± 70.5	0.61
Red cell distribution width (%)	49.6 ± 4.2	49.6 ± 6.9	49.5 ± 6.5	0.06
Blood urea nitrogen (mg/dL)	125 ± 34	154 ± 77.9	72.2 ± 44.4	**< 0.001**
Creatinine (mg/dL) (min-max)	2.7 (0.5-13.5)	3.8 (0.6-13.5)	1 (0.5-11.6)	**0.003**
Sodium (mmol/L)	134.3 ± 7.43	133.4 ± 8.2	136.1 ± 5.5	0.2
Potassium (mmol/L)	5.17 ± 1	5.4 ± 1.1	4.73 ± 0.5	**0.006**
Calcium (mg/dL)	8 ± 0.73	7.9 ± 0.74	8.33 ± 0.66	0.058
Alanine aminotransferase (U/L) (min-max)	36 (6-1518)	30.5 (6-1518)	40 (9-200)	0.65
Aspartate aminotransferase (U/L) (min-max)	40 (10-6366)	65 (10-6366)	32 (12-202)	0.07
Albumin (g/dL)	2.7 ± 0.6	2.46 ± 0.5	3.14 ± 0.5	**< 0.001**
Glasgow prognostic score	2 (0-2)	2 (1-2)	1 (0-2)	**< 0.001**

Table 2. **Clinical variables of patients according to survival**

	All patients (n = 53)	Non-survivors (Group 1) (n = 34)	Survivors (Group 2) (n = 19)	*p value*
Mitral valve, n (%)				
Native	32 (60.4)	20 (58.2)	12 (63.2)	0.75
Prosthesis	1 (1.88)	0 (0)	1 (5.26)	0.17
Aortic valve, n (%)				
Native	15 (28.3)	9 (26.5)	6 (31.6)	0.69
Prosthesis	6 (11.3)	6 (17.6)	0 (0)	0.052
sPAP (mmHg)	34.3 ± 12.7	34.4 ± 13	34.1 ± 12.6	0.94
Ejection fraction (%)	56.7 ± 6.9	55.9 ± 7.68	58.1 ± 5.1	0.22
TEE evaluation, n (%)				
Aortic vegetation	21 (39.6)	15 (44.1)	6 (31.6)	0.37
Mitral vegetation	33 (62.3)	20 (58.8)	13 (68.4)	0.48
Tricuspid vegetation	2 (3.7)	2 (5.8)	0 (0)	0.28
Abscess	2 (3.7)	2 (5.8)	0 (0)	0.28
Fistula	1 (1.88)	1 (2.9)	0 (0)	0.45
Perforation	2 (3.7)	1 (2.9)	1 (5.2)	0.67
Pseudoaneurysm	3 (5.6)	3 (8.8)	0 (0)	0.18
Paravalvular leakage	3 (5.6)	3 (8.8)	0 (0)	0.18
Prosthetic valve dehiscence	1 (1.88)	1 (2.9)	0 (0)	0.45
Vegetation size (cm)				
Aortic valve	0.39 ± 0.59	0.45 ± 0.62	0.28 ± 0.52	0.33
Mitral valve	0.73 ± 0.7	0.7 ± 0.75	0.77 ± 0.62	0.75
Septic emboli, n (%)	16 (30.2)	13 (38.2)	3 (15.7)	0.08
Blood culture positivity, n (%)	53 (100)	34 (100)	19 (100)	0.16
Microorganism, n (%)				
Streptococci	12 (22.6)	6 (17.6)	6 (31.5)	0.16
Brucella	0 (0)	0 (0)	0 (0)	0.16
Candida	1 (1.8)	0 (0)	1 (5.2)	0.16
Corynebacterium striatum	0 (0)	0 (0)	0 (0)	0.16
Escherichia coli	1 (1.8)	0 (0)	1 (5.2)	0.16
Enterococcus faecalis	10 (18.8)	7 (20.6)	3 (15.7)	0.16
Staphylococcus aureus	20 (37.7)	16 (47)	4 (21)	0.16
Methicillin-resistant *S. aureus*	8 (15)	8 (23.5)	0 (0)	0.16
Methicillin-susceptible *S. aureus*	13 (24.5)	9 (26.5)	4 (21)	0.16
Coagulase negative staphylococcus	8 (15)	6 (17.6)	2 (10.5)	0.16
Serratia marcescens	0 (0)	0 (0)	0 (0)	0.16
Stenotrophomonas maltophilia	0 (0)	0 (0)	0 (0)	0.16
Surgery, n (%)	17 (32.1)	8 (23.5)	9 (47.4)	0.07

