Cardiovasc Surg Int 2025;12(3):223-232
http://dx.doi.org/DOI: 10.5606/e-cvsi.2025.1990
www.e-cvsi.org

Cardiovascular Surgery and Interventions

Original Article Open Access

Osaka Prognostic Score for predicting mortality after iliac artery revascularization

Onur Erdoğan[1], Tuğba Erdoğan[2], Samet Sevinç[1], Ömer Taşbulak[1], Cafer Panç[1], Ömer Çelik[1], Ahmet Arif Yalçın[1]

[1]Department of Cardiology, İstanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, İstanbul, Türkiye
[2]Department of Internal Medicine, Division of Geriatrics, İstanbul University İstanbul Faculty of Medicine, İstanbul, Türkiye

Received: August 08, 2025 ***Accepted:*** September 11, 2025 ***Published online:*** October 21, 2025

ABSTRACT

Objectives: This study aims to investigate the utility of Osaka Prognostic Score (OPS) in predicting all-cause mortality in patients undergoing endovascular treatment (EVT) for iliac artery stenosis.

Patients and methods: Between January 2015 and December 2020, a total of 183 patients (165 males, 18 females; mean age: 61.0±8.8 years; range, 36 to 92 years) who underwent EVT for iliac artery stenosis were retrospectively analyzed. The patients were categorized into four OPS groups (0-3). Mortality outcomes were confirmed using national death records. The OPS performance was compared with the Glasgow Prognostic Score (GPS) and the Systemic Inflammation Score (SIS) using receiver operating characteristic (ROC) analysis. Multivariate logistic regression was used to assess independent predictors of mortality.

Results: Overall mortality was 26.8%. Higher OPS scores were significantly associated with increased mortality (OPS 0: 14.1%, OPS 3: 60%, p<0.001). The OPS remained an independent predictor of all-cause mortality (odds ratio [OR]=3.452, 95% confidence interval [CI]: 1.342-8.878, p=0.010). The OPS ≥2 was also associated with higher rates of myocardial infarction and restenosis. The OPS demonstrated superior prognostic performance (area under curve [AUC]=0.682) compared to GPS (AUC=0.671) and SIS (AUC=0.571).

Conclusion: The OPS independently predicts all-cause mortality after EVT for iliac artery stenosis and outperforms other inflammation-based scores. Its simplicity and reliance on routine biomarkers make it a practical tool for risk stratification in peripheral artery disease.

Keywords: Endovascular procedures, inflammation, malnutrition, mortality, peripheral artery disease, prognosis.

Peripheral artery disease (PAD), a common global health problem which affects more than 230 million adults worldwide,[1] is the third most frequent clinical presentation of atherosclerosis, following coronary artery disease and cerebrovascular disease. It is often characterized by multiple coexisting risk factors such as, advanced age, diabetes mellitus, hypertension, dyslipidemia, and smoking, underscoring the need for a collaborative, multidisciplinary treatment approach.[1] In general, PAD presents a wide clinical spectrum; it can be asymptomatic or accompanied by symptoms, including non-disabling intermittent claudication, disabling intermittent claudication, ischemic rest pain, ischemic ulceration or gangrene.[2] Patients with PAD are at high risk for cardiovascular death and limb-related complications. Several studies have shown that the five-year all-cause mortality rate in PAD patients can reach up to 30%, with cardiovascular causes accounting for a significant proportion.[3]

Among the anatomical subtypes of PAD are aorto-iliac, femoropopliteal, and infrapopliteal segmental lesions. The aorto-iliac region, particularly the iliac arteries that provide more than 50% of iliofemoral blood flow, plays a crucial role. Iliac artery stenosis is associated with intermittent claudication, decreased exercise capacity, and reduced quality of life.

Currently, endovascular therapy (EVT) has become the first-line treatment for iliac artery stenosis due to its lower morbidity and shorter recovery period compared

Corresponding author: Onur Erdoğan, MD. İstanbul Mehmet Akif Ersoy Göğüs Kalp ve Damar Cerrahisi Eğitim ve Araştırma Hastanesi, Kardiyoloji Kliniği, 34303 Küçükçekmece, İstanbul, Türkiye
E-mail: onurerdogan86@gmail.com

Citation:
Erdoğan O, Erdoğan T, Sevinç S, Taşbulak Ö, Panç C, Çelik Ö, et al. Osaka Prognostic Score for predicting mortality after iliac artery revascularization. Cardiovasc Surg Int 2025;12(3):223-232. doi: 10.5606/e-cvsi.2025.1990.

to surgical bypass.[4,5] Techniques such as balloon angioplasty, primary stenting, and "kissing-stents" have been reported with high technical success rates (>95%) and low periprocedural complication rates.[4] However, long-term mortality and the need for reintervention remain considerable, highlighting the importance of patient-related vulnerabilities which cannot be fully explained by anatomical factors alone.[6]

Over the past decade, various clinical and biochemical scoring systems have been developed to predict prognosis in PAD. Although classification systems such as Fontaine and Rutherford offer risk stratification based on symptom severity, they remain limited in predicting long-term mortality. Models such as PREVENT III[7] and WIfI[8] are more specific to patients with critical limb ischemia. In addition, inflammation and nutrition-based composite indexes, such as the Naples Prognostic Score (NPS), Prognostic Nutritional Index (PNI), Systemic Immune-Inflammation Index (SII), Hemoglobin, Albumin, Lymphocyte, Platelet (HALP), and the albumin/C-reactive protein (CRP) have been associated with adverse outcomes in cardiovascular and peripheral vascular diseases.[9-14]

In this context, the Osaka Prognostic Score (OPS), which combines CRP, serum albumin, and total lymphocyte count (TLC), is a simple and easily applicable indicator reflecting systemic inflammation and malnutrition. Initially validated in gastrointestinal oncology,[15] the OPS has recently gained attention in cardiovascular settings such as contrast-induced nephropathy after coronary angiography and stroke.[16,17]

However, to the best of our knowledge, no study to date has investigated the prognostic value of OPS in patients undergoing EVT for iliac artery disease. In the present study, we, therefore, aimed to investigate the prognostic value of the OPS in predicting all-cause mortality in patients undergoing EVT for iliac artery stenosis.

