RESEARCH ARTICLE

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The Naples Prognostic Score as a Predictive Tool for Saphenous Vein Graft Disease in Post-coronary Artery Bypass Grafting Patients

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Abstract

Background and Aim: Coronary artery bypass grafting (CABG) remains the gold standard for managing complex coronary artery disease. However, saphenous vein graft disease (SVGD) significantly undermines long-term graft patency, with up to 50% of grafts failing within 10 years. Chronic inflammation, oxidative stress, and nutritional deficiencies are central to SVGD pathophysiology, underscoring the need for comprehensive predictive tools. This study evaluates the Naples Prognostic Score (NPS), a composite index of inflammatory and nutritional markers, as a predictor of SVGD.

Materials and Methods: A retrospective analysis was conducted on 514 patients who underwent CABG and follow-up angiography between 2019 and 2022. Patients were categorized into SVGD (n=252) and the control (n=197) groups based on significant stenosis (≥50%) in at least one saphenous vein graft. NPS was calculated using albumin levels, lymphocyte-monocyte ratio, neutrophil-lymphocyte ratio, and cholesterol parameters. Logistic regression and receiver operating characteristic (ROC) curve analyses were performed to evaluate NPS as an independent predictor of SVGD.

Results: The SVGD group demonstrated significantly higher rates of diabetes (59.4% vs. 49.2%, p=0.033), smoking (41.2% vs. 29.3%, p=0.011), and chronic kidney disease (27% vs. 17.8%, p=0.021). NPS stratification revealed a higher prevalence of high-risk patients (NPS Group 3: 42.5% vs. 29.6%; p=0.052) in the SVGD cohort. Multivariate regression identified smoking [Odds ratios (OR)=3.02; p=0.001], graft age (OR=1.07; p=0.011), albumin levels (OR=0.91; p<0.001), and NPS (OR=1.27; p=0.023) as independent predictors of SVGD. ROC analysis demonstrated strong predictive accuracy for NPS, supporting its clinical applicability.

Conclusion: NPS is a robust, independent predictor of SVGD, integrating systemic inflammatory and nutritional parameters to enhance risk stratification. Its adoption in clinical workflows may guide targeted therapeutic interventions and improve graft patency outcomes. Further prospective studies are warranted to validate its utility across diverse populations and optimize long-term CABG success.

Keywords: Coronary artery bypass grafting (CABG), saphenous vein graft disease (SVGD), Naples prognostic score (NPS), inflammation

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INTRODUCTION

Coronary artery disease (CAD) remains one of the leading causes of morbidity and mortality worldwide. Coronary artery bypass grafting (CABG) is the gold standard for the management of complex CAD, particularly in patients with multivessel disease or left main coronary artery stenosis. Despite the success of CABG, saphenous vein grafts (SVGs), which are commonly used as conduits, demonstrate high rates of disease and failure over time. Studies indicate that SVG patency decreases by 3-12% in the first postoperative months and up to 50% within 10 years due to severe stenosis or occlusion.[1]

The pathophysiology of saphenous vein graft disease (SVGD) is multifactorial, encompassing thrombotic occlusion, intimal hyperplasia, and accelerated atherosclerosis.[2] Early failure, often within the first month, is primarily attributed to thrombosis, while intermediate failure results from intimal hyperplasia compromising luminal flow dynamics. Long-term occlusion is frequently driven by progressive atherosclerosis within the graft. Chronic inflammation is a key contributor to these processes, with endothelial dysfunction, oxidative stress, and the release of pro-inflammatory cytokines exacerbating graft deterioration.[2,3]

Established risk factors for SVGD include smoking, diabetes mellitus, hypertension, and dyslipidemia.[2,4] These factors enhance inflammation and oxidative stress, further impairing endothelial function and accelerating disease progression. Efforts to mitigate SVGD involve both surgical and medical strategies, including optimal conduit selection, surgical technique improvements, anti-thrombotic therapy, and statin use. Lifestyle modifications, such as smoking cessation and dietary changes, also play a critical role.[2,5]

In this study, we evaluate the Naples Prognostic Score (NPS) as a potential tool for predicting SVGD. NPS integrates inflammatory and nutritional status markers, offering a holistic view of the underlying pathophysiology. This study explores whether NPS could be an effective predictor of SVGD and a practical tool in clinical decision-making.

METHODS

This retrospective study analyzed data from 514 patients who underwent CABG and subsequent coronary angiography at two centers between 2019 and 2022. Patients were categorized into two groups: those with significant stenosis (≥50%) in at least one SVG beyond the anastomotic site (SVGD group) and those without significant stenosis (control group).

