



Research

Uric Acid-to-Albumin Ratio as A Prognostic Marker after Carotid Artery Stenting

Karotis Arter Stentleme Sonrası Prognostik Bir Belirteç Olarak Ürik Asit/ Albümin Oranı

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ABSTRACT

Objective: To evaluate the prognostic value of the uric acid-to-albumin ratio (UAR) for predicting major adverse cardiovascular and cerebrovascular events (MACCE) after carotid artery stenting (CAS).

Methods: A total of 332 patients who underwent CAS were retrospectively enrolled in the study. For each patient, the uric acid level was divided by the corresponding serum albumin level to calculate the UAR. Patients were followed up for the occurrence of MACCE. The association between UAR and MACCE was analyzed to determine its predictive value.

Results: Patients were followed up for an average of 45.30±27.26 months. Those who experienced MACCE were significantly older and were more likely to have undergone prior carotid endarterectomy. Additionally, they exhibited elevated serum creatinine and uric acid levels. In contrast, serum albumin and hemoglobin levels were significantly lower in the MACCE group. Furthermore, patients who developed MACCE had longer lesion lengths, required longer stents, and experienced higher rates of periprocedural stroke and in-hospital mortality. Multivariable logistic regression analysis identified a history of endarterectomy, elevated serum creatinine levels, and higher UAR as independent predictors of MACCE.

Conclusion: Higher UAR values were significantly associated with increased risk of adverse outcomes. These findings suggest that UAR may serve as a simple, accessible, and cost-effective biomarker for risk stratification and prognosis in patients undergoing CAS.

Keywords: Carotid artery, stent, uric acid, albumin, prognosis

ÖZ

Amaç: Bu çalışmanın amacı, karotis arter stentleme (CAS) sonrası majör kardiyovasküler ve serebrovasküler olayları (MACCE) öngörmeye ürik asit/ albümin oranının (UAR) prognostik değerini değerlendirmektir.

Gereç ve Yöntem: CAS uygulanan toplam 332 hasta retrospektif olarak çalışmaya dahil edildi. Her hastada, serum ürik asit düzeyi ilgili serum albümin düzeyine bölünerek UAR hesaplandı. Hastalar, MACCE gelişimi açısından takip edildi. UAR ile MACCE arasındaki ilişki, öngörü değerini belirlemek amacıyla analiz edildi.

Bulgular: Hastalar ortalama 45,30±27,26 ay süreyle takip edildi. MACCE gelişen hastalar anlamlı olarak daha ileri yaşta olup, daha yüksek oranda önceden karotis endarterektomi öyküsüne sahipti. Ayrıca, bu grupta serum kreatinin ve ürik asit düzeyleri daha yüksek bulundu. Buna karşın, serum albümin ve hemoglobin düzeyleri MACCE grubunda anlamlı olarak daha düşüktü. Bunun yanı sıra, MACCE gelişen hastalarda lezyon uzunluğu daha fazla, daha uzun stent kullanımı, periprosedürel inme ve hastanede yatış sırasında mortalite oranı daha yüksek saptandı. Çok değişkenli lojistik regresyon analizinde, endarterektomi öyküsü, yüksek serum kreatinin düzeyi ve yüksek UAR değeri, MACCE için bağımsız belirleyiciler olarak tespit edildi.

Sonuç: Yüksek UAR değerleri, olumsuz sonuç riskinde anlamlı bir artış ile ilişkili bulundu. Bu bulgular, UAR'nin CAS uygulanan hastalarda risk sınıflandırması ve prognoz değerlendirilmesinde basit, kolay erişilebilir ve maliyet etkin bir biyobelirteç olarak kullanılabileceğini düşündürmektedir.