PATIENTS AND METHODS

This single-center, retrospective study was conducted at İstanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital, Department of Cardiology between January 2015 and December 2020. Initially, patients with iliac artery disease who received EVT were screened. Demographic characteristics, clinical findings, laboratory results, and procedural details were retrieved from hospital records of patients. Mortality data were validated through death notification records from the National Social Security Administration. Patients who underwent emergent endovascular intervention due to acute embolism, those with a prior history of aortic or iliac aneurysm or dissection, those without regular follow-up, those who did not adhere to prescribed dual antiplatelet therapy after the procedure, those with missing clinical or laboratory data, and patients with concomitant infectious or autoimmune diseases were excluded from the study. Finally, a total of 183 patients (165 males, 18 females; mean age: 61.0±8.8 years; range, 36 to 92 years) who met the inclusion criteria were recruited. Written informed consent was obtained from each patient. The study protocol was approved by the İstanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital Ethics Committee (Date: 30.06.2025, No: 2025.06-51). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Blood sampling

Blood samples were obtained from each patient on the morning after a 12-h fasting, prior to the procedure to measure serum albumin, estimated glomerular filtration rate (eGFR), total cholesterol, low-density lipoprotein (LDL) and high-density lipoprotein (HDL) levels, and CRP. Complete blood count parameters, including lymphocyte count, were also recorded. Serum albumin levels were measured using the Cobas® 6000 c501 analyzer (Roche Holding AG, Basel, Switzerland), while hematological parameters were assessed with an automated hematology analyzer (Mindray BC-6000, Shenzhen Mindray Bio-Medical Electronics Co., Ltd., China).

The OPS was determined based on three parameters: CRP, serum albumin, and TLC. A score of 1 point was assigned for CRP >10.0 mg/L, albumin <3.5 g/dL, and TLC <1600/μL; otherwise, 0 points were given for each variable. The total OPS was obtained by summing these individual scores, yielding a cumulative score ranging from 0 to 3. Patients were subsequently categorized into four groups based on their total OPS. The calculation methods for OPS, as well as for the Glasgow Prognostic Score (GPS) and the Systemic Inflammation Score (SIS), are illustrated in Figure 1.

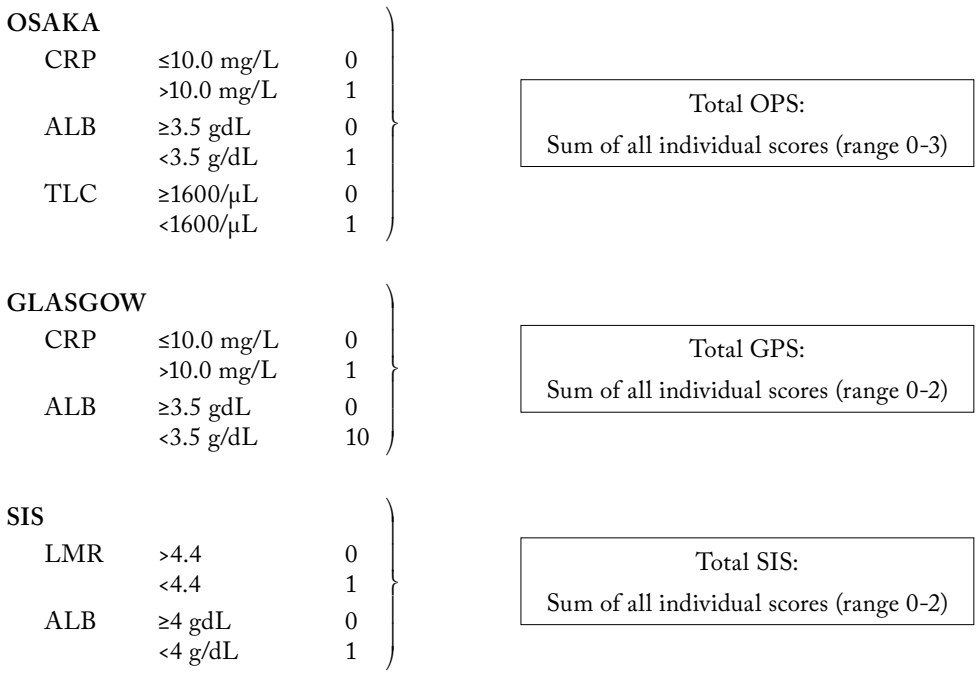

Figure 1. Scoring criteria and components of the OPS, GPS, and SIS.

CRP: C-reactive protein; ALB: Albumin; TLC: Total lymphocyte count; OPS: Osaka Prognostic Score; GPS: Glasgow Prognostic Score; LMR: lymphocyte-to-monocyte ratio; SIS: Systemic Inflammation Score.

Procedure

All endovascular interventions were performed under systemic heparin administration targeting an activated clotting time of 300 sec. The catheter balloons and stents used during the procedure and the choice of crossing approach was left to the discretion of the operating physician. Procedural success was defined as achieving less than 30% residual stenosis following intervention.

