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Under Smoke: The Impact of Smoking on Inflammatory Indices in Patients with Saphenous Vein Graft Degeneration: A Retrospective Study

Duman Altında Kalan Greftler: Sigara Kullanımının Safen Ven Greft Dejenerasyonu Olan Hastalarda Enflamatuvar İndeksler Üzerine Etkisi: Retrospektif Bir Çalışma

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Abstract

Objective: The goal of this research was to examine how cigarette use influences circulating inflammation indicators among individuals experiencing saphenous vein graft deterioration (SVGD) following coronary artery bypass grafting procedures.

Material and Methods: A total of 213 patients diagnosed with SVGD between 2017 and 2023 were retrospectively reviewed. Based on smoking status, the cohort was divided into two groups: smokers (n=131) and non-smokers (n=82). Hematological and biochemical data were collected, and several inflammatory indices were calculated, including neutrophil-to-lymphocyte ratio (NLR), lymphocyte-to-monocyte ratio, systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), pan-immune-inflammation value (P-IV), atherogenic index of plasma (AIP), and prognostic nutritional index. Comparisons between groups were performed using appropriate statistical methods. Additionally, multivariate logistic regression analysis was conducted to identify independent predictors of smoking among patients with SVGD.

Results: Smokers demonstrated significantly elevated inflammatory markers, including NLR (p=0.027), SII (p=0.002), SIRI (p<0.001), P-IV (p<0.001), and AIP (p=0.048), compared to non-smokers. Regression analysis revealed that male sex [odds ratio (OR): 0.296, p=0.024], younger age (OR: 0.945, p=0.023), peripheral artery disease (OR: 3.61, p=0.036), and neutrophil count (OR: 1.23, p=0.045) were independently associated with smoking.

Conclusion: Smoking is significantly associated with increased systemic inflammation in patients with SVGD. These findings highlight the contribution of smoking-induced inflammatory and prothrombotic pathways to the pathogenesis and progression of graft degeneration, emphasizing the urgent need for smoking cessation in this population.

Keywords: Coronary artery bypass, saphenous vein, graft occlusion, vascular, smoking, inflammation

Öz

Amaç: Bu çalışma, koroner arter bypass greftleme cerrahisi sonrası safen ven greft dejenerasyonu (SVGD) gelişen hastalarda sigara kullanımının sistemik enflamatuvar belirteçler üzerindeki etkisini araştırmayı amaçladı.

Yöntem ve Gereçler: 2017 ile 2023 yılları arasında hastanemizde SVGD tanısı alan toplam 213 hasta retrospektif olarak incelendi. Hastalar sigara kullanma durumlarına göre iki gruba ayrıldı: sigara içenler (n=131) ve içmeyenler (n=82). Her iki gruptan hematolojik ve biyokimyasal

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veriler toplandı, ayrıca nötrofil/lenfosit oranı (NLR), lenfosit/monosit oranı, sistemik immün-enflamasyon indeksi (SII), sistemik enflamasyon yanıt indeksi (SIRI), pan-immün-enflamasyon değeri (P-IV), plazma aterojenik indeksi (AIP) ve prognostik nutrisyonel indeks gibi çeşitli enflamatuvar indeksler hesaplandı. Gruplar arasındaki farklar uygun istatistiksel yöntemlerle karşılaştırıldı. Ayrıca, SVGD'li hastalarda sigara kullanımı ile ilişkili bağımsız değişkenleri belirlemek amacıyla çok değişkenli lojistik regresyon analizi yapıldı.

Bulgular: Sigara içen hastalarda, içmeyenlere kıyasla NLR (p=0,027), SII (p=0,002), SIRI (p<0,001), P-IV (p<0,001) ve AIP (p=0,048) gibi enflamatuvar belirteçlerin anlamlı düzeyde yüksek olduğu gözlendi. Regresyon analizinde erkek cinsiyet [odds oranı (OO): 0,296, p=0,024], daha genç yaş (OO: 0,945, p=0,023), periferik arter hastalığı varlığı (OO: 3,61, p=0,036) ve nötrofil sayısı (OO: 1,23, p=0,045) sigara kullanımıyla bağımsız olarak ilişkili bulundu.

Sonuç: Sigara kullanımı, SVGD'li hastalarda sistemik enflamasyonun artışıyla anlamlı şekilde ilişkilidir. Bu bulgular sigara içiminin neden olduğu enflamatuvar ve protrombotik yolların greft dejenerasyonunun patogenezine ve ilerlemesine olan katkısını ortaya koymakta ve bu popülasyonda sigarayı bırakmanın acil gerekliliğini vurgulamaktadır.