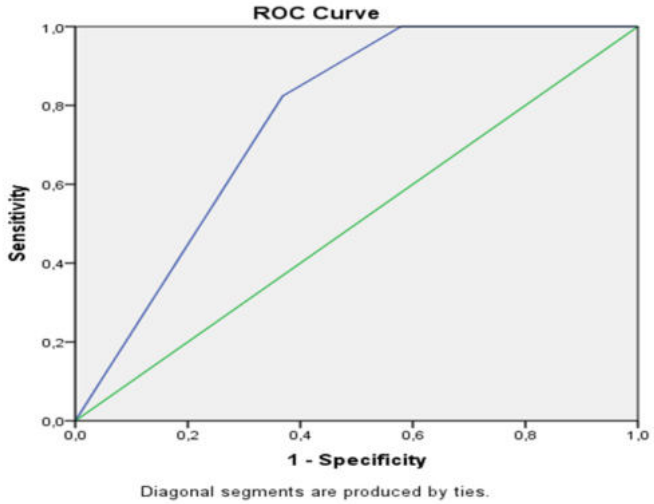

Fig. 1. Optimal cut-off value of Glasgow prognostic score found in roc curve analysis to predict in-hospital mortality

ined the basic laboratory and demographic characteristics of the patients, the non-survivor group was older (mean age group 1: 69.6 ± 15.1 vs. group 2: 60.1 ± 16.1 years; $p = 0.03$), and the number of patients with kidney failure (group 1: 24 (70.6%) vs. group 2: 5 (26.3%); $p = 0.002$) and diabetes (group 1: 15 (44.1%) vs. group 2: 3 (15.7%); $p = 0.03$) was higher in this group. When the groups were examined in terms of laboratory parameters, hemoglobin levels were lower in the non-survivor group, while the numbers of white blood cells, neutrophils, creatin, and blood urea nitrogen were higher. GPS and potassium levels were higher in the non-survivor group, while albumin levels were lower in this group. The two groups were similar in terms of other demographic and laboratory parameters. Vegetation sizes were similar between the two groups in both mitral valve and aortic valve IE. Vegetation on the native mitral valve was observed in 32 (60.4%) patients and vegetation on the prosthetic mitral valve was observed in one patient. Fifteen patients had vegetation on the native aortic valve and six patients had vegetation on the prosthetic aortic valve. The two groups were similar in terms of microorganisms grown in blood cultures, complications after IE and surgical need after complications. Thirty-four patients died during their intensive care follow-up, and the mean follow-up period was 24.1 ± 18.6 days. In the ROC analysis, the GPS had a sensitivity of 82.4% and a specificity of 36.8% at a cut-off value of ≥ 1.5 in estimating in-hospital mortality. The area under the

Table 3. Results of roc curve analysis to predict in-hospital mortality

Variable	AUC (95%)	Cut-off	*p value*	Sensitivity (%)	Specificity (%)
Glasgow prognostic score	0.765 (0.616-0.913)	≥ 1.5	**0.002**	82.4	36.8

Table 4. Univariate and multivariate logistic regression analysis of independent predictors of mortality

	Univariate analysis		Multivariate analysis	
Variables	**OR (95% CI)**	***p value***	**OR (95% CI)**	***p value***
Age	1.040 (1.001-1.081)	**0.044**	1,014 (0.969-1.062)	0.540
Hypertension	3.394 (0.955-12.057)	0.059	0.348 (0.077-1.579)	0.171
Chronic renal failure	6.720 (1.907-23.684)	**0.003**	0.153 (0.036-0.653)	**0.011**
Heart failure	1.821 (0.329-10.071)	0.492	0.659 (0.095-4.573)	0.673
Perforation	0.545 (0.032-9.249)	0.675	2.199 (0.090-53.782)	0.629

curve was 0.765 (95% CI: 0.616-0.913; p = 0.002) (Fig. 1) (Table 3). In Univariate logistic regression analysis, chronic renal failure (OR: 6.720; 95% CI: 1.907-23.684; p = 0.003), age (OR: 1.040; 95% CI: 1.001-1.081; p = 0.044) were independent variables in predicting mortality. In the multivariate logistic regression analysis, only chronic renal failure (OR: 0.153; 95% CI: 0.036-0.653; p = 0.011) was found to be a predictor of mortality (Table 4). Kaplan-Meier survival analysis also revealed that long-term survival was significantly reduced in patients with a high GPS level (Log-rank: p = 0.003) (Fig. 2).

DISCUSSION

To the best of our knowledge, the present study is the first to show an association between GPS and in-hospital mortality in IE patients. We demonstrated that long-term survival was reduced in patients with a high GPS. In addition, chronic renal failure was found to be an independent predictor of mortality.

Inflammation plays an essential role in the etiopathogenesis of cardiovascular diseases. Increased inflammatory markers are associated with poor prognosis in cardiovascular diseases [13, 18-20]. Inflammatory markers correlate with the mortality rate in various cancer types [21, 22]. The present study showed that GPS based on serum CRP and albumin levels had prognostic value in IE as in cancer patients. The elevation of inflammatory markers, such as leukocyte and neutrophil counts together with the GPS in the nonsurvivor group, supports this result.