Treatment and follow-up

After EVT, all participants received acetylsalicylic acid (100 mg) and clopidogrel (75 mg) for at least one month. Additional medications such as cilostazol, beta-blockers, angiotensin-converting enzyme inhibitors, nitrates, and statins were administered based on standardized treatment protocols.

The patients were routinely followed at one and three months after the procedure, and then at six-month intervals based on symptom status. At each visit, a physical examination and ankle-brachial index (ABI) measurement were performed. Those who described symptoms or observed a decrease in ABI measurements were re-evaluated with Duplex ultrasound and computed tomography (CT) angiography, if necessary. Restenosis was defined as a luminal diameter reduction greater than 50% on postprocedural CT angiography, and a peak systolic velocity index (PSVI) exceeding 2.4 on Duplex ultrasound.

Statistical analysis

Statistical analysis was performed using the IBM SPSS for Windows version 26.0 software (IBM Corp., Armonk, NY, USA). Continuous variables were expressed in mean ± standard deviation (SD) or median and interquartile range (IQR), depending on data distribution assessed by the Kolmogorov-Smirnov test. Categorical variables were expressed in number and frequency. Comparisons between groups were conducted using the Student t-test or Mann-Whitney U test for continuous variables, and the chi-square test or Fisher exact test for categorical variables, as appropriate. Univariate logistic regression analysis was initially performed to identify potential predictors of all-cause mortality. Variables with a p value of <0.05 in the univariate analysis were included in the multivariate logistic regression model to determine independent predictors. However, to avoid model overfitting due to the

Table 1
Baseline demographic, clinical, and laboratory characteristics of the patients stratified by mortality status

Variables	All patients	Mortality (-)	Mortality (+)	*p*
Age (year) (mean±SD)	61.04±8.79	62.10±7.31	54.0±16.09	0.21
Sex, n (%)				0.78
Male	165 (90.2)	120 (89.6)	45 (91.8)	
Female	18 (9.8)	14 (10.4)	4 (8.2)	
Height (mean±SD)	169.7±5.72	170.15±5.72	166.67±5.77	0.37
Weight (mean±SD)	76.04±8.93	76.45±9.36	73.33±5.77	0.10
BMI (mean±SD)	26.45±3.27	26.43±3.27	26.56±4.06	0.12
HT, n (%)	130 (71.0)	87 (64.9)	43 (87.8)	0.003
DM, n (%)	101 (55.2%	69 (51.5)	32 (65.3)	0.13
CKD, n (%)	40 (21.9)	19 (14.2)	21 (42.9)	<0.001
COPD, n (%)	58 (31.7)	35 (26.1)	23 (46.9)	0.007
AF, n (%)	12 (6.6)	6 (4.5)	6 (12.2)	0.08
CAD, n (%)	123 (67.2)	84 (62.7)	39 (79.6)	0.03
CVA, n (%)	25 (13.7)	17 (12.7)	8 (16.3)	0.52
Previous PAD, n (%)	53 (29)	35 (26.2)	18 (36.7)	0.16
Smoking, n (%)	131 (71.6)	99 (73.9)	32 (65.3)	0.26
Rutherford Classification n (%)				0.05
2	39 (21.3)	33 (24.6)	6 (12.2)	
3	101 (55.2)	75 (56.0)	26 (53.1)	
4	26 (14.2)	18 (13.4)	8 (16.3)	
5	14 (7.7)	6 (4.5)	8 (16.3)	
6	3 (1.6)	2 (1.5)	1 (2)	
LVEF (%), median (min-max)	60 (50-60)	60 (55-65)	55 (40-60)	<0.001
Total cholesterol (mg/dL), median (min-max)	175 (150-218)	179 (151.5-230)	172.5 (142.75-187.75)	0.06
HDL (mg/dL), (mean±SD)	38.61±8.48	38.2±8.9	41.67±4.93	0.19
LDL (mg/dL), median (min-max)	102 (76.5-140)	102 (79.5-144.5)	102 (68.25-102)	0.08
Triglyceride (mg/dL), median (min-max)	171.5 (115-245.25)	175 (115-269)	153 (110-190)	0.12
Lymphocyte (10^3/mm^3), median (min-max)	2.21 (1.75-2.75)	2.26 (1.77-2.79)	2 (1.57-2.65)	0.11
Neutrophil (10^3/mm^3), (mean±SD)	5.67±1.49	5.56±1.43	6.37±2.03	0.01
Monocyte (10^3/mm^3), median (min-max)	0.75 (0.60-0.94)	0.72 (0.60-0.94)	0.77 (0.58-0.96)	0.53
Platelets (10^3/mm^3), median (min-max)	259 (217.5-318)	255.5 (217.25-302.75)	279 (217-354)	0.40
CRP (mg/L), median (min-max)	6.9 (3.08-21.3)	5.58 (2.96-15.24)	15.4 (4.83-35.14)	0.001
Albumin (g/dL), median (min-max)	4.2 (3.89-4.44)	4.2 (3.99-4.45)	4 (3.64-4.42)	0.009
Creatinine (mg/dL), median (min-max)	0.92 (0.73-1.14)	0.9 (0.71-1.05)	1.05 (0.8-1.38)	0.002
GFR, median (min-max)	87.37 (68.31-102.88)	89.17 (73.87-103.07)	77.54 (53.52-102.45)	0.06
OPS, n (%)				<0.001
0	85 (46.4)	73 (54.5)	12 (24.5)	
1	72 (39.3)	49 (36.6)	23 (46.9)	
2	21 (11.5)	10 (7.5)	11 (22.4)	
3	5 (2.7)	2 (1.5)	3 (6.1)	
GPS, n (%)				<0.001
0	97 (53)	83 (61.9)	14 (28.6)	
1	73 (39.9)	44 (32.8)	29 (59.2)	
2	13 (7.1)	7 (5.2)	6 (12.2)	
SIS, n (%)				0.23
0	100 (54.6)	78 (58.2)	22 (44.9)	
1	76 (41.5)	52 (38.8)	24 (49)	
2	7 (3.8)	4 (3)	3 (6.1)	