Anahtar Kelimeler: Koroner arter baypas, safenöz ven, gref tıkanması, vasküler, sigara içme, enflamasyon

INTRODUCTION

Coronary artery bypass grafting (CABG) surgery, a cornerstone of revascularization therapy, is widely performed for advanced coronary lesions. The saphenous vein graft (SVG), favored over arterial grafts due to ease of access and lower perioperative risk, is commonly used. However, SVGs demonstrate considerable rates of degeneration and occlusion over time, with 3-12% occluding prior to hospital discharge, 8-25% failing within the first year, and only 50-60% remaining patent after a decade (1,2).

SVG degeneration (SVGD) is a multifactorial process. Three pathophysiological mechanisms lead to SVG failure: thrombosis and technical failure predominate in the early post-CABG period (first week to first month); intimal hyperplasia occurs between one month and one year; and atherosclerosis develops after one year. SVG atherosclerosis progresses more rapidly than native coronary artery disease, often presenting as concentric, diffuse lesions that lack a fibrous cap and are prone to rupture. Endothelial dysfunction and inflammatory responses trigger these changes (1,3). Smoking exacerbates many of these factors by increasing vascular inflammation, impairing endothelial function, and creating a pro-atherogenic environment (4).

Inflammatory indices derived from hematological parameters, such as neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SII), systemic inflammatory response index (SIRI), atherogenic index of plasma (AIP), and pan-immune inflammation value (P-IV), have recently gained prominence as prognostic markers in clinical practice (5-8). However, their relationship with SVGD has been studied only in limited contexts.

This study hypothesizes that smoking markedly elevates systemic inflammation in patients with SVGD and that this effect can be objectively quantified using widely available inflammatory indices.

MATERIAL AND METHODS

Study Design and Population

We included individuals who underwent coronary angiography at our cardiology clinic from January 2017 to December 2023 and were identified as having ≥50% stenosis in at least one SVG. A total of 213 patients were divided into non-smokers (n=82) and smokers (n=131) based on smoking status. University of Health Sciences Türkiye, Hamidiye Faculty of Medicine Ethics Board approved the study (decision no: 10/20, date: 08.05.2025). The study was carried out in compliance with the ethical principles outlined in the Declaration of Helsinki.

Exclusion Criteria

Patients with active infection, malignancy, chronic inflammatory diseases, autoimmune disorders, or hematological diseases were excluded.

Definitions

SVGD was defined as ≥50% stenosis in the graft lumen outside the anastomosis site. Smoking was defined as self-reported consumption of at least one cigarette per day for the past six months.

Data Collection

Demographic data, comorbidities (hypertension, diabetes mellitus, peripheral artery disease, etc.), medical treatment histories, and laboratory results were obtained from hospital records. Hematological and biochemical parameters were derived from blood samples collected within 24 hours of coronary angiography.

Inflammatory Index Calculations:

- NLR: neutrophil count/lymphocyte count
- LMR: lymphocyte count/monocyte count

- SII: (neutrophil × platelet)/lymphocyte
- SIRI: (neutrophil × monocyte)/lymphocyte
- P-IV: (neutrophil × monocyte × platelet)/lymphocyte
- AIP: log (triglycerides/HDL)
- Prognostic nutritional index (PNI): [10 × serum albumin (g/dL)] + [0.005 × total lymphocyte count (mm³)]

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics 27. Normality was assessed using the Kolmogorov-Smirnov test. Normally distributed variables were presented as mean ± standard deviation; non-normal variables were presented as median (interquartile range). Group comparisons were performed using t-tests or Mann-Whitney U tests for continuous variables and chi-square tests for categorical variables. To identify factors associated with smoking status, variables with p-values <0.10 in the univariate analysis (age, sex, peripheral artery disease, neutrophil count, monocyte count, and white blood cell count) were entered into a backward stepwise multivariate logistic regression model. Results were reported as odds ratios (OR) with 95% confidence intervals (CI). A two-tailed p-value <0.05 was considered statistically significant.

RESULTS

Of the 213 patients included, 131 (61.5%) were active smokers and 82 (38.5%) were non-smokers. The smoker group was significantly younger (66.3±8.7 vs. 69.0±8.2 years; p=0.029) and had a higher proportion of males (90.1% vs. 79.3%; p=0.027) (Table 1).