Anahtar Kelimeler: Karotis arter, stent, ürik asit, albümin, prognoz

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INTRODUCTION

Carotid artery stenosis represents a significant and potentially preventable risk factor for ischemic stroke (1). Even moderate degrees of luminal narrowing can substantially compromise cerebral blood flow and increase the propensity for thromboembolic events. The predominant etiology of this condition is atherosclerosis, characterized by the progressive accumulation of lipids, inflammatory cells, and fibrous tissue within the arterial wall (2,3). Advanced plaques may undergo rupture or erosion, releasing thrombogenic material capable of obstructing intracranial arteries and precipitating acute ischemic episodes. Evidence from both clinical observations and large-scale epidemiological studies indicates that individuals with carotid atherosclerotic lesions—regardless of the presence of symptoms—exhibit a markedly elevated stroke risk compared with those with normal carotid anatomy (4,5). Therefore, early detection and timely preventive interventions are essential strategies to mitigate the overall burden of cerebrovascular disease. Carotid artery stenting (CAS) has emerged as a less invasive alternative to carotid endarterectomy in the management of extracranial carotid artery stenosis, particularly in patients at high surgical risk. CAS is often preferred in cases in which surgery poses technical or anatomical challenges, including high carotid bifurcation, prior endarterectomy with restenosis, or prior neck irradiation (6,7).

A growing body of research has shown that atherosclerosis is not merely a lipid accumulation disorder but involves sustained inflammatory activity at multiple stages of the disease (8,9). The uric acid-to-albumin ratio (UAR) is recognized as a novel combined biomarker that enables the simultaneous assessment of oxidative stress and the inflammatory response. During inflammatory processes, serum albumin levels decrease because albumin is a negative acute-phase reactant, while uric acid levels rise and are associated with oxidative stress and cellular damage, making UAR a comprehensive indicator of inflammatory and oxidative burden (10-13). Clinical studies have demonstrated that elevated UAR has value in cardiovascular and cerebrovascular diseases, coronavirus disease 2019 infection, and malignancies (14-17). Therefore, UAR is emerging not merely as a biochemical ratio but as an accessible, low-cost, and practical marker with growing clinical relevance in both prognostic risk assessment and the evaluation of the systemic inflammation-oxidative stress axis. Since carotid revascularization procedures carry a residual risk of post-interventional complications, identifying accessible and reliable biomarkers is of growing clinical interest. The UAR may serve as a useful indicator

for risk stratification in this population. By examining its association with adverse outcomes after stenting, we sought to determine whether this parameter can help predict prognosis and guide patient management, and to evaluate the UAR's potential to predict major adverse cardiovascular and cerebrovascular events (MACCE) following CAS.

METHODS

The records of patients treated with CAS at a tertiary care center between January 2018-January 2024 were retrospectively reviewed. Eligible individuals had either symptomatic carotid stenosis of $\geq 70\%$ or asymptomatic stenosis of $\geq 90\%$. The diagnosis of carotid artery stenosis was established in line with the criteria of the European Stroke Organization guidelines (18). A total of 545 files were evaluated; after applying the exclusion criteria, 332 patients remained in the final cohort. The study received approval from University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital Clinical Research Ethics Committee (approval no: 2025-01-05, date: 24.10.2025) and adhered to the principles of the Declaration of Helsinki.

Patients with infection, inflammatory diseases, hematological disorders, malignancy, end-stage renal or hepatic disease, total occlusion of the carotid artery, those receiving uricosuric therapy, or those who had a recent acute coronary syndrome were excluded from the study. As part of the initial work-up at hospital presentation, venous blood was obtained from each patient. These samples were subsequently analyzed in the clinical laboratory to assess biochemical indicators and to perform a complete blood count. The uric acid level measured for each patient was divided by the corresponding albumin value to calculate the UAR.

All CAS procedures were conducted via the transfemoral approach under local anesthesia. An 8F introducer sheath was initially inserted to obtain arterial access. Diagnostic angiography of the carotid arteries was then performed, most frequently using an 8F JR4 guiding catheter. The catheter was advanced to a position proximal to the stenotic segment, and angiographic imaging was acquired in ipsilateral oblique projections of approximately 30-45 degrees, as well as in lateral projections from the contralateral side, to ensure adequate visualization of the lesion. The extent of luminal narrowing was quantified in accordance with the European Carotid Surgery trial method. All angiographic images were reviewed by two interventional physicians with substantial procedural experience, both of whom were blinded to the demographic and clinical characteristics of the patients to avoid observer bias. In every case, a

