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Is the Naples prognostic score useful for predicting heart failure mortality

Aim	The Naples prognostic score (NPS) simultaneously evaluates inflammation and malnutrition, which are two main factors that play a role in the pathophysiology and prognosis of heart failure (HF). In this study, we aimed to examine the relationship of NPS with in-hospital mortality of hospitalized patients with a diagnosis of HF.
Material and Methods	A total of 496 hospitalized HF patients included in this study. The patients were divided into two groups as deceased and living. The clinical and demographic characteristics of each patient were recorded. NPS of each patient was calculated.
Results	NPS was significantly higher in the deceased group compared to the living group (3.6±0.61, 3.21±0.97, respectively; p=0.003). According to multivariate regression analysis: NPS (OR: 1.546, 95% CI: 1.027–2.327; p=0.037), systolic blood pressure (OR: 0.976, 95% CI: 0.957–0.995; p=0.015), and white blood cell count (OR: 1.072, 95% CI: 1.007–1142; p=0.03) are independent predictors for in-hospital mortality in HF patients.
Conclusion	This study demonstrated a strong correlation between NPS and mortality in HF. This new score can be used to predict the prognosis of HF as it shows both the level of inflammation and nutrition.

Keywords	Naples prognostic score; heart failure; ınflammation; nutrition
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Introduction

Heart failure (HF) still continues to have a high mortality rate, and its frequency increase with the increasing age of the population. The HF prevalence is around 10% over the age of 70 [1]. The mortality after diagnosis of heart failure is 50% at 10 yrs and 10% at 5 yrs [1].

Despite advances in treatment options in recent years, the lifetime risk of HF remains high. Many risk models have been developed to reduce mortality and hospitalizations in HF and to increase the quality of life [2].

Inflammation is very important in the pathophysiology of HF, as in other cardiovascular diseases [3, 4]. In recent studies, hematologic inflammatory biomarkers, such as the neutrophil-lymphocyte ratio and the monocyte-lymphocyte ratio, were found to have poor prognostic value in HF [5–9]. Those findings contributed importantly to the risk classification and mortality assessment of HF patients. Another factor affecting the prognosis of HF is malnutrition [10, 11]. Hypoalbuminemia and cachexia are indicators of malnutrition, and studies have shown that these indicators adversely affect the prognosis of HF [12, 13]. Most of the HF prognosis studies considered either inflammation or malnutrition.

The Naples prognostic score (NPS) is a newly defined score calculated from the serum total albumin, total cholesterol, the neutrophil-lymphocyte ratio (NLR), and the monocyte-lymphocyte (NMR) ratio [14]. The NPS score was especially useful in evaluating the prognosis of oncologic malignancies [14]. To the best of our knowledge, the NPS score has not been examined in the cardiovascular area. NPS simultaneously evaluates inflammation and malnutrition, which are two main factors that play a role in the pathophysiology and prognosis of HF. Somewhere state that a high NPS might be expected to indicate a poor prognosis. Therefore, we thought that the NPS would provide more accurate results for evaluating the prognosis of HF patients. In this study, we aimed to examine the relationship of the NPS with in-hospital mortality in hospitalized patients diagnosed with HF.

Material and Methods

Study Population

A total of 562 HF patients with reduced ejection fraction and acute decompensated or moderate HF were followed in our clinic during the last three years. 496 of these patients

were included in this study; 66 patients were excluded due to missing data. Those included in the study were mostly HF patients with reduced left ventricular ejection fraction (LVEF), LVEF <%40, plus a small number of patients with mildly reduced LVEF (%40 ≤LVEF; <%50) hospitalized with acute decompensation.

We excluded patients with cardiogenic shock, cardiac arrest, chronic inflammatory diseases, severe infection, major surgery, trauma, acute pulmonary embolism, stroke, respiratory failure, and tumor over the past three months; we also excluded patients without complete clinical data. This study was conducted according to the Declaration of Helsinki, and it was approved by the local ethics committee. The patients' data were assessed retrospectively using the hospital registry system.