Comorbidities and Clinical Characteristics

Peripheral artery disease (PAD) was significantly more prevalent in smokers (22.9% vs. 7.3%, p=0.003). No significant differences were observed between groups with respect to hypertension, diabetes, chronic kidney disease, or cerebrovascular disease (p>0.05) (Table 1).

Laboratory Findings

Smokers had significantly higher neutrophil counts (5.2 vs. 4.2 ×10³/μL, p<0.001), monocyte counts (0.53 vs. 0.47 ×10³/μL, p=0.001), and white blood cell counts (8.0 vs. 7.2 ×10³/μL, p=0.006). No significant differences were found in lymphocyte counts, hemoglobin, platelet counts, cholesterol, or glucose levels. Inflammatory indices NLR (2.55 vs. 2.25, p=0.027), SII (576 vs. 457, p=0.002), SIRI (1.33 vs. 0.99, p<0.001), P-IV (319 vs. 230, p<0.001), and AIP (0.52 vs. 0.45, p=0.048) were significantly elevated in smokers, whereas LMR was significantly lower (3.84 vs. 4.78, p=0.007) (Table 2).

Regression Analyses

Multivariate logistic regression identified male sex (OR: 0.2963; 95% CI: 0.1031-0.8518; p=0.024), younger age (OR: 0.9454; 95% CI: 0.9007-0.9923; p=0.023), PAD (OR: 3.61; 95% CI: 1.08-12.02; p=0.036), and neutrophil count (OR: 1.23; 95% CI: 1.00-1.52; p=0.045) as independent factors associated with smoking (Table 3).

Table 1. Demographic and clinical data of the study population

	Smoking (-) n=82	Smoking (+) n=131	p-value
Male gender, n (%)	65 (79.3%)	118 (90.1%)	**0.027**
Age, years (mean ± SD)	69±8.2	66.3±8.7	**0.029**
Hypertension, n (%)	69 (84.1%)	115 (87.8%)	0.451
Diabetes mellitus, n (%)	43 (52.4%)	85 (64.9%)	0.071
Hyperlipidemia, n (%)	53 (64.6%)	91 (69.5%)	0.463
Peripheral arterial disease, n (%)	6 (7.3%)	30 (22.9%)	**0.003**
Chronic kidney disease, n (%)	20 (24.4%)	38 (29%)	0.461
Cerebrovascular accident, n (%)	4 (4.9%)	8 (6.1%)	0.705
Medical treatment, n (%)			
Antiaggregant	76 (92.7%)	123 (93.4%)	0.729
Anticoagulant	4 (4.9%)	8 (6.1%)	0.705
Beta blocker	61 (74.4%)	92 (70.2%)	0.511
ACE/ARB	54 (65.9%)	84 (64.1%)	0.797
Spironolactone	12 (14.6%)	20 (15.3%)	0.900
Statin	69 (84.1%)	112 (85.5%)	0.788
Calcium channel blocker	43 (52.4%)	63 (48.1%)	0.537

ACE/ARB: Angiotensin converting enzyme/angiotensin receptor blocker, SD: Standard deviation

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Table 2. Laboratory parameters and inflammatory indices

	Smoking (-) n=82	Smoking (+) n=131	p-value
Age of saphenous vein graft (median, IQR)	5 (4-12)	7 (4-14)	0.189
Saphenous graft (median, IQR)	2 (2-3)	2 (1-2)	0.194
Hemoglobin, mg/dL (mean ± SD)	13.3±2.1	13.5±2.3	0.567
WBC, × 1000 µL (median, IQR)	7.2 (6.3-8.4)	8 (6.8-9.6)	0.006
Platelet, × 1000 µL (median, IQR)	218 (174-260)	236 (187-273)	0.200
Neutrophil, × 1000 µL (median, IQR)	4.2 (3.5-5.3)	5.2 (4.1-6.3)	**<0.001**
Lymphocyte, × 1000 µL (median, IQR)	2 (1.53-2.4)	2 (1.46-2.67)	0.606
Monocyte, × 1000 µL (median, IQR)	0.47 (0.35-0.58)	0.53 (0.43-0.64)	**0.001**
Total cholesterol, mg/dL (median, IQR)	159 (136-179)	164 (140-190)	0.349
LDL, mg/dL (median, IQR)	90 (77-110)	94 (74-114)	0.671
HDL, mg/dL (median, IQR)	43 (37-50)	42 (35-48)	0260
Triglyceride, mg/dL (median, IQR)	120 (80-147)	133 (93-197)	0.067
Creatinine mg/dL (mean ± SD)	1.08±0.24	1.12±0.35	0.850
Albumin, mg/L (mean ± SD)	40.2±5.3	41.1±5.9	0.263
Glucose, mg/dL (median, IQR)	135 (94-178)	132 (99-172)	0.884
Indexes (median, IQR)			
Neutrophil-lymphocyte ratio	2.25 (1.65-3.03)	2.55 (1.88-3.74)	**0.027**
Lymphocyte-monocyte ratio	4.78 (3.14-6)	3.84 (2.76-5)	**0.007**
Triglyceride/HDL	0.94 (0.69-1.20)	0.95 (0.65-1.46)	**0.294**
SII index	457 (317-644)	576 (409-874)	**0.002**
SIRI	0.99 (0.63-1.33)	1.33 (0.92-2.07)	**<0.001**
PNI index	50.6 (48.3-52.9)	50.8 (47-57)	0.333
P-IV index	230 (122-294)	319 (191-554)	**<0.001**
AIP	0.45 (0.23-0.61)	0.52 (0.38-0.68)	**0.048**