distal embolic protection device was utilized prior to stent deployment to minimize the risk of periprocedural cerebral embolization. The selection of stent type—whether closed-cell or open-cell—and the decision to perform predilatation or postdilatation were left to the operator's clinical judgment based on vascular anatomy and lesion morphology. Self-expanding stents were implanted in all interventions. Following stent placement, a final biplane angiographic assessment of both the treated carotid segment and the intracranial circulation was performed prior to the retrieval of the protection device. Technical success was defined as residual stenosis of less than 30% in the treated arterial segment under optimal inflation pressure, or the restoration of satisfactory antegrade flow if precise measurement was not feasible.

All patients were monitored at scheduled clinical visits in months 1, 3, 6, and 12 during the first year following the intervention and annually thereafter. In-stent restenosis requiring revascularization was defined as a $\geq 70\%$ luminal reduction within the stented segment after the index procedure (19). To reduce the risk of thromboembolic events, all participants received dual antiplatelet therapy prior to the intervention. Aspirin was administered at a dose of 100 mg daily, and clopidogrel was prescribed at 75 mg daily following a 300 mg loading dose. After CAS, clopidogrel therapy was continued for 4-6 weeks in asymptomatic patients and for approximately three months in symptomatic patients. Aspirin treatment was maintained indefinitely across all study participants. In addition, unless a contraindication was present, high-dose statin therapy was routinely recommended for all patients.

The primary endpoint of the study was the occurrence of MACCE. This composite outcome included cardiovascular mortality, non-fatal myocardial infarction, non-fatal cerebrovascular events—such as ischemic stroke or transient ischemic attack—and in-stent restenosis requiring revascularization during the follow-up period.

Statistical Analysis

All statistical analyses were performed using IBM SPSS statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean $\pm$ standard deviation and compared using the independent samples t-test or Mann-Whitney U test, depending on the distribution assessed by the Shapiro-Wilk test. Categorical variables were presented as counts and percentages and compared using the chi-square or Fisher's exact test, as appropriate.

Univariable logistic regression analyses were conducted to identify potential predictors of MACCE. Variables with

a p-value < 0.05 in the univariable analysis were entered into multivariable logistic regression models to determine independent predictors. Two models were constructed: model A included clinical and procedural variables (age, prior endarterectomy, creatinine, uric acid, albumin, hemoglobin, and lesion length), while model B incorporated the UAR. Odds ratios (ORs) and 95% confidence intervals (CIs) were reported for all regression models. A two-tailed p-value < 0.05 was considered statistically significant.

RESULTS

A total of 332 individuals were monitored for an average of 45.30 ± 27.26 months. The mean age of the cohort was 66.90 ± 8.59 years, and 90 participants (25.6%) were female. MACCE occurred in 65 patients. When comparing those who experienced MACCE with those who did not, several differences emerged. Patients with MACCE were older (68.98 ± 8.02 years vs. 66.43 ± 8.66 years; $p = 0.030$) and had a higher prevalence of prior endarterectomy history (7.7% vs. 1.0%; $p = 0.007$). They also demonstrated elevated creatinine levels (1.37 ± 1.17 mg/dL vs. 0.96 ± 0.26 mg/dL; $p = 0.021$) and uric acid levels (7.32 ± 5.83 mg/dL vs. 5.96 ± 1.43 mg/dL; $p = 0.001$). In contrast, albumin levels (3.89 ± 0.68 g/dL vs. 4.07 ± 0.47 g/dL; $p = 0.027$) and hemoglobin levels (12.10 ± 1.96 g/dL vs. 12.95 ± 1.65 g/dL; $p = 0.001$) were significantly lower in the MACCE group. Additionally, patients who developed MACCE had longer lesion lengths (27.47 ± 6.28 mm vs. 25.24 ± 6.28 mm; $p = 0.025$), received longer stents (39.69 ± 10.45 mm vs. 36.43 ± 7.76 mm; $p = 0.013$), and had higher rates of periprocedural stroke (13.8% vs. 1.7%; $p < 0.001$) and in-hospital mortality (4.6% vs. 0%; $p < 0.001$). Clinical and procedural characteristics of the patients are presented in Tables 1 and 2.