Clinical characteristics and physical examination findings of the patients were recorded at the time of admission. Bedside echocardiography was performed for each patient. LVEF was calculated by the modified Simpson method. Demographic characteristics, fasting blood glucose concentrations, total blood counts, renal function parameters, including urea, creatinine, glomerular filtration rate, and electrolyte concentrations, C-reactive protein (CRP), lipid profiles [low-density lipoprotein cholesterol (LDL–C), high-density lipoprotein cholesterol (HDL–C), and triglyceride (TG) concentration], albumin, aspartate transaminase (AST) were among the clinical variables recorded. Demographic and laboratory data are listed in Table 1. Patients were considered as hypertensive (HT) if the systolic blood pressure/diastolic blood pressure was above 140/90 mmHg in two or more measurements, or if the patients were using any antihypertensive agent. Diabetes mellitus (DM) was defined if fasting blood glucose was above 126 mg/dl, if postprandial blood glucose was above 200 mg/dl, if glycated hemoglobin above 6.5%, or if the patient was using any anti-diabetic medication.

Table 1. Baseline clinical and demographic characteristics of the study population

Variable		Death: Yes (n=116) Group 1	Death: No (n=380) Group 2	p value
Age (yr) (range)		70.8±10.2 (47-90)	68.5±11.8 (31-93)	0.136
Gender, male		72 (62)	239 (62.8)	0.128
DM		43 (37.06)	185 (48.6)	0.103
HT		57 (49.1)	191 (50.2)	0.944
HL		18 (15.5)	45 (11.8)	0.377
Smoking		28 (24.1)	102 (26.8)	0.63
CAD		65 (56.03)	233 (61.3)	0.516
CRF		28 (24.1)	78 (20.5)	0.422
Stroke		5 (4.3)	16 (4.2)	0.992
Respiratory failure		23 (19.8)	64 (16.8)	0.539
Malignancy		9 (7.8)	19 (5)	0.372
LVEF		36.65±12.33	38.0±11.6	0.378
Systolic blood pressure (mmHg)		106.03±21.93	117.4±24.1	0.0001*
Diastolic blood pressure (mmHg)		64.97±10.94	68.6±11.7	0.003*
NPS	0	0 (0)	5 (1.3)	0.003*ab
	1	2 (1.7)	22 (5.7)	
	2	3 (2.6)	52 (13.6)	
	3	37 (31.9)	111 (29.2)	
	4	75 (64.6)	192 (50.5)	
Medication	Asa	45 (38.7)	198 (52.1)	0.064
	P2y12 İnh.	20 (17.2)	67 (17.6)	0.877
	Bb	77 (66.3)	261 (68.6)	0.693
	ACEI/ARB	35 (30.1)	152 (40)	0.137
	Statın	18 (15.5)	85 (22.3)	0.224
	Spırono-lactone	36 (31)	124 (32.6)	0.871
	Antıcoagulan	46 (39.6)	125 (32.8)	0.212
	Dıuretıc	81 (69.8)	226 (59.4)	0.108
	CCB	28 (24.1)	75 (19.7)	0.473
	Dıgoxın	23 (19.8)	68 (17.8)	0.714

Data are mean±SD or count (percentage).
* Significant difference, Yes vs No. DM, diabetes mellitus; HT, hypertension; HL, hyperlipidemia; CAD, coronary artery disease; CRF, chronic renal failure; LVEF, left ventricular ejection fraction; NPS, Naples prognostic score; ASA, asetil salicylic acid; BB, beta blocker; ACEI, angiotensin converting enzyme inhibitor, ARB, angiotensin receptor blocker. CCB, calcium channel blocker.
a: Significant difference between score 2 and score 3;
b: Significant difference between score 2 and score 4.

Calculation of the NPS

To compute the NPS, as defined by Galizia et al. [14], for serum albumin <40 g/l, for total cholesterol ≤180 mg/dl, and for NLR >2.96, or LMR ≤4.44 each was assigned 1 point or, otherwise, 0 points were assigned. The NPS was defined as the sum of the points. The patients were classified according to their NPS: 0, 1 or 2, and 3 or 4 [14].