BMI: Body mass index; HT: Hypertension; DM: Diabetes mellitus; CKD: Chronic kidney disease; COPD: Chronic obstructive pulmonary disease; AF: Atrial fibrillation; CAD: Coronary artery disease; CVA: Cerebrovascular accident; PAD: Peripheral artery disease; LVEF: Left ventricular ejection fraction; HDL: High-density lipoprotein; LDL: Low-density lipoprotein; CRP: C-reactive protein; GFR: Glomerular filtration rate; OPS: Osaka Prognostic Score; GPS: Glasgow Prognostic Score; SIS: Systemic Inflammation Score.

limited number of events (49 death events), the number of covariates included in the multivariate regression was restricted. Therefore, only the variables with the highest odds ratios (ORs) in univariate analysis and clinical plausibility were included. Multicollinearity among variables was assessed and variables with significant collinearity were excluded from the final model. Stroke and myocardial infarction (MI) were not included in the multivariate model, as they are directly associated with mortality and may act as intermediate outcomes rather than independent predictors, which could distort the regression model by introducing overadjustment bias.[18] The receiver operating characteristic (ROC) curve analysis was used to evaluate the discriminatory ability of the OPS, GPS, and Systemic SIS in predicting mortality. The area under the curve (AUC), sensitivity, specificity, and positive likelihood ratio were calculated for each score. A p value of <0.05 was considered statistically significant with 95% confidence interval (CI).

RESULTS

Of a total of 183 patients included in the study, 49 (26.8%) died during follow-up. For survival analyses, patients who were alive at that time were censored at their last follow-up date.

Patients in the mortality group were more likely to have hypertension (87.8% *vs.* 64.9%, p=0.003), chronic kidney disease (42.9% *vs.* 14.2%, p<0.001), chronic obstructive pulmonary disease (46.9% *vs.* 26.1%, p=0.007), and coronary artery disease (79.6% *vs.* 62.7%, p=0.03). No significant differences were observed between groups regarding diabetes mellitus, cerebrovascular disease, smoking status, or a history of PAD (Table 1).

Laboratory investigations revealed that patients in the mortality group had significantly higher CRP levels (15.4 *vs.* 5.6 mg/L, p=0.001), lower serum albumin (4.0 *vs.* 4.2 g/dL, p=0.009), and lower left ventricular ejection fraction values (55% *vs.* 60%, p<0.001). Creatinine levels were higher (p=0.002),

Table 2
Procedural variables and angiographic characteristics in the total cohort and by mortality status.

	All patients			Mortality (-)			Mortality (+)			
Variables	n	%	Mean±SD	n	%	Mean±SD	n	%	Mean±SD	p
Chronic total occlusion	86	47.3		69	51.9		17	34.7		0.04
Total lesion length (mm)										
>5 cm										
5-10 cm										
>10 cm										
TASC II										0.89
A	56	30.8		39	29.3		17	34.7		
B	61	33.5		44	33.1		17	34.7		
C	46	25.3		35	26.3		11	22.4		
D	18	9.9		14	10.5		4	8.2		
Diffuse disease	111	62		77	59.2		34	69.4		0.21
Previous intervention	52	28.4		34	25.4		18	36.7		0.13
Complication in procedure										0.94
Dissection	22	12		16	11.9		6	12.2		
Rupture	4	2.2		3	2.2		1	2		
Distal embolization	2	1.1		2	1.5		0	0		
Others (stent jump, plaque shift, stent disunite)	7	3.8		5	3.7		2	4.1		
Residual stenosis	37	20.2		27	20.1		10	20.4		0.97
Procedural success	174	95.1		126	94		48	98		0.28
Follow-up time (mo)			37.1±14.2			36.8±14.4			39.9±12.6	0.32

SD: Standard deviation; TASC II: Trans-Atlantic Inter-Society Consensus II.

and neutrophil counts were elevated (p=0.013) in the mortality group (Table 1).

There were no significant differences in rates of procedural complications and residual stenosis between survivors and non-survivors. However, patients with higher OPS scores had worse follow-up outcomes. Restenosis was more frequently seen among patients with OPS ≥2 (OPS 2: 23.8%, OPS 3: 20%), and the incidence of MI during follow-up increased with higher OPS (OPS 2: 23.8%, OPS 3: 20%) (Table 2).

The OOPS distribution was significantly different between survivors and non-survivors (p<0.001). Higher OPS was associated with increased mortality (OPS 0: 14.1%, OPS 1: 31.9%, OPS 2: 52.4%,

Table 3
Follow-up outcomes stratified by OPS group

	OPS 0		OPS 1		OPS 2		OPS 3		
Variables	n	%	n	%	n	%	n	%	*p*
Restenosis	12	14.1	11	15.3	5	23.8	1	20	0.32
In hospital thrombosis	0	0	2	2.8	1	4.8	0	0	0.34
MI in follow-up	11	12.9	12	16.7	5	23.8	1	20	0.65
Stroke in follow-up	4	4.7	2	2.8	0	0	0	0	0.69
Mortality	12	14.1	23	31.9	11	52.4	3	60.0	<0.001

OPS: Osaka Prognostic Score; MI: Myocardial infarction.