AIP: Atherogenic index of plasma, HDL: High density lipoprotein, LDL: Low density lipoprotein, P-IV: Pan-immune inflammation value, PNI: Prognostic nutritional index, SII: Systemic inflammation index, SIRI: Systemic inflammatory response index, WBC: White blood cell, IQR: Interquartile range, SD: Standard deviation

Table 3. Regression analyses

	Univariate regression			Multivariate regression		
	OR	95% CI	p-value	OR	95% CI	p-value
Male gender	0.4212	0.1925-0.9217	**0.030**	0.2963	0.1031-0.8518	**0.024**
Age, years	0.9637	0.9324-0.9961	**0.028**	0.9454	0.9007-0.9923	**0.023**
Diabetes mellitus	0.5967	0.3399-1.0473	0.072	0.5203	0.2168-1.2485	0.143
Peripheral arterial disease	3.7624	1.4909-9.4948	**0.005**	3.6101	1.0842-12.0211	**0.036**
WBC	1.1728	1.0144-1.3560	**0.031**	1.1414	0.9855-1.3218	0.077
Neutrophil	1.3447	1.1113-1.6270	**0.002**	1.2379	1.0040-1.5264	**0.045**
Monocyte	9.6863	2.0959-44.7648	**0.003**	14.863	0.0589-374.490	0.101
Triglyceride	1.0043	0.9991-1.0095	0.104			
Neutrophil-lymphocyte ratio	1.1746	0.9759-1.4138	0.088	1.0882	0.6974-1.6982	0.709
Lymphocyte-monocyte ratio	0.8864	0.7927-0.9912	**0.034**	0.8459	0.6641-1.0775	0.175

Table 3. Continued

	Univariate regression			Multivariate regression		
	OR	95% CI	p-value	OR	95% CI	p-value
SII index	1.0006	0.9999-1.0014	0.087	0.9986	0.9966-1.0007	0.196
SIRI	1.2178	0.9222-1.6082	0.164			
P-IV index	1.0007	0.9997-1.0018	0.170			
AIP	3.5391	0.9628-13.0090	**0.050**	3.8177	0.9096-16.0239	0.067

AIP: Atherogenic index of plasma, P-IV: Pan-immune inflammation value, SII: Systemic inflammation index, SIRI: Systemic inflammatory response index, WBC: White blood cell, CI: Confidence interval, OR: Odds ratio

DISCUSSION

This study evaluated the effects of smoking on systemic inflammatory indices in patients with SVGD. Our research findings indicate that individuals who smoke have significantly elevated levels of inflammatory markers, including NLR, SII, SIRI, P-IV, and AIP. Additionally, male sex, younger age, PAD, and neutrophil levels were independently associated with smoking.

The impact of smoking on systemic inflammation has been extensively documented. Smoking triggers endothelial dysfunction, leukocyte activation, oxidative stress, and pro-inflammatory cytokine release, accelerating atherosclerotic processes in the vascular wall (4,8). SII, by integrating platelet, neutrophil, and lymphocyte counts, reflects both inflammatory and thrombotic activity. Its significant elevation in smokers with SVGD highlights the prothrombotic milieu induced by smoking, which may accelerate graft atherosclerosis and plaque instability through enhanced platelet-neutrophil interactions (9).