Statistical Analysis

All statistical analyses were performed using SPSS 25.0 (IBM SPSS Statistics 25 software [IBM Corp., Armonk, NY, USA]). Continuous variables are reported as mean±standard deviation (SD) median (IQR: 25^{th} – 75^{th} percentiles) and categorical variables are reported as number and percent. Kolmogorov Smirnov and Shapiro Wilk tests were used to identify normal distributions. For independent group comparisons, we used the independent samples t-test when parametric test conditions were satisfied and the Mann–Whitney U test when parametric test conditions were not satisfied. Difference between categorical variables was analyzed with Chi–Square analysis. Independent predictors of mortality were determined using multivariate logistic regression analysis. All variables with a p-value of <0.05 in the univariate analysis and variables known as risk factors for HF were examined in the multivariate model. Statistical significance was determined as p<0.05.

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Table 2. Laboratory values of the study population

Variable	Death		p value
	Yes (n=116) Group 1	No (n=380) Group 2	
WBC (µl/ml)	11.1±5.19	9.93±3.69	0.118 (z=-1.562)
HMG (g/dl)	11.88±1.91	12.04±2.08	0.546 (t=0.604)
RDW (%)	17.41±2.65	16.26±2.56	0.0001* (z=-3.583)
Glucose (mg/dl)	154.66±79.15	157.92±84.43	0.656 (z=-0.446)
Creatine (mg/dl)	1.73±0.89	1.39±0.69	0.0001* (z=-3.517)
GFR (ml/dk/1.73 m²)	52.13±17.54	60.4±23.8	0.001* (t=3.318)
Sodium (mmol/l)	134.9±6.84	136.84±5.28	0.02* (z=-2.331)
Potassium (mmol/l)	4.55±0.73	4.45±0.67	0.226 (z=-1.211)
AST (U/l)	53.69±58.68	42.92±37.25	0.235 (z=-1.188)
CRP (mg/l)	5.29±5.82	4.59±6.46	0.017* (z=-2.387)
Uric Acid (mg/dl)	9.24±3.72	8.41±3.34	0.076 (z=-1.776)
Platelets (10^3/l)	237.09±113.58	234±87.84	0.748 (z=-0.321)
Lymphocytes (K/uL)	1.18±0.83	1.34±0.72	0.016* (z=-2.405)
Neutrophils (10^3/l)	8.43±4.69	7.33±3.18	0.097 (z=-1.658)
Monocytes (10^3/l)	0.70±0.28	0.64±0.27	0.068 (z=-1.826)
Albumin (g/l)	3.43±0.57	3.63±0.60	0.007* (z=-2.699)
Total cholesterol (mg/dl)	125.77±33.44	143.34±42.74	0.001* (z=-3.381)
PLR	302.8±291.0	247.5±222.87	0.114 (z=-1.58)
NLR	10.07±11.52	7.86±8.17	0.004* (z=-2.847)
LMR	2.0±1.9	2.9±4.8	0.004* (z=-2.872)
NPS	3.6±0.6	3.21±0.97	0.003* (z=-2.956)

Data are mean±SD and Median (IQR: 25th – 75th percentiles);
z: Mann Whitney U test; t: Independent samples t test;
*Significant difference, Yes vs No. WBC, white blood cell;
HMG, hemogram; RDW. red cell distribution width;
GFR, glomerular filtration rate; AST, aspartate aminotransferase;
CRP, C-reactive protein; PLR, platelet-lymphocyte ratio;
PLR, neutrophil-lymphocyte ratio; LMR, lymphocyte-monocyte ratio; NPS; Naples prognostic score.

Results

The patients were divided into two groups: Group 1, patients who died in-hospital (n=116; 23.3%); Group 2, patients alive at the end of the follow-up period (n=380; 76.7%), Table 1. The mean age was 70.8±10.2 yrs in Group 1 and 68.5±11.8 yrs in Group 2. 72 (p=0.128) patients in Group 1 and 239 patients in Group 2 were male. 298 patients had ischemic HF. 65 (%56.03) of these patients are in Group 1. The mean LVEF values of the patients were 36.65±12.33% in Group 1 and 37.96±11.65% in Group 2. There was no significant difference between the percentage of Group 1 vs Group 2 in terms of HT (57; 49.1%, 191; 50.2%, p=0.944), DM (43; 37.06%, 185; 48.6%, p=0.103), smoking (28; 24.1%, 102; 26.8%, p=063), CRF (28; 24.1%, 78; 20.5%, p=0422), and medications. However, there were significant differences in Group 1 vs Group 2 for systolic (106.03±21.93 mmHg, 117.45±24.09 mmHg, p=0.0001) and diastolic (64.97±10.94 mmHg, 68.65±11.67 mmHg, p=0.003) blood pressure, Table 1. When evaluated in terms of percentages of Group 1 and Group 2 patients receiving optimal medical treatment, there were no significant differences between the two groups: beta blocker, 77 (66.3%), 261 (68.6%), p=0.693; angiotensin converting enzyme inhibitor/angiotensin receptor blocker, 35 (30.1%), 152 (40%), p=0.137; Spironolactone: 36 (31%), 124 (32.6%), p=0.871; diuretic, 81 (69.8%), 226 (59.4%), p=0.108).