Table 4
Results of univariate and multivariate logistic regression analyses for predictors of all-cause mortality

	Univariate analyses			Multivariate analyses Model 1			Multivariate analyses Model 2		
Variables	OR	95% CI	*p*	OR	95% CI	*p*	OR	95% CI	*p*
Age	1.023	0.987-1.060	0.23				0.988	0.946-1.032	0.60
Sex	0.762	0.238-2.437	0.65				0.646	0.184-2.266	0.50
BMI	1.286	0.987-1.677	0.06						
HT	3.872	1.535-9.763	0.004	2.562	0.948-6.925	0.06	2.777	1.013-7.614	0.05
DM	1.773	0.899-3.496	0.10						
CKD	4.539	2.154-9.567	<0.001	3.288	1.481-7.300	0.003	3.574	1.498-8.528	0.004
COPD	2.502	1.267-4.942	0.008						
AF	2.977	0.912-9.718	0.07						
CAD	2.321	1.066-5.054	0.03	1.904	0.806-4.497	0.14	1.865	0.793-4.385	0.15
CVA	1.343	0.539-3.345	0.53						
Previous PAD	1.642	0.818-3.298	0.16						
MI in follow-up	9.579	3.956-23.194	<0.001						
Stroke in follow-up	6.000	1.062-33.889	0.04						
OPS	4.067	1.725-9.589	0.001	3.392	1.326-8.673	0.01	3.452	1.342-8.878	0.01
GPS 1/0	3.907	1.873-8.150	<0.001						
GPS 2/0	5.082	1.487-17.363	0.01						
SIS 1/0	1.071	0.507-2.259	0.86						
SIS 2/0	1.400	0.123-15.923	0.79						

OR: Odds ratio; CI: Confidence interval; BMI: Body mass index; HT: Hypertension; DM: Diabetes mellitus; CKD: Chronic kidney disease; COPD: Chronic obstructive pulmonary disease; AF: Atrial fibrillation; CAD: Coronary artery disease; CVA: Cerebrovascular accident; PAD: Peripheral artery disease; MI: Myocardial infarction; OPS: Osaka Prognostic Score; GPS: Glasgow Prognostic Score; SIS: Systemic Inflammation Score.

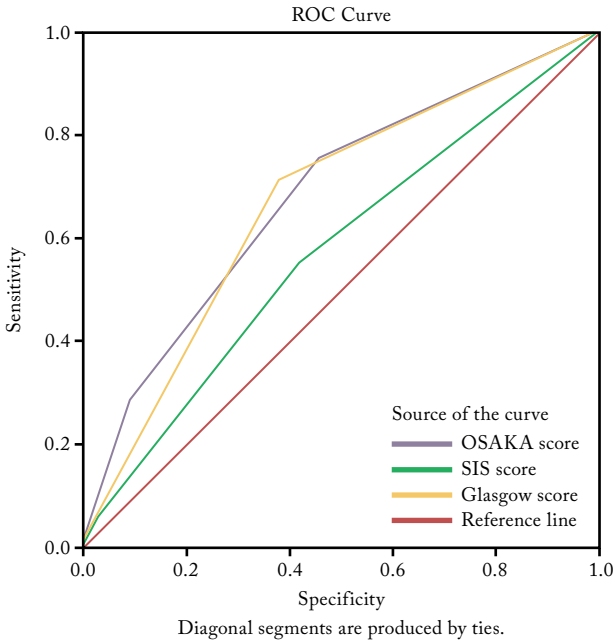

Figure 2. Receiver operating characteristic OPS, GPS, and SIS for predicting all-cause mortality.

ROC: Receiver operating characteristic; OPS: Osaka Prognostic Score; GPS: Glasgow Prognostic Score; SIS: Systemic Inflammation Score.

OPS 3: 60%) (Table 3). The OPS was found to be an independent mortality predictor in the univariate analysis (OR=3.392, 95% CI: 1.326-8.673, p=0.01), and this association remained significant after adjustment for clinical factors (OR=3.452, 95% CI: 1.342-8.878, p=0.01) (Table 4).

According to the ROC analysis, OPS exhibited superior discriminative performance (AUC=0.682) compared to GPS (AUC=0.671) and SIS (AUC=0.571) (Figure 2). An OPS cut-off value of ≥2 yielded a specificity of 91% and a sensitivity of 29% with a positive likelihood ratio of 3.19.

DISCUSSION

In the present study, we investigated the prognostic value of the OPS, a composite marker of systemic inflammation and nutritional status, in patients undergoing EVT for iliac artery stenosis. We believe that evaluating this holistic biomarker that reflects systemic frailty may contribute significantly to conventional risk stratification approaches in the management of PAD. Our results demonstrated that higher OPS values were significantly associated with increased all-cause mortality. The OPS outperformed other inflammation-based scoring systems, such as the GPS and the SIS, in predicting mortality. Patients with an OPS ≥2 had markedly higher rates of death, MI, and restenosis during follow-up. Moreover, OPS remained an independent predictor of mortality even after adjusting for major clinical confounders including age, gender, hypertension, chronic kidney disease, and coronary artery disease. These findings suggest that OPS may be a simple, cost-effective, and clinically applicable tool for risk stratification in this patient population. While the area under the ROC curve (AUC) for OPS was in the moderate range (AUC=0.682), this still reflects a meaningful level of prognostic discrimination. Although the discriminative power is not exceptionally high, OPS can identify patients at elevated risk, particularly when interpreted alongside other clinical or anatomical parameters. Notably, using a threshold of OPS ≥2, specificity was high (91%) despite a relatively low sensitivity (29%), indicating that the score is particularly effective in correctly identifying those truly at risk. Given its ease of calculation, affordability, and reliance on widely available markers (CRP, albumin, and TLC), The OPS remains a valuable and accessible component of clinical risk stratification, particularly for identifying patients who may benefit from closer monitoring and more intensive management.

