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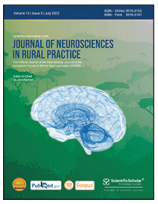

Original Article

Clinical and radiological outcomes of extracranial carotid artery stent placement: A single-center study

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ABSTRACT

Objectives: Carotid artery stenting (CAS) and carotid endarterectomy are established treatments for carotid artery stenosis. We evaluated the early and mid- to late-term clinical and radiological outcomes of patients who underwent CAS.

Materials and Methods: This retrospective study included 98 patients (112 arteries), who underwent CAS. Baseline demographics, stent types, embolic protection devices, and procedural complication rates within 30-day and 3-year post-CAS, including transient ischemic attack (TIA), stroke, death, and stent restenosis, were analyzed.

Results: The 30-day complication rates included TIA (5.1%), ipsilateral stroke (4.1%), and death (4.1%). At three-year follow-up, TIA (8.5%), ipsilateral stroke (2.1%), restenosis (1.1%), and death (6.4%) were observed. Contralateral carotid artery angiography revealed neointimal hyperplasia in two vessels (1.9%) and 70–99% restenosis 1 (1%). Notably, a significant association was observed between neointimal hyperplasia and stent geometry, with a higher incidence observed in open-cell stents compared to closed-cell stents ($P = 0.03$).

Conclusion: Our study demonstrated comparable early-term and lower mid- to late-term complication rates compared to prior studies. A multidisciplinary approach with meticulous technique, appropriate materials, and careful patient selection can optimize CAS outcomes.

Keywords: Atherosclerosis, Carotid artery stenting, Carotid stenosis, Open-cell stents, Stroke

INTRODUCTION

Stroke, the second leading cause of death worldwide, is a major contributor to disability.[1] Approximately 87% of strokes are ischemic, with 10–20% attributed to extracranial carotid artery stenosis; this stenosis is most frequently caused by atherosclerosis.[2-4] Atherosclerosis, a chronic arterial disease, is characterized by cholesterol-lipid-calcium deposition in the arterial wall.[5] It contributes to ischemic stroke by significantly narrowing the arterial lumen due to atheroma plaque growth or thrombus formation with mild narrowing.[6] Atherosclerotic plaques develop at arterial branching points, such as the carotid artery bifurcation, carotid siphon, cervical vertebral artery, and basilar artery origin.[7] Carotid artery stenosis can be symptomatic or remain asymptomatic.[5]

The management of carotid artery disease includes risk factor modification, medical therapies, such as antiplatelet and lipid-lowering agents, carotid endarterectomy (CEA), and endovascular treatment. CEA, a well-established procedure since the 1950s, has demonstrated efficacy for the treatment of symptomatic and asymptomatic stenoses ≥70%, proving superior to medical therapies.[2,8,9] However, carotid artery stenting (CAS) has become a frequent alternative in recent years.[2] Early complications following CAS include stroke, myocardial infarction, hyperperfusion syndrome, and death; mid- to late-term complications include transient ischemic attack (TIA), stroke, death, and stent restenosis.[2,5] Stents are classified as open- and closed-cell based on their free cell area.[10] Open-cell stents, with a free cell area >5 mm^2, conform well to vessel contour, facilitating delivery. However, they cover less of the lesion, potentially increasing embolization and restenosis risks as atherosclerotic material might protrude through the struts.[10,11] The growing popularity of CAS, a less invasive procedure, can be attributed to factors, such as advanced age, comorbidities, and the higher complication rates associated with surgery. We evaluated the early- and mid- to late-term clinical and radiological outcomes of patients, who underwent CAS.

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MATERIALS AND METHODS

Data collection

This retrospective study included patients aged >40 years with atherosclerotic stenosis of the extracranial carotid arteries, who underwent endovascular stent treatment following evaluation by a neurovascular council (Neurology and Radiology departments) between January 2009 and June 2012. Patients with no clinical and radiological follow-up data, no atherosclerotic carotid artery narrowing or narrowing outside the cervical segment, and those who underwent simultaneous vertebrobasilar system stenting were excluded.