Univariable logistic regression revealed that several clinical and procedural parameters were independent predictors of MACCE (Table 3). According to the univariable logistic regression results, age, a prior history of endarterectomy, increased creatinine and uric acid levels, and longer lesion and stent lengths were associated with a higher risk of MACCE, whereas higher albumin and hemoglobin levels were associated with a decreased risk. Multivariable logistic regression identified key predictors of MACCE across two models (Table 4). In model A, a history of endarterectomy (OR: 5.007; 95% CI: 1.015-24.702; $p = 0.048$) and elevated creatinine (OR: 2.277; 95% CI: 1.286-4.032; $p = 0.005$) were significantly associated with a higher risk of MACCE, while age, albumin, hemoglobin, uric acid, and lesion length were not significantly associated with MACCE risk. In model B, which included UAR, history of endarterectomy (OR: 4.874;

95% CI: 1.014-23.984; $p=0.049$) and creatinine (OR: 2.225; 95% CI: 1.251-3.958; $p=0.006$) remained significant, and UAR emerged as an independent predictor (OR: 1.904; 95% CI: 1.074-3.377; $p=0.028$), whereas age, hemoglobin, and lesion length were not significant. These findings indicate that a history of endarterectomy, creatinine, and UAR were relevant factors in MACCE risk stratification.

DISCUSSION

The principal finding of our investigation was that prior endarterectomy, elevated baseline creatinine, and UAR emerged as significant and independent predictors of MACCE. Notably, the strong associations of prior endarterectomy and creatinine with adverse outcomes were robust across two multivariable models, underscoring their importance as risk markers. These findings suggest that patients with a combination of prior surgery and underlying

renal dysfunction were at a particularly high risk. While univariable analysis also identified factors such as advanced age, lower hemoglobin and albumin levels, and lesion and stent lengths as predictors of MACCE, our multivariable analysis identified prior endarterectomy, creatinine, and UAR as the core factors for risk stratification in this patient cohort.

The UAR has recently been proposed as a composite biomarker that reflects interconnected mechanisms involved in the pathogenesis of atherosclerosis. Elevated serum uric acid contributes to oxidative stress, endothelial dysfunction, and vascular inflammation, all of which promote plaque initiation and progression (20). Hyperuricemia can reduce nitric oxide bioavailability, stimulate smooth muscle cell proliferation, and enhance proinflammatory cytokine activity (21). In contrast, albumin has antioxidant and anti-inflammatory properties and plays a stabilizing role in

Table 1. Clinical and biochemical variables of the patients

	All population (n=332)	MACCE (-) (n=287)	MACCE (+) (n=65)	p-value
Age, years	66.90±8.59	66.43±8.66	68.98±8.02	0.030
Gender, n (%)				0.410
Male	262 (74.4)	211 (73.5)	51 (78.5)	
Female	90 (25.6)	76 (26.5)	14 (21.5)	
Hypertension, n (%)	276 (78.6)	224 (78.3)	52 (80)	0.764
Diabetes mellitus, n (%)	143 (40.6)	115 (40.1)	28 (43.1)	0.656
Hyperlipidemia, n (%)	208 (59.1)	173 (60.3)	35 (53.8)	0.341
Smoking, n (%)	134 (38.1)	109 (38)	25 (38.5)	0.942
CAD history, n (%)	253 (71.9)	201 (70)	52 (80)	0.107
PAD history, n (%)	48 (13.6)	37 (12.9)	11 (16.9)	0.404
Presence of symptoms, n (%)	103 (29.3)	82 (28.6)	21 (32.3)	0.550
Endarterectomy history, n (%)	8 (2.3)	3 (1.0)	5 (7.7)	0.007
Glucose, mg/dL	128.93±58.14	127.79±59.34	133.94±52.65	0.246
Creatinine, mg/dL,	1.04±0.58	0.96±0.26	1.37±1.17	0.021
Uric acid, mg/dL	6.21±2.85	5.96±1.43	7.32±5.83	0.001
Albumin	4.03±0.52	4.07±0.47	3.89±0.68	0.027
CRP, mg/dL	1.70±2.33	1.66±2.38	1.85±2.15	0.284
TC, mg/dL	194.68±54.86	196.23±53.71	187.56±55.43	0.260
LDL-C, mg/dL	120.99±44.3	122.28±44.28	115.12±44.28	0.349
HDL-C, mg/dL	41.23±10.38	41.36±10.06	40.63±11.67	0.214
Triglycerides, mg/dL	163.11±91.15	162.70±80.94	165.01±128.81	0.310
Hemoglobin, g/dL	12.79±1.74	12.95±1.65	12.10±1.96	0.001
WBC, 10 ³ /μL	8.02±2.17	7.99±2.09	8.17±2.53	0.735
Platelets, 10 ³ /μL	249.17±84.33	249.56±83.55	247.42±88.34	0.543
UAR	1.58±0.91	1.48±0.39	2.01±1.91	0.001