In patients undergoing EVT for iliac artery stenosis, systemic inflammation plays a crucial role in determining both short- and long-term outcomes.[19] While procedural success rates are usually high with current endovascular techniques, long-term morbidity and mortality remain significant, suggesting that patient-related factors, such as chronic inflammation, may substantially influence prognosis.[20] Inflammation contributes to atherosclerotic disease progression, plaque instability, endothelial dysfunction, and thrombosis, all of which can adversely affect vessel patency and promote restenosis after intervention.[21] Moreover, chronic low-grade inflammation has been associated with impaired collateral vessel formation, delayed wound healing, and increased cardiovascular event rates, particularly in older adults and patients with multiple comorbidities.[22] C-reactive protein is one of the most extensively studied biomarkers of inflammation. Numerous clinical investigations have demonstrated that elevated CRP levels are strongly and independently associated with adverse outcomes in patients with cardiovascular

diseases.[23] Therefore, assessing inflammation prior to intervention may help identify high-risk patients who could benefit from closer monitoring, targeted anti-inflammatory strategies, or more aggressive secondary prevention measures. In this context, inflammation is not merely a byproduct of atherosclerosis, but a central pathophysiological mechanism which directly impacts procedural durability and patient survival in PAD involving the iliac arteries.[24]

In parallel with inflammation, malnutrition is increasingly recognized as a critical determinant of poor outcomes in patients with PAD, particularly those undergoing endovascular intervention for iliac artery stenosis.[25,26] Malnutrition leads to impaired wound healing, decreased muscle mass, reduced functional capacity, and higher susceptibility to infection, all of which can adversely impact recovery and long-term prognosis.[18,27]

Hypoalbuminemia commonly reflects malnutrition, systemic inflammation, or cachexia, or combinations of them. A dedicated meta-analysis on peripheral arterial disease patients undergoing angioplasty and revascularization demonstrates that low preoperative serum albumin confers a significantly elevated risk of in-hospital mortality and morbidity.[28]

Moreover, malnutrition and inflammation often coexist and interact synergistically, forming a vicious cycle which accelerates atherosclerotic progression and diminishes vascular repair mechanisms.[29]

Previous studies have proposed that this bidirectional relationship between inflammation and malnutrition may promote systemic atherosclerosis. Rein et al.[30] reported that systemic inflammation was more pronounced in patients with PAD than in those with coronary artery disease, suggesting that PAD may reflect a broader polyvascular pathology rather than an isolated peripheral arterial process. Systemic inflammation, through elevated cytokines, oxidative stress, and immune cell infiltration, causes malnutrition through impaired appetite, increased resting energy expenditure, and accelerated breakdown of muscle proteins and overall protein stores.[31-33]

Conversely, malnutrition itself can exacerbate atherosclerosis by impairing immune function and vascular integrity. This mutually reinforcing interplay is referred to as the malnutrition-inflammation-atherosclerosis (MIA) syndrome.[32] Composite scores such as the PNI, NPS, and the OPS integrate nutritional and inflammatory parameters, providing a more comprehensive risk assessment. In patients with PAD, particularly those with advanced disease or undergoing revascularization, routine assessment of nutritional status is essential. Identifying and addressing malnutrition may improve functional recovery, reduce complications, and enhance overall survival. Although initially developed in oncology, the OPS has increasingly attracted attention in cardiovascular medicine. Özbeyaz and Algül[16] investigated the role of OPS in patients with acute coronary syndrome undergoing percutaneous coronary intervention (PCI). They found that higher OPS scores were significantly associated with an increased risk of contrast-induced nephropathy, longer hospitalization, and in-hospital complications Similarly, Liu et al.[17] examined OPS and the NPS in patients with spontaneous intracerebral hemorrhage and demonstrated that both were independently predictive of six-month poor functional outcomes. Our findings are in line with prior literature evaluating the prognostic role of the OPS in various cardiovascular settings. Although these prior studies primarily focused on coronary or systemic vascular populations, our study extends these findings to peripheral arterial disease, particularly involving the iliac arteries.

These findings support the growing interest in OPS as a generalizable biomarker of systemic frailty, applicable beyond cancer. In cardiovascular populations, where inflammation and malnutrition are known contributors to adverse outcomes, OPS may offer incremental value. Unlike disease-specific scores, OPS reflects broader physiological reserve and immune-nutritional status, making it suitable for integration into routine risk stratification algorithms.

One of the strengths of our study lies in its focus on a relatively under-investigated population-patients with isolated iliac artery stenosis undergoing EVT. Furthermore, by comparing OPS with other established inflammatory indices such as GPS and SIS, we provide evidence supporting its superior discriminative ability. The use of routinely collected laboratory data enhances the score's potential for clinical integration.

Nonetheless, this study has several limitations. First, it is a single-center, retrospective study, which may limit the generalizability of the findings. Second, the relatively small sample size and low event rate may have reduced the statistical power for subgroup analyses. Third, nutritional status and inflammatory markers were only assessed at baseline; serial measurements could have provided insights into dynamic risk prediction. Finally, despite multivariate adjustment, residual confounding cannot be entirely excluded.