Ethics approval for the study was granted by Health Sciences University Türkiye, Hamidiye Scientific Research Ethics Board on February 24, 2023, ethics approval no.: 2023/4-4/5.

Patients with a minimum of one year of follow-up post-CABG were included. Patients with acute coronary syndrome, active cancer, decompensated heart failure, rheumatological diseases, or a history of pulmonary embolism were excluded from the study.

Demographic data, medical history, and laboratory parameters were retrieved from hospital records. NPS was calculated using parameters outlined in Table 1, including albumin levels, lymphocyte-monocyte ratio (LMR), neutrophil-lymphocyte ratio (NLR), and cholesterol levels.

Statistical analyses were performed using SPSS version 27.0. Normality was assessed using histograms and the Kolmogorov-Smirnov test. Continuous variables were expressed as mean ± standard deviation or median with interquartile range (IQR) based on distribution. Group comparisons employed independent t-tests or Mann-Whitney U tests for continuous data and chi-square tests for categorical variables. Receiver operating characteristic curves determined the sensitivity and specificity of predictors. Variables with $p<0.2$ in univariate analyses were included in multivariate logistic regression, with results expressed as Odds ratio (OR) and 95% confidence intervals. Statistical significance was defined as $p<0.05$.

RESULTS

A total of 514 patients were included in this study, providing a robust dataset for analysis. The demographic and clinical data are summarized comprehensively in Table 2. Of the study population, the SVGD group comprised 252 patients, while the control group included 197 individuals without significant SVGD.

Key differences between the groups were observed in the prevalence of comorbidities and lifestyle factors. Diabetes mellitus was significantly more prevalent in the SVGD group (59.4%) compared to the control group (49.2%; p=0.033). Smoking rates were also notably higher in the SVGD group at

Table 1. Calculation of Naples prognostic score

Variables	Cut-off value	Points	NPS group
Serum albumin (mg/dL)	≥4	0	Group 1: 0 point
	<4	1	Group 2: 1 or 2 points
Total cholesterol (mg/dL)	>180	0	Group 3: 3 or 4 points
	≤180	1	
NLR	≤2.96	0	
	>2.96	1	
LMR	>4.44	0	
	≤4.44	1	

LMR: Lymphocyte monocyte ratio, NLR: Neutrophil lymphocyte ratio, NPS: Naples prognostic score

41.2% compared to 29.3% in the control group (p=0.011). Chronic kidney disease was present in 27% of SVGD patients, compared to 17.8% in the control group (p=0.021). There were no significant differences between groups in terms of hypertension, peripheral arterial disease, or cerebrovascular accident prevalence.

Analysis of medication usage revealed distinct patterns. Statin therapy was more common among SVGD patients (67.1%) compared to the control group (55.8%; p=0.015). Similarly, spironolactone use was higher in the SVGD group (19.4%) versus the control group (10.2%, p=0.007). Angiotensin converting enzyme inhibitors were used by 66.3% of the SVGD group compared to 53.3% in the control group (p=0.005). The use of beta blockers, calcium channel blockers, and diuretics showed varying degrees of statistical difference, with diuretic use significantly higher in the SVGD group (21% vs. 13.7%; p=0.044).

Laboratory markers provided additional distinctions. Table 3 summarizes the laboratory findings of the groups. The LMR was significantly lower in the SVGD group (median: 0.8286; p=0.002). However, no significant differences were noted for the NLR between the groups (p=0.737). Total cholesterol, high density lipoprotein, low density lipoprotein, and triglyceride levels showed no statistically significant differences, suggesting lipid profiles were similar across groups.

The median age of SVGs was higher in the SVGD group (8 years; IQR: 4-13) compared to the control group (5 years; IQR: 4-10; p=0.005). The number of grafts was also greater in the SVGD group, with a median of 2 grafts (IQR: 2-3) compared to the control group, which had a median of 2 grafts (IQR: 1-2) (p<0.001).

Table 4 demonstrates the number of grafts to each coronary artery. The patients without a SVG implant to the left anterior descending artery had left internal mammary artery anastomoses instead. Table 4 also demonstrates the diseased grafts and the coronary arteries to which they are anastomosed.

When stratified by NPS, the distribution differed significantly. The SVGD group had a higher proportion of patients in Naples group 3 (42.5%) compared to the control group (29.6%; p=0.052).