MACCE: Major adverse cardiovascular and cerebrovascular events, CAD: Coronary artery disease, PAD: Peripheral artery disease, CRP: C-reactive protein, TC: Total cholesterol, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, UAR: Uric acid-to-albumin ratio, WBC: White blood count

maintaining endothelial integrity. Lower albumin levels may therefore indicate impaired vascular protection and heightened inflammatory burden. When assessed together, an increased UAR may signify a shift toward a pro-oxidative and pro-atherogenic state (22).

Inflammatory markers have emerged as significant predictors of clinical outcomes following CAS (23,24). This chronic inflammatory milieu promotes endothelial dysfunction, smooth muscle cell proliferation, and extracellular matrix remodeling, all of which increase the susceptibility to restenosis and thromboembolic complications after stent deployment. Furthermore, heightened inflammation can accelerate atherosclerotic progression both locally and systemically, contributing to a higher incidence of MACCE (25). In line with earlier studies, our findings indicated that an elevated UAR was associated with worse clinical outcomes in patients undergoing CAS, suggesting that this composite biomarker might reflect underlying pro-atherogenic and pro-inflammatory processes that contribute to post-stent complications and increased cardiovascular risk.

Other findings of the present study merit consideration. Patients with a history of carotid endarterectomy may have an increased risk of subsequent MACCE, as prior carotid endarterectomy does not eliminate the long-term

vulnerability to vascular events. Indeed, longitudinal data indicate that approximately 31.5% of patients who undergo carotid endarterectomy develop new major coronary events—such as myocardial infarction, coronary artery bypass grafting, or percutaneous coronary intervention—within 10 years (26). Furthermore, the presence of polyvascular disease, defined as stenosis involving multiple vascular territories, has been identified as a significant predictor of long-term MACCE in this patient population (27). Renal function has emerged as an important determinant of adverse outcomes following CAS. Elevated serum creatinine levels, even in the absence of overt renal failure, have been associated with a higher likelihood of long-term MACCE (28). A study by Donahue et al. (29) found that patients with chronic kidney disease who underwent CAS had a higher incidence of acute kidney injury and of subsequent adverse events, including myocardial infarction and stroke. Furthermore, research by AbuRahma et al. (30) demonstrated that chronic renal insufficiency, as indicated by elevated serum creatinine levels, adversely affects both early and late clinical outcomes post-CAS. These findings underscore the importance of renal function assessment in the preoperative evaluation of patients undergoing CAS, as impaired renal function may exacerbate the risk of adverse vascular events.