In conclusion, the OPS, an easily applicable biomarker incorporating CRP, albumin, and lymphocyte count, was found to independently predict all-cause mortality in patients undergoing EVT for iliac artery stenosis. Our results suggest that OPS may provide incremental prognostic value over traditional inflammation-based indices and contribute to a more holistic risk stratification model in PAD. Given its simplicity, cost-effectiveness, and reliance on routinely available laboratory parameters, OPS may represent a promising adjunct to existing clinical risk scores. Future multi-center, prospective studies with larger patient populations are warranted to validate our findings and determine whether integrating OPS into risk prediction algorithms improves patient outcomes. Additionally, interventional trials exploring whether modifying inflammation or nutritional status can lower OPS and thereby improve prognosis would be of great clinical interest.

Acknowledgements: The authors would like to thank the medical records team and vascular imaging unit at Istanbul Mehmet Akif Ersoy Thoracic and Cardiovascular Surgery Training and Research Hospital for their support in data collection and technical assistance throughout this study.

Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Conceptualization, formal analysis, writing-original draft: O.E., T.E.; Methodology: O.E.; Investigation: C.P., O.E.; Data curation: Ö.T., O.E.; Writing-review & editing: T.E., C.P., O.E., Ö.T.; Supervision: Ö.Ç., A.A.Y. All authors have read and approved the final manuscript.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Song P, Rudan D, Zhu Y, Fowkes FJI, Rahimi K, Fowkes FGR, et al. Global, regional, and national prevalence and risk factors for peripheral artery disease in 2015: An updated systematic review and analysis. Lancet Glob Health 2019;7:e1020-30. doi: 10.1016/S2214-109X(19)30255-4.
2. Mazzolai L, Teixido-Tura G, Lanzi S, Boc V, Bossone E, Brodmann M, et al. 2024 ESC Guidelines for the management of peripheral arterial and aortic diseases. Eur Heart J 2024;45:3538-700. doi: 10.1093/eurheartj/ehae179.
3. Rantner B, Kollerits B, Pohlhammer J, Stadler M, Lamina C, Peric S, et al. The fate of patients with intermittent claudication in the 21st century revisited - results from the CAVASIC Study. Sci Rep 2017;8:45833. doi: 10.1038/srep45833.
4. Salem M, Hosny MS, Francia F, Sallam M, Saratzis A, Saha P, et al. Management of Extensive Aorto-Iliac Disease: A systematic review and meta-analysis of 9319 patients. Cardiovasc Intervent Radiol 2021;44:1518-35. doi: 10.1007/s00270-021-02785-6.
5. de Donato G, Bosiers M, Setacci F, Deloose K, Galzerano G, Verbist J, et al. 24-Month data from the BRAVISSIMO: A Large-scale prospective registry on iliac stenting for TASC A & B and TASC C & D lesions. Ann Vasc Surg 2015;29:738-50. doi: 10.1016/j.avsg.2014.12.027.
6. Klein WM, van der Graaf Y, Seegers J, Moll FL, Mali WP. Long-term cardiovascular morbidity, mortality, and reintervention after endovascular treatment in patients with iliac artery disease: The Dutch Iliac Stent Trial Study. Radiology 2004;232:491-8. doi: 10.1148/radiol.2322030725.
7. Schanzer A, Mega J, Meadows J, Samson RH, Bandyk DF, Conte MS. Risk stratification in critical limb ischemia: Derivation and validation of a model to predict amputation-free survival using multicenter surgical outcomes data. J Vasc Surg 2008;48:1464-71. doi: 10.1016/j.jvs.2008.07.062.
8. Mills JL Sr, Conte MS, Armstrong DG, Pomposelli FB, Schanzer A, Sidawy AN, et al. The Society for Vascular Surgery Lower Extremity Threatened Limb Classification System: Risk stratification based on wound, ischemia, and foot infection (WIfI). J Vasc Surg 2014;59:220-34.e1-2. doi: 10.1016/j.jvs.2013.08.003.
9. Pan D, Guo J, Su Z, Meng W, Wang J, Guo J, et al. Association of prognostic nutritional index with peripheral artery disease in US adults: A cross-sectional study. BMC Cardiovasc Disord 2024;24:133. doi: 10.1186/s12872-024-03815-0.
10. Artac I, Karakayali M, Omar T, Ilis D, Arslan A, Hakan Sahin M, et al. Predictive value of the Naples prognostic score on long-term outcomes in patients with peripheral artery disease revascularized via percutaneous intervention. Ann Vasc Surg 2024;102:121-32. doi: 10.1016/j.avsg.2023.11.028.
11. Tian M, Li Y, Wang X, Tian X, Pei LL, Wang X, et al. The Hemoglobin, Albumin, Lymphocyte, and Platelet (HALP) score is associated with poor outcome of acute ischemic stroke. Front Neurol 2021;11:610318. doi: 10.3389/fneur.2020.610318.