Results of the regression analyses were listed in Table 5. Univariate regression analysis identified several predictors of SVGD, including diabetes mellitus [OR=1.5060; p=0.0329],

Table 2. Demographic and clinical data of the study population

	SVGD (252)	Control group[a] (197)	P-value
Male gender, n (%)	213 (84.5%)	167 (84.8%)	0.942
Age, years (mean ± SD)*	68 (62-75)	68 (62-75)	0.228
Hypertension, n (%)	218 (86.5%)	162 (82.2%)	0.213
Diabetes mellitus, n (%)	149 (59.4%)	97 (49.2%)	0.033
Hyperlipidemia, n (%)	174 (69.3%)	125 (63.5%)	0.190
Previous percutaneous treatment of coronary atherosclerotic disease	36 (14.2%)	26 (13.1%)	0.740
Peripheral arterial disease, n (%)	45 (17.9%)	27 (13.7%)	0.234
Chronic kidney disease, n (%)	68 (27%)	35 (17.8%)	0.021
Cerebrovascular accident, n (%)	24 (9.5%)	14 (7.1%)	0.361
Smoking, n (%)	96 (41.2%)	55 (29.3%)	0.011
Medical treatment, n (%)			
Anti-aggregant	232 (92.1%)	182 (92.4%)	0.899
Anti-coagulant	33 (131%)	20 (10.2%)	0.338
Beta blocker	203 (80.6%)	152 (77.2%)	0.380
ACE/ARB	167 (66.3%)	105 (53.3%)	0.005
Spironolactone	49 (19.4%)	20 (10.2%)	0.007
Statin	169 (67.1%)	110 (55.8%)	0.015
Calcium channel blocker	61 (24.2%)	43 (21.8%)	0.553
Alpha blocker	12 (4.8%)	9 (4.6%)	0.923
Diuretics	53 (21%)	27 (13.7%)	0.044

*Chi-square test was used for parameters with parametric distribution and Mann-Whitney U test was used for parameters non-parametric distributions

[a]Patients with patent saphenous vein grafts without disease.

ACE/ARB: Angiotensin converting enzyme/angiotensin receptor blocker, SVGD: Saphenous vein graft disease, SD: Standard deviation

Table 3. Laboratory parameters and inflammatory indices			
	SVGD (252)	**Control group (197)**	**P-value**
Age of saphenous vein graft (median, IQR)	8 (4-13)	5 (4-10)	**0.005**
Saphenous graft (median, IQR)	2 (2-3)	2 (1-2)	**<0.001**
Hemoglobin, mg/dL (median, IQR)	13 (11.5-14.7)	13.6 (12-14.9)	0.077
WBC, x 1000 µL (median, IQR)	7.8 (6.6-9.5)	7.6 (6.3-9)	0.805
Platelet, x 1000 µL (median, IQR)	217 (176-265)	218 (179-253)	0.675
Total cholesterol, mg/dL (median, IQR)	165 (140-199)	176 (140-202)	0.382
LDL, mg/dL (median, IQR)	94 (75-120)	101 (75-127)	0.536
HDL, mg/dL (median, IQR)	42 (35-49)	42 (37-50)	0.440
Triglyceride mg/dL (median, IQR)	132 (92-198)	136 (96-175)	0.984
Albumin g/L (median, IQR)	40.8 (37-44)	41 (35-44)	0.264
Lymphocyte-monocyte ratio (median, IQR)	3.4 (2.26-4.6)	3.87 (2.77-5.2)	**0.006**
Neutrophil-lymphocyte ratio (median, IQR)	2.42 (1.9-3.5)	2.4 (1.8-3.3)	0.737
Triglyceride/HDL (median, IQR)	3.1 (2.1-5)	3.3 (2-4.7)	0.789
SII index (median, IQR)	524 (355-765)	492 (365-764)	0.452
Naples group, n (%)			0.052
Group 1	28 (19.2%)	27 (23.5%)	
Group 2	56 (38.4%)	54 (47%)	
Group 3	62 (42.5%)	34 (29.6%)	
Naples score, n (%)	2 (1-3)	2 (1-3)	**0.020**

HDL: High density lipoprotein, LDL: Low density lipoprotein, SII: Systemic inflammation index, SVGD: Saphenous vein disease, WBC: White blood cell, IQR: Interquartile range