Creatinine (1.73±0.89 vs 1.39±0.69, p=0.0001), red blood cell distribution width (RDW) (17.41±2.65 vs 16.26±2.56, p=0.0001), CRP (5.29±5.82 vs 4.59±6.46, p=0.017), NLR (10.07±11.52 vs 7.86±8.17, p=0.004), and NPS (3.6±0.61 vs 3.21±0.97, p=0.003) were found to be higher in Group 1, the deceased group, Table 2. Glomerular filtration rate GFR (52.13±17.54 vs 60.4±23.8, p=0.001), sodium (134.9±6.84 vs 136.84±5.28, p=0.02), total cholesterol (125.77±33.44 vs 143.34±42.74, p=0.001), albumin (3.43±0.57 vs 3.63±0.6, p=0.007), and LMR (2.03±1.9 vs 2.9±4.76, p=0.004) were lower in Group 1, Table 2.

Table 3 shows the results of the regression analysis. According to multivariate regression analysis; high NPS (OR: 1.546, 95% CI: 1.027–2.327; p=0.037), low systolic blood pressure (OR: 0.976, 95% CI: 0.957–0.995; p=0.015) and high white blood cell count (OR: 1.072, 95% CI: 1.007–1142; p=0.03) are independent predictors of in-hospital mortality in HF patients. As a result of the ROC analysis for the probability values obtained with the multivariate model in Table 3, the AUC value was 73.7% (p=0.0001; 95% CI: 0.676–0.798).

Discussion

The results of this study showed that NPS was a strong predictor of in-hospital mortality in HF. The NPS is a new scoring system that evaluates inflammation and malnutrition together. The NPS was first described by Galizia G et al [14]. It was also studied to evaluate the prognosis of cancer patients, such as those with gastric and esophageal squamous cell carcinoma. A study conducted in 2021 showed that NPS can be used to independently predict the survival of gastric cancer in cases that have undergone surgery [15]. Another study showed that NPS is a useful independent prognostic score for patients with resected esophageal squamous cell carcinoma [16]. However, this score has not been previously studied in the cardiovascular area or, specifically, for HF. Malnutrition and inflammation have a pivotal role at HF. We thought that this scoring system would be very useful at evaluating the prognosis of HF, since it simultaneously

Table 3. Univariate and multivariate regression analyses

	Variable	p value	OR	95% CI for OR	
				Lower Limit	Upper Limit
Univariate regression analysis	Age	0.136	1.018	0.994	1.041
	DM	0.104	0.648	0.385	1.093
	HT	0.944	0.982	0.59	1.635
	HL	0.378	1.385	0.671	2.856
	Smoking	0.63	0.866	0.482	1.556
	CAD	0.516	0.843	0.504	1.411
	CRF	0.422	1.279	0.701	2.333
	Stroke	0.992	0.993	0.266	3.704
	Respiratory Failure	0.54	1.227	0.638	2.359
	Malignancy	0.376	1.573	0.577	4.288
	LVEF	0.391	0.99	0.969	1.012
	Systolic blood pressure	0.0001*	0.977	0.965	0.99
	Diastolic blood pressure	0.016*	0.971	0.948	0.994
	WBC	0.032*	1.067	1.006	1.131
	HMG	0.545	0.962	0.849	1.09
	RDW	0.001*	1.172	1.066	1.289
	Glucose	0.763	1	0.996	1.003
	Creatine	0.001*	1.705	1.244	2.337
	GFR	0.006*	0.983	0.971	0.995
	Sodium	0.01*	0.945	0.904	0.987
	Potassium	0.25	1.241	0.859	1.791
	AST	0.061	1.005	1	1.011
	CRP	0.394	1.017	0.979	1.056
	Uric Acid	0.065	1.07	0.996	1.15
	Beta blocker	0.693	0.897	0,522	1,541
	ACEI/ARB	0.138	0.66	0,381	1,143
	Spironolactone	0.871	0.955	0,552	1,655
	Diuretic	0.109	1.565	0,904	2,709
	NPS	0.002*	1.793	1.248	2.577
Multivariate regression analysis	Systolic blood pressure	0.015*	0.976	0.957	0.995
	Diastolic blood pressure	0.346	1.018	0.981	1.057
	WBC	0.03*	1.072	1.007	1.142
	RDW	0.14	1.086	0.973	1.213
	Creatine	0.111	1.523	0.907	2.557
	GFR	0.871	1.002	0.982	1.021
	Sodium	0.253	0.973	0.928	1.02
	NPS	0.037*	1.546	1.027	2.327