Albumin is one of the variables in the GPS. Albumin, synthesized by hepatocytes, is the most abundant plasma protein. In addition to its role in osmotic pressure, it is also a good indicator of nutritional status and has antioxidant and anti-inflammatory properties [23, 24]. Therefore, lower albumin levels may cause increased inflammation and oxidative stress. Albumin is also an acute phase reactant protein; during the inflammatory process, albumin synthesis decreases due to decreased synthesis in hepatocytes, increased leakage into the interstitial space and catabolism [16]. Hypoalbuminemia, which occurs due to increased inflammation, is a powerful predictor of mortality in cardiovascular disease [17]. Hypoalbuminemia is associated with coagulation factors and a prothrombotic state [25]. Low albumin levels might be associated with a poor prognosis in IE patients.

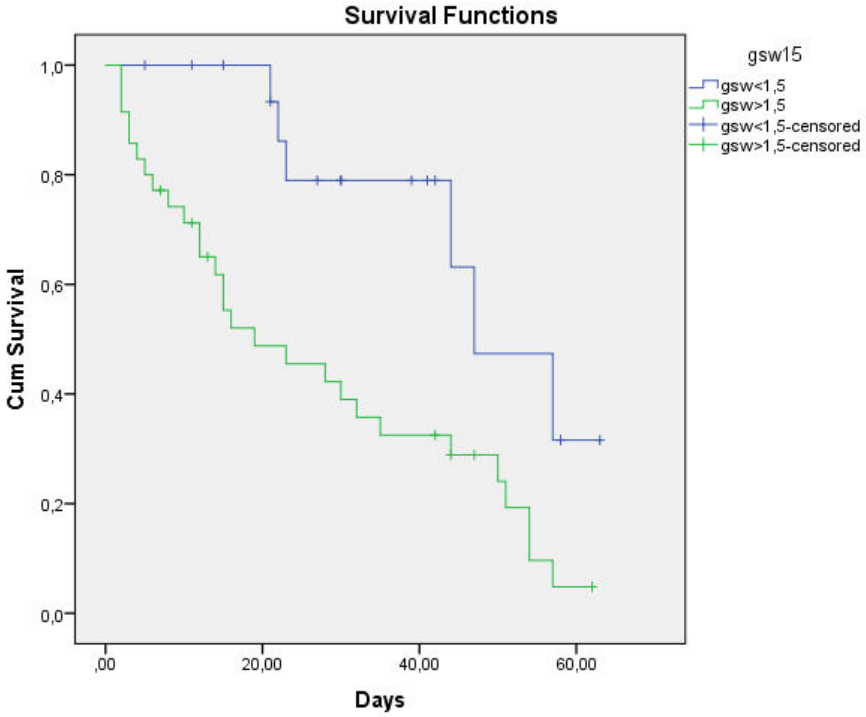

Fig. 2. Kaplan-Meier survival curve for low and high Glasgow prognostic score (GPS) groups. In-hospital mortality occured more often in patients with a higher GPS. While the mean life expectancy was 26.8 days in the group with GPS > 1.5, it was 47.9 days in the group with GPS < 1.5 (p = 0.003).

The prevalence of chronic kidney disease increases every year and the number of patients undergoing dialysis accelerates accordingly. Such patients are more susceptible to complications such as malnutrition, cardiovascular events, anemia and infections compared to healthy individuals [26]. As renal function declines with age, renal clearance gets lower in elderly patients. In the current study, the high rate of renal failure in the nonsurvivor group may be attributed to the fact that they were not under an adequate antimicrobial treatment due to avoidance of nephrotoxicity or drug side effects.

GPS is a specific index of systemic inflammation and malnutrition calculated by the combination of CRP and albumin. GPS predicts mortality in various types of cancer and can identify critically ill patients in many diseases [27-30]. As expected, low hemoglobin, presence of DM and advanced age, which are associated with adverse outcomes in infectious diseases, were more common in the high GPS group of the current study.

Limitations

The study is a retrospective, single-center research, and its sample size is relatively small. Retrospective nature of the study might have caused some factors that affected the results to be overlooked. The present study should be supported by multicenter, long-term prospective studies investigating the use of GPS in predicting prognosis in IE patients.

CONCLUSION

A high GPS is an independent indicator of in-hospital mortality in IE patients. GPS , determined using albumin and CRP levels, is a simple and practical index for predicting the prognosis in hospitalized patients with IE.

Authors' Contribution

Study Conception: NE, MAŞ; Study Design: MAŞ, NE; Supervision: EE, AGÖ; Funding: AGÖ; Materials: MAŞ; Data Collection and/or Processing: MAŞ, NE; Statistical Analysis and/or Data Interpretation: CA; Literature Review: CA; Manuscript Preparation: NE, CA and Critical Review: AGÖ.

Conflict of interest

The authors disclosed no conflict of interest during the preparation or publication of this manuscript.

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