Table 4. Distribution of saphenous grafts		
Distribution of saphenous grafts	**SVGD (n=252)**	**Control (n=197)**
LAD	121 (48.4%)	109 (55.3%)
CX	103 (40.8%)	127 (64.4%)
RCA	105 (41.6%)	104 (52.7%)
SVGD distribution		
LAD	113 (44.8%)	
CX	87 (34.5%)	
RCA	95 (37.6%)	

LAD: Left anterior descending artery, CX: Left circumflex artery, RCA: Right coronary artery, SVGD: Saphenous vein graft disease

chronic kidney disease (OR=1.7106; p=0.0219), and smoking (OR=1.6945; p=0.0114). In multivariate regression, key independent predictors were identified: Smoking (OR=3.0163; p=0.0013), Age of the saphenous vein graft (OR=1.0656; p=0.0111), albumin levels (OR=0.9143; p<0.0001) NPS (OR=1.2733; p=0.0228)

DISCUSSION

This study highlights the NPS as a robust independent predictor of SVGD, showcasing its relevance in clinical settings where long-term graft patency is critical. NPS, through its unique integration of inflammatory and nutritional biomarkers, offers a nuanced understanding of the systemic factors that drive graft failure. By combining parameters such as albumin levels, LMR, NLR, and cholesterol levels, the score provides a holistic snapshot of a patient's physiological state, which is directly relevant to graft health.[6,7]

The inclusion of inflammation as a cornerstone in NPS's design reflects the critical role of systemic inflammatory processes in the pathophysiology of SVGD.[2,4] Inflammation contributes to endothelial dysfunction, intimal hyperplasia, and accelerated atherosclerosis, all of which compromise graft integrity.[2,5] Moreover, the nutritional markers embedded within the NPS framework, such as albumin levels, underscore the interplay between systemic health and localized vascular responses.

This comprehensive approach makes NPS an invaluable tool in the identification of patients at higher risk for SVGD. The ability of NPS to stratify patients based on both inflammatory burden and nutritional deficits allows clinicians to tailor postoperative management strategies. For example, patients with elevated NPS values may benefit from more aggressive anti-inflammatory therapies, nutritional supplementation, or intensified monitoring protocols. Beyond its predictive capacity, NPS serves as a potential guide for optimizing therapeutic interventions and improving clinical outcomes.

The versatility of NPS is further demonstrated by its applicability across diverse patient populations. While this study primarily evaluates its role in SVGD, the broader implications of NPS

Table 5. Regression analyses

	Univariate regression			Multivariate regression		
	OR	**%95 CI**	**P-value**	**OR**	**%95 CI**	**P-value**
Diabetes mellitus	1.5060	1.0339-2.1936	**0.0329**			
Chronic kidney disease	1.7106	1.0807-2.7074	**0.0219**			
Smoking	1.6945	1.1264-2.5492	**0.0114**	3.0163	1.5385-5.9135	**0.0013**
ACE/ARB	1.7215	1.1739-2.5243	**0.0054**			
Spironolactone	2.1362	1.2229-3.7315	**0.0076**			
Statin	1.6104	1.0960-2.3663	**0.0152**			
Age of saphenous vein graft (mean ± SD)	1.0454	1.0139-1.0778	**0.0044**	1.0656	1.0146-1.1191	**0.0111**
Saphenous graft (mean ± SD)	1.5343	1.322-2.3643	**0.0122**			
Albumin g/L (median, IQR)	0.9239	0.9041-0.9441	**<0.0001**	0.9143	0.8913-0.9379	**<0.0001**
Lymphocyte-monocyte ratio (median, IQR)	0.8286	0.7356-0.9333	**0.0020**			
Naples group, n (%)	1.2925	0.9389-1.7792	**0.1156**	0.3604	0.1396-0.9303	**0.0349**
Naples score, n (%)	1.2733	1.0342-1.5677	**0.0228**			

CI: Confidence interval, OR: Odds ratio, ACE/ARB: Angiotensin converting enzyme/angiotensin receptor blocker, SD: Standard deviation, LMR: Lymphocyte-monocyte ratio

in cardiovascular and systemic disease management suggest that it could be integrated into routine clinical workflows. The predictive power of NPS, validated in this and other studies, supports its use not only as a risk stratification tool but also as a marker for treatment efficacy in managing graft health.