* Significant effect. OR, odds ratio; 95% CI, 95% confidence interval; DM, diabetes mellitus; HT, hypertension; HL, hyperlipidemia; CAD, coronary artery disease; CRF, chronic renal failure; LVEF, left ventricular ejection fraction; WBC, white blood cell; HMG, hemogram; RDW, red cell distribution width; GFR, glomerular filtration rate; AST, aspartate aminotransferase; CRP, C-reactive protein; ACEI/ARB, angiotensin converting enzyme inhibitor / angiotensin receptor blocker; NPS, Naples prognostic score.

considers malnutrition and inflammation. In the results we obtained, the prediction of in-hospital mortality of NPS was quite strong.

HF is a chronic disease. The mortality rate is high, and it is important to determine the prognostic factors. Inflammation and malnutrition are two main factors affecting prognosis. Inflammation plays an important role in both the pathogenesis, progression, and poor outcome of HF [4, 16]. The humoral immune system and inflammatory biomarkers activated during inflammation cause cardiac remodeling and worsen cardiac systolic and diastolic function. Depending on the immune system activation, changes occur in the number of neutrophils, lymphocytes, monocytes and platelets, and these changes become evident as heart failure progresses. The commonality in the pathophysiology of both ischemic and non-ischemic HF is the correlation between increased serum markers of inflammation and adverse clinical outcome [4]. The most commonly used inflammation biomarkers are the NLR, the LMR, and the platelet-lymphocyte ratio. Many previous studies have shown that a high NLR is associated with high mortality in both coronary artery disease and HF [17–19]. In a study of patients with decompensated HF, a high NLR was found to be correlated with a poor prognosis [19]. A 2021 study that included 1701 HF patients found that a low LMR was associated with high long-term mortality [20]. In the current study, the NLR was higher in the deceased group (Group 1), and the LMR is lower in the deceased group. Our findings agree with the literature in this respect.

There are studies showing that malnutrition is a poor prognostic factor for HF [21]. In advanced HF, increased anorexia with increased catabolism predominates. This, in turn, leads to an imbalance of carbohydrates, fats, and proteins that negatively affects the prognosis [22]. In many studies, the importance of malnutrition, hypoalbuminemia, low cholesterol, and cachexia has been shown in the prognosis of HF [23]. In a study evaluating 1673 HF patients, the prognostic nutritional index was shown to be independently associated with long-term survival in patients hospitalized for decompensated HF [24]. In another study, a high controlling nutritional status (CONUT) (low CONUT score 0–4 points, high CONUT score 5–9 points) score was found to be associated with in-hospital mortality in hospitalized patients with acute decompensated HF [25]. In our study, albumin and total cholesterol values were lower in the deceased group. Our findings agree with the literature in this respect, and they agree with another study showing that mortality was high in HF patients with low blood pressure and with high white blood cell count [26].

Conclusion

This study is the first to compare the relationship between NPS and mortality in HF. The findings showed a strong correlation between NPS and mortality in hospitalized HF patients. Thus, NPS can be used to predict HF prognosis and mortality.

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Limitations

The current study has certain limitations. First, it was a single-center study that included a relatively small number of patients. Second, some patients were excluded because of missing clinical data and/or laboratory variables. Finally, the presence of multiple comorbidities, frailty, and the low rate of optimal medical treatment of the patients may have affected the in-hospital mortality rate.

No conflict of interest is reported.

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