12. Ye Z, Hu T, Wang J, Xiao R, Liao X, Liu M, et al. Systemic immune-inflammation index as a potential biomarker of cardiovascular diseases: A systematic review and meta-analysis. Front Cardiovasc Med 2022;9:933913. doi: 10.3389/fcvm.2022.933913.
13. Erdogan O, Erdogan T, Panc C, Tasbulak O, Altunova M, Arif Yalcin A, et al. Prognostic nutritional index as a new prediction tool for all-cause mortality in patients with chronic limb-threatening ischemia undergoing endovascular therapy. Sisli Etfal Hastan Tip Bul 2024;58:346-53. doi: 10.14744/SEMB.2024.70094.
14. Panç C, Güler A, Gürbak İ, Taşbulak Ö, Güner A, Kalkan AK, et al. Association between CRP/albumin ratio and long-term mortality in patients with chronic limb-threatening ischemia undergoing endovascular therapy below the knee: The ACHILES-BTK registry. Ann Vasc Surg 2022;82:172-80. doi: 10.1016/j.avsg.2021.11.008.
15. Fujino S, Myoshi N, Saso K, Sasaki M, Ishikawa S, Takahashi Y, et al. The inflammation-nutrition score supports the prognostic prediction of the TNM stage for colorectal cancer patients after curative resection. Surg Today 2020;50:163-70. doi: 10.1007/s00595-019-01861-2.
16. Özbeyaz NB, Algül E. A new parameter in predicting contrast-induced nephropathy: Osaka prognostic score. Rev Assoc Med Bras (1992) 2024;70:e20240423. doi: 10.1590/1806-9282.20240423.
17. Liu R, Chen C, Zhao Y, Tang Y, Shen W, Xie Z. The Osaka prognostic score and Naples prognostic score: Novel biomarkers for predicting short-term outcomes after spontaneous intracerebral hemorrhage. BMC Neurol 2023;23:272. doi: 10.1186/s12883-023-03287-3.
18. Arnold M, Barbul A. Nutrition and wound healing. Plast Reconstr Surg 2006;117:42S-58. doi: 10.1097/01.prs.0000225432.17501.6c.
19. Koeckerling D, Zielasek C, Stähli P, Wohlfarth B, Rosenov A, Helfenstein F, et al. Patient-specific risk factors for reintervention following primary endovascular treatment of iliac artery disease. J Vasc Surg 2024;79:847-55.e5. doi: 10.1016/j.jvs.2023.12.012.
20. Bleda S, De Haro J, Acin F, Varela C, Esparza L, López de Maturana I. Inflammatory burden predicts long-term outcomes in endovascular therapy in peripheral arterial disease. Ann Vasc Surg 2013;27:459-66. doi: 10.1016/j.avsg.2012.02.027.
21. Libby P. Inflammation in atherosclerosis. Nature 2002;420:868-74. doi: 10.1038/nature01323.
22. Ridker PM. C-reactive protein and the prediction of cardiovascular events among those at intermediate risk: Moving an inflammatory hypothesis toward consensus. J Am Coll Cardiol 2007;49:2129-38. doi: 10.1016/j.jacc.2007.02.052.
23. Romero-Cabrera JL, Ankeny J, Fernández-Montero A, Kales SN, Smith DL. A systematic review and meta-analysis of advanced biomarkers for predicting incident cardiovascular disease among asymptomatic middle-aged adults. Int J Mol Sci 2022;23:13540. doi: 10.3390/ijms232113540.
24. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. N Engl J Med 2005;352:1685-95. doi: 10.1056/NEJMra043430.
25. Yokoyama M, Watanabe T, Otaki Y, Watanabe K, Toshima T, Sugai T, et al. Impact of objective malnutrition status on the clinical outcomes in patients with peripheral artery disease following endovascular therapy. Circ J 2018;82:847-56. doi: 10.1253/circj.CJ-17-0731.
26. Lin TY, Chou HH, Hsieh TH, Huang HL, Hung SC. Malnutrition is associated with increased morbidity and death in dialysis patients undergoing endovascular therapy for peripheral artery disease. Eur J Vasc Endovasc Surg 2022;64:225-33. doi: 10.1016/j.ejvs.2022.03.045.
27. Campos AC, Groth AK, Branco AB. Assessment and nutritional aspects of wound healing. Curr Opin Clin Nutr Metab Care 2008;11:281-8. doi: 10.1097/MCO.0b013e3282fbd35a.
28. Hanggrainy OS BB, Satria G, Umar A, Nugraha A, Tobing AHL, Nasution IH. Albumin serum as a predictor of prognosis in patients with peripheral artery disease post angioplasty: A systematic review and meta-analysis. J Indones Thorac Cardiol Vasc Surg (JINATCVS) 2024;1:29-33.
29. Ishii H, Aoyama T, Takahashi H, Kamoi D, Tanaka M, Yoshikawa D, et al. Serum albumin and C-reactive protein levels predict clinical outcome in hemodialysis patients undergoing endovascular therapy for peripheral artery disease. Atherosclerosis 2013;227:130-4. doi: 10.1016/j.atherosclerosis.2012.11.034.
30. Rein P, Saely CH, Silbernagel G, Vonbank A, Mathies R, Drexel H, et al. Systemic inflammation is higher in peripheral artery disease than in stable coronary artery disease. Atherosclerosis 2015;239:299-303. doi: 10.1016/j.atherosclerosis.2015.01.021.
31. Ross R. Atherosclerosis--an inflammatory disease. N Engl J Med 1999;340:115-26. doi: 10.1056/NEJM199901143400207.
32. Stenvinkel P, Heimbürger O, Lindholm B, Kaysen GA, Bergström J. Are there two types of malnutrition in chronic renal failure? Evidence for relationships between Malnutrition, Inflammation and Atherosclerosis (MIA syndrome). Nephrol Dial Transplant 2000;15:953-60. doi: 10.1093/ndt/15.7.953.
33. Pecoits-Filho R, Lindholm B, Stenvinkel P. The Malnutrition, Inflammation, and Atherosclerosis (MIA) syndrome -- the heart of the matter. Nephrol Dial Transplant 2002;17 Suppl 11:28-31. doi: 10.1093/ndt/17.suppl_11.28.