Table 2. Angiographic and procedural characteristics of the patients

	All population (n=332)	MACCE (-) (n=287)	MACCE (+) (n=65)	p-value
Lesion localization, n (%)				0.085
ICA	344 (97.7)	282 (98.3)	62 (95.4)	
CCA	7 (2)	5 (1.7)	2 (3.1)	
Both ICA and CCA	1 (0.3)	0 (0)	1 (1.5)	
Lesion length (mm)	25.67±6.38	25.24±6.28	27.47±6.28	0.025
Predilatation, n (%)	50 (14.2)	40 (13.9)	10 (15.4)	0.765
Stent type, n (%)				0.530
Open-cell design	113 (32.1)	90 (231.4)	23 (35.4)	
Closed-cell design	239 (67.9)	197 (68.6)	42 (64.6)	
Stent length (mm)	37.04±8.40	36.43±7.76	39.69±10.45	0.013
Stent expansion rate, %	83.12±8.45	83.29±8.37	82.42±8.79	0.633
Postdilatation, n (%)	309 (87.8)	253 (88.2)	56 (86.2)	0.861
Jumping, n (%)	31 (8.8)	23 (8)	8 (12.3)	0.289
Periprocedural MI, n (%)	2 (0.6)	1 (0.3)	1 (1.5)	0.336
Periprocedural stroke, n (%)	14 (4)	5 (1.7)	9 (13.8)	<0.001
Hospitalization period, days	3.60±4.29	3.43±4.13	4.36±4.92	0.252
In-hospital mortality, n (%)	3 (0.9)	0 (0.0)	3 (4.6)	<0.001
Follow-up (months), IQR	45.30±27.26	45.24±27.71	45.58±25.35	0.889
MACCE: Major adverse cardiovascular and cerebrovascular events, ICA: Internal carotid artery, CCA: Common carotid artery, IQR: Interquartile range, MI: Myocardial infarction				

Study Limitation

This study has several limitations that should be acknowledged. First, it is a single-center, retrospective analysis, which may limit the generalizability of the findings and introduce the potential for selection and information biases. Second, residual confounding from unmeasured variables, such as medication adherence, lifestyle factors, or procedural nuances, cannot be excluded. Finally, because this was a retrospective study, causality cannot be established; the observed associations between a history of endarterectomy, creatinine, UAR, and MACCE should be interpreted as associative rather than causal. prospective multicenter studies with larger cohorts are warranted to validate these findings and to further clarify the prognostic role of UAR in risk stratification.

Table 3. Univariable logistic regression for prediction of MACCE

	p-value	OR	95% CI
Age	0.031	1.037	1.003-1.072
Endarterectomy history	0.006	7.861	1.829-33.789
Creatinine	0.001	3.007	1.609-5.618
Uric acid	0.014	1.175	1.034-1.335
Albumin	0.013	0.524	0.314-0.875
Hemoglobin	0.001	0.759	0.650-0.888
Lesion length	0.018	1.057	1.010-1.107
Stent length	0.007	1.040	1.011-1.070
MACCE: Major adverse cardiovascular and cerebrovascular events, OR: Odds ratio, CI: Confidence interval			

Table 4. Multivariable logistic regression for prediction of MACCE

Model A			
	p-value	OR	95% CI
Age	0.242	1.026	0.983-1.071
Endarterectomy history	0.048	5.007	1.015-24.702
Creatinine	0.005	2.277	1.286-4.032
Uric acid	0.079	1.136	0.985-1.309
Albumin	0.258	0.697	0.373-1.302
Hemoglobin<	0.185	0.884	0.736-1.061
Lesion length	0.058	1.050	0.998-1.103
Model B			
Age	0.257	1.025	0.982-1.070
Endarterectomy history	0.049	4.874	1.014-23.984
Creatinine	0.006	2.225	1.251-3.958
Hemoglobin<	0.160	0.880	0.735-1.052
Lesion length	0.053	1.051	0.999-1.105
UAR	0.028	1.904	1.074-3.377
MACCE: Major adverse cardiovascular and cerebrovascular events, OR: Odds ratio, CI: Confidence interval, UAR: Uric acid-to-albumin ratio			

CONCLUSION

In conclusion, elevated UAR, prior carotid endarterectomy, and higher baseline creatinine independently predict long-term MACCE in patients undergoing carotid interventions. The findings underscore the value of UAR as a composite biomarker reflecting oxidative stress, inflammation, and vascular dysfunction, which can help identify high-risk patients and guide targeted strategies to reduce future cardiovascular and cerebrovascular events.