The marked elevations in SIRI and, especially, P-IV in the smoking group are also clinically relevant. Recent studies have demonstrated the strong prognostic value of these composite indices in various acute and chronic cardiovascular conditions, including prediction of in-hospital mortality in infective endocarditis and in acute pulmonary embolism (10,11). Smokers exhibited a significantly lower lymphocyte-to-monocyte ratio (LMR) than non-smokers. The reduction in LMR reflects smoking-induced monocyte predominance and relative lymphopenia, consistent with enhanced monocyte-driven chronic inflammation and impaired adaptive immune responses. This finding further supports the pivotal role of the monocyte-macrophage pathway in smoking-related acceleration of intimal hyperplasia and atherosclerosis within SVGs.

These findings reinforce the notion that the heightened immune–inflammatory activation observed in our smoking cohort represents a systemic prothrombotic and proatherogenic state that extends beyond the graft and likely accelerates SVGD.

Although PNI did not differ significantly between smokers and non-smokers in our cohort, this index has repeatedly been shown to predict long-term mortality and adverse cardiovascular outcomes in patients with heart failure with reduced ejection fraction, even in those with implantable cardioverter-defibrillators (12). In the present study, the absence of a smoking-related difference in PNI may indicate that the malnutrition-inflammation complex is not primarily driven by smoking in the SVGD setting; nevertheless, routine nutritional assessment remains important for long-term graft patency and survival in all post-CABG patients.

AIP, indicative of a dyslipidemic profile, was significantly higher in smokers, reflecting smoking's atherogenic impact on lipid metabolism (13). AIP is a strong predictor of future cardiovascular events in CAD patients (14).

The younger age and higher proportion of males among smokers likely reflect earlier smoking initiation, higher smoking rates among men, and earlier exposure to cardiovascular risk factors (15,16). These demographic factors may influence inflammatory responses and graft degeneration.

Another notable finding was the significantly higher prevalence of PAD among smokers, suggesting systemic atherosclerosis affecting multiple vascular beds and indicating that SVGD may mirror deterioration of systemic vascular health (17).

From a clinical perspective, these low-cost, routinely available inflammatory indices (NLR, SII, SIRI, P-IV, and AIP) provide a simple, objective tool for risk stratification after CABG. Their incorporation into routine follow-up may enable early identification of smokers at highest risk of rapid graft degeneration, thereby guiding intensified smoking-cessation interventions, closer angiographic surveillance, and tailored anti-inflammatory or antiplatelet therapies. Such inflammation-based risk stratification has the potential to improve long-term graft survival and overall prognosis in this high-risk population.

Previous studies have highlighted NLR and SII as valuable markers not only in acute syndromes but also in chronic vascular diseases. Zhang et al. (18) demonstrated NLR's diagnostic and prognostic utility, and Oksuz et al. (19) reported associations between SII and graft patency post-CABG. Our results corroborate these findings. Strengths of this study include verification of

smoking status through medical records and follow-up rather than sole reliance on patient self-report, and a comprehensive evaluation of inflammatory indices.

Study Limitations

Limitations include the retrospective design, potential selection bias, lack of quantitative smoking exposure assessment (pack-years), and SVGD diagnosis based solely on coronary angiography without advanced imaging modalities such as IVUS or OCT. The single-center setting may limit generalizability.

CONCLUSION

In conclusion, smoking significantly elevates inflammatory markers in patients with SVGD, indicating that SVGD reflects not only localized graft disease but also a systemic inflammatory response. Smoking cessation is critical not only to prevent new lesions but also to preserve existing graft patency.

This study provides clinicians with important insights, emphasizing the need for close monitoring of inflammatory biomarkers in smokers with SVGD. Incorporating these indices into routine clinical practice may guide prediction of graft prognosis and personalization of treatment strategies.

Ultimately, the detrimental effects of smoking on graft survival are quantitatively demonstrated via inflammatory indices, underscoring that smoking cessation is not merely a lifestyle choice but a therapeutic necessity to maintain graft patency.

*Ethics

Ethics Committee Approval: University of Health Sciences Türkiye, Hamidiye Faculty of Medicine Ethics Board approved the study (decision no: 10/20, date: 08.05.2025).

Informed Consent: Retrospective study.

Footnotes

Authorship Contributions

Concept: Ş.K., M.B., S.Y., A.L.O., Design: S.A., M.Ş., Data Collection or Processing: M.B., S.Y., Analysis or Interpretation: Ş.K., S.A., M.Ş., M.U., A.L.O., Literature Search: M.Ş., M.U., Writing: Ş.K., S.A.

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