Inflammatory processes play a pivotal role in graft disease, particularly through mechanisms such as intimal hyperplasia and atherosclerosis. Low albumin levels, a component of NPS, are associated with heightened inflammatory states and poor clinical outcomes. Our findings align with prior research demonstrating the negative prognostic implications of hypoalbuminemia in cardiovascular disease.[7,8]

Reduced LMR in the SVGD group reflects an imbalance between anti-inflammatory lymphocytes and pro-inflammatory monocytes,[9] underscoring the systemic inflammatory milieu associated with graft failure. Similar trends have been observed in other cardiovascular and vascular pathologies, suggesting LMR's utility as a prognostic marker.[10]

Smoking emerged as a strong independent predictor of SVGD, consistent with its established role in endothelial dysfunction, oxidative stress, and systemic inflammation. Smoking cessation remains a critical component of postoperative management to improve graft patency.

Graft age was another significant determinant, with older grafts showing increased vulnerability to intimal hyperplasia and atherosclerosis. This finding underscores the importance of optimizing graft selection and surgical techniques to enhance long-term outcomes. Arterial grafts, known for superior patency rates, should be prioritized where feasible.

Diabetes mellitus and chronic kidney disease further exacerbate SVGD risk by promoting systemic inflammation and endothelial dysfunction. Enhanced surveillance and targeted interventions for these high-risk populations are imperative.

NPS offers a novel approach to risk stratification, combining multiple inflammatory and nutritional markers.[11,12] Its predictive value in SVGD aligns with prior evidence supporting its utility in various clinical settings. Future studies should explore its application in larger, diverse populations and compare its performance to other prognostic tools.

Beyond confirming the predictive validity of NPS, prospective studies should investigate its role in guiding therapeutic strategies. For instance, higher-risk individuals identified via NPS may benefit from intensified medical therapy or closer surveillance. Additionally, the integration of advanced biomarkers with NPS could enhance the discriminatory power of NPS.

Study Limitations

Several limitations of this study warrant consideration. First, its retrospective design may introduce selection bias and limit the ability to establish causality. Despite efforts to retrieve comprehensive data from medical records, the potential for incomplete or missing documentation remains. Second, the study population was drawn from two centers, which may affect the generalizability of the findings to broader or more diverse patient groups. Third, although we included patients with a minimum of one year of postoperative follow-up, the duration of follow-up varied among participants, potentially influencing graft outcome assessments. Moreover, changes in medical therapy or lifestyle factors over time could not be

thoroughly captured in a retrospective framework, potentially impacting the progression of SVGD.

An important limitation of our study is the lack of echocardiographic data as it was not commonly recorded in the database we acquired our data from. A higher NPS is associated with a worse left ventricular function[12]; and this should be kept in mind when interpreting NPS results.

Additionally, while the NPS was shown to be a useful predictor, the cross-sectional measurement of its components (e.g., albumin levels, LMR) does not fully account for longitudinal fluctuations in nutritional or inflammatory status. Further, we did not include other emerging biomarkers of inflammation and oxidative stress, which might yield deeper insights into SVGD pathophysiology. Finally, as this study focused solely on angiographically evident SVG stenosis, noninvasive imaging or functional assessments were not performed, and could provide valuable complementary information in future research.

Higher NPS is associated with an increased risk of vein graft occlusion due to persistent inflammation and endothelial dysfunction. Patients with high NPS may benefit from aggressive lipid-lowering therapy (statins), anti-inflammatory strategies, and close follow-up after CABG.

These limitations highlight the need for prospective, multicenter studies with standardized follow-up protocols and more extensive biomarker profiling to validate and refine the prognostic utility of NPS for SVGD.

CONCLUSIONS

This study demonstrates that the NPS is an independent and robust predictor of SVGD, reflecting the interplay between systemic inflammation and nutritional status. Smoking and graft age also emerged as notable contributors to graft failure risk. These findings emphasize the potential value of incorporating NPS into clinical risk stratification and targeted therapeutic strategies, although further prospective research is warranted to confirm its utility and optimize long-term patient outcomes.

Ethics

Ethics Committee Approval: This study was approved by the Health Sciences University Hamidiye Scientific Research Ethics Board on February 24, 2023, ethics approval no.: 2023/4-4/5.

Informed Consent: As this was a retrospective study, informed consent was waived.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Ş.K., S.A., M.Ş., S. Dil, S.D., D.M., A.L.O., Concept: Ş.K., S.A., A.T., S.D., Design: S.A., A.T., M.Ş., A.L.O., Data Collection or Processing: Ş.K., M.Ş., S. Dil, S.Y., D.M., Analysis or Interpretation: Ş.K., S.A., M.Ş., S.Y., Literature Search: Ş.K., S.A., S. Dil, S.Y., A.L.O., Writing: Ş.K., S.A., A.L.O.

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