ETHICS

Ethics Committee Approval: The study received approval from University of Health Sciences Türkiye, Bakırköy Dr. Sadi Konuk Training and Research Hospital Clinical Research Ethics Committee (approval no: 2025-01-05, date: 24.10.2025) and adhered to the principles of the Declaration of Helsinki.

Informed Consent: Retrospective study.

FOOTNOTES

Authorship Contributions

Concept: A.K., E.O., H.A.S., D.K., F.N.T.Ç., L.Ö., Design: A.K., E.O., M.K., H.A.S., L.Ö., M.Ç., Data Collection or Processing: A.K., C.Y., E.O., M.K., D.K., F.N.T.Ç., L.Ö., M.Ç., Analysis or Interpretation: A.K., C.Y., M.K., H.A.S., D.K., F.N.T.Ç., L.Ö., M.Ç., Literature Search: A.K., C.Y., H.A.S., D.K., M.Ç., Writing: A.K., C.Y.

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REFERENCES

1. Aliotta L, Gavazzi LM, Malfa P, Foti PV, Palmucci S, Lo Greco MC, et al. Management and treatment of carotid stenosis: overview of therapeutic possibilities and comparison between interventional radiology, surgery and hybrid procedure. *Diagnostics (Basel)*. 2025;15:1679.
2. Bir SC, Kelley RE. Carotid atherosclerotic disease: a systematic review of pathogenesis and management. *Brain Circ*. 2022;8:127-36.
3. Libby P, Hansson GK. From focal lipid storage to systemic inflammation: JACC review topic of the week. *J Am Coll Cardiol*. 2019;74:1594-607.
4. Rothwell PM, Gibson R, Warlow CP. Interrelation between plaque surface morphology and degree of stenosis on carotid angiograms and the risk of ischemic stroke in patients with symptomatic carotid stenosis. On behalf of the European Carotid Surgery Trialists' Collaborative group. *Stroke*. 2000;31:615-21.
5. Nicolaides AN, Kakkos SK, Griffin M, Sabetai M, Dhanjil S, Tegos T, et al.; Asymptomatic Carotid Stenosis and Risk of Stroke (ACSRS) study group. Severity of asymptomatic carotid stenosis and risk of

- ipsilateral hemispheric ischaemic events: results from the ACSRS study. *Eur J Vasc Endovasc Surg.* 2005;30:275-84.
6. AbuRahma AF. The new 2023 European Society for vascular surgery (ESVS) carotid guidelines: the transatlantic perspective. *Eur J Vasc Endovasc Surg.* 2023;65:5-6.
7. White CJ, Brott TG, Gray WA, Heck D, Jovin T, Lyden SP, et al. Carotid artery stenting: JACC state-of-the-art review. *J Am Coll Cardiol.* 2022;80:155-70.
8. Hansson GK, Libby P. The immune response in atherosclerosis: a double-edged sword. *Nat Rev Immunol.* 2006;6:508-19.
9. Hansson GK. Inflammation, atherosclerosis, and coronary artery disease. *N Engl J Med.* 2005;352:1685-95.
10. Roche M, Rondeau P, Singh NR, Tarnus E, Bourdon E. The antioxidant properties of serum albumin. *FEBS Lett.* 2008;582:1783-7.
11. Sautin YY, Johnson RJ. Uric acid: the oxidant-antioxidant paradox. *Nucleosides Nucleotides Nucleic Acids.* 2008;27:608-19.
12. Wen S, Arakawa H, Tamai I. Uric acid in health and disease: from physiological functions to pathogenic mechanisms. *Pharmacol Ther.* 2024;256:108615.
13. Du L, Zong Y, Li H, Wang Q, Xie L, Yang B, et al. Hyperuricemia and its related diseases: mechanisms and advances in therapy. *Signal Transduct Target Ther.* 2024;9:212.
14. Duman H, İpek E, Durak H, Şahin MA, Ergül E, Yılmaz AS, et al. Uric acid to albumin ratio as a predictive marker for intracoronary thrombus severity in ST-segment elevation myocardial infarction (STEMI) patients undergoing primary percutaneous coronary intervention (PCI). *Med Sci Monit.* 2024;30:e945832.
15. Çakır Güney B, Küçük İ, Güney B, Tükel N, Serindağ Z, Önal Taştan Y, et al. The effect of uric acid/albumin ratio on prognosis of patients followed up with COVID-19 diagnosis. *Nam Kem Med J.* 2025;13:125-32.
16. Gao B, Bao Y, Meng M, Yu L, Lu Y, Sa R, et al. Association of serum uric acid with risk of stroke in US adults: a cross-sectional study from NHANES 1999-2020. *J Stroke Cerebrovasc Dis.* 2023;32:107206.
17. Sayan M, Satir Turk M, Ozkan D, Kankoc A, Tombul I, Celik A. The role of serum uric acid and uric acid to albumin ratio for predicting of lymph node metastasis in lung cancer treated surgically by vats. *Port J Card Thorac Vasc Surg.* 2023;30:31-6.
18. Bonati LH, Kakkos S, Berkefeld J, de Borst GJ, Bulbulia R, Halliday A, et al. European Stroke Organisation guideline on endarterectomy and stenting for carotid artery stenosis. *Eur Stroke J.* 2021;6:I-XLVII.
19. Garcia-Garcia HM, McFadden EP, Farb A, Mehran R, Stone GW, Spertus J, et al.; Academic Research Consortium. Standardized end point definitions for coronary intervention trials: the academic research consortium-2 consensus document. *Circulation.* 2018;137:2635-50.
20. Kuwabara M, Niwa K, Hisatome I, Nakagawa T, Roncal-Jimenez CA, Andres-Hernando A, et al. Asymptomatic hyperuricemia without comorbidities predicts cardiometabolic diseases: five-year Japanese cohort study. *Hypertension.* 2017;69:1036-44.
21. Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. *N Engl J Med.* 2008;359:1811-21. Erratum in: *N Engl J Med.* 2010;362:2235.
22. Ramirez AJ, Christen AI, Sanchez RA. Serum uric acid elevation is associated to arterial stiffness in hypertensive patients with metabolic disturbances. *Curr Hypertens Rev.* 2018;14:154-60.
23. Wijeratne T, Menon R, Sales C, Karimi L, Crewther S. Carotid artery stenosis and inflammatory biomarkers: the role of inflammation-induced immunological responses affecting the vascular systems. *Ann Transl Med.* 2020;8:1276.
24. Scalise E, Costa D, Bolboacă SD, Ielapi N, Bevacqua E, Cristodoro L, et al. The role of inflammation biomarkers in carotid artery stenosis procedures. *Ann Vasc Surg.* 2025;110(Pt A):34-46.
25. Wasser K, Schnaudigel S, Wohlfahrt J, Psychogios MN, Knauth M, Gröschel K. Inflammation and in-stent restenosis: the role of serum markers and stent characteristics in carotid artery stenting. *PLoS One.* 2011;6:e22683.
26. Go C, Avgerinos ED, Chaer RA, Ling J, Wazen J, Marone L, et al. Long-term clinical outcomes and cardiovascular events after carotid endarterectomy. *Ann Vasc Surg.* 2015;29:1265-71.
27. Thierstein L, Pereira-Macedo J, Duarte-Gamas L, Reis P, Myrcha P, Andrade JP, et al. Polyvascular disease influences long-term cardiovascular morbidity in carotid endarterectomy. *Ann Vasc Surg.* 2024;102:236-43.
28. Karbasi-Afshar R, Saburi A, Taheri S. Clinical associations between renal dysfunction and vascular events: a literature review. *ARYA Atheroscler.* 2013;9:203-9.
29. Donahue M, Visconti G, Focaccio A, Selvetella L, Baldassarre M, Viviani Anselmi C, et al. Acute kidney injury in patients with chronic kidney disease undergoing internal carotid artery stent implantation. *JACC Cardiovasc Interv.* 2015;8:1506-14.
30. AbuRahma AF, Alhalbouni S, Abu-Halimah S, Dean LS, Stone PA. Impact of chronic renal insufficiency on the early and late clinical outcomes of carotid artery stenting using serum creatinine vs glomerular filtration rate. *J Am Coll Surg.* 2014;218:797-805.