We collected baseline demographic data, including the number and types of stents and embolic protection devices used during CAS. Furthermore, we evaluated complication rates within 30-day and 3-year post-CAS, including TIA, stroke, death, and stent restenosis. Restenosis was defined as luminal narrowing ≥70% in computed tomography angiography (CTA) using a 64-slice multislice computed tomography system (Somatom Sensation 64; Siemens Medical Solutions, Erlangen, Germany). Non-ionic contrast material (50 cc) was administered intravenously at 5 cm/s using the bolus-tracking technique. Images were reconstructed and analyzed using maximum intensity projections and multiplanar reconstruction methods.

The Medical Research Ethics Committee of Uludag University Faculty of Medicine (2013–11/26) approved the study protocol. The study was performed in accordance with the Declaration of Helsinki.

Carotid stenting procedure

Patients received acetylsalicylic acid (ASA) (300 mg/day) and clopidogrel (75 mg/day) for at least five to seven days before the procedure. Procedures were performed in the angiography unit using a biplane digital subtraction angiography (DSA) system (Axiom Artis; Siemens, Erlangen, Germany). A femoral artery approach was used in patients, with continuous electrocardiogram, blood pressure, and oxygen saturation monitoring. All procedures were performed under local anesthesia. A vascular sheath (6–8 F), sized appropriately for the chosen stent and technique, was inserted into the femoral artery. Patients without prior diagnostic DSA underwent aortic arch angiography, selective carotid angiography, and selective cerebral angiography to assess for sequential lesions or additional pathologies. The percentage of stenosis was calculated using the North American Symptomatic CEA Trial.

All procedures involved intra-arterial administration of 5000 U heparin. A guide catheter (Envoy 8F; Cordis Neurovascular Inc., Miami, Florida, USA) or sheath (90 cm 6–7F Destination; Terumo Medical Corporation, Tokyo, Japan) was exchanged for the diagnostic catheter and positioned in the external carotid artery using a 0.035-inch guidewire. A filter-type distal embolic protection device was deployed through the coaxial system. The filter, located at the tip of a 0.014-inch guidewire, was advanced through the guide catheter or sheath and deployed in the petrosal segment of the internal carotid artery. Pre-dilation with low-profile (3 × 20 mm) percutaneous transluminal angioplasty balloons was performed for lesions with >90% stenosis. Then, a self-expanding stent was positioned to cover the entire stenotic segment and deployed over the filter system guidewire. Post-dilation with percutaneous transluminal angioplasty balloons (5 × 20 mm or 6 × 20 mm) within the deployed stent was performed in all patients. The angiographic evaluation confirmed stent deployment and the absence of intracranial emboli before procedure completion. Patients were monitored for 24–48 h post-procedure before discharge. Intravenous heparin (1000 U) was administered hourly for the first 12 h of hospitalization, followed by a switch to low-molecular-weight heparin for five days. Long-term antiplatelet therapy included clopidogrel (75 mg/day) for six months and daily aspirin (ASA) (100 mg/day) indefinitely.

Statistical analysis

The Kolmogorov–Smirnov test was used to assess data normality, and Levene's test was used to verify the homogeneity of variance. Continuous variables are expressed as means ± standard deviations (normally distributed data) or medians with interquartile ranges (non-normally distributed data). Differences between continuous variables were analyzed using the Mann–Whitney U-test (independent samples) or the Wilcoxon test (paired samples). Categorical variables are expressed as frequencies and percentages, with differences assessed using the Chi-square test. $P < 0.05$ was considered statistically significant. Statistical analyses were performed using the Statistical Package for the Social Sciences version 20 (IBM Corp., Armonk, NY, USA).

RESULTS

Endovascular treatment was administered to 98 patients (112 arteries). The demographic data of the patients is presented in Table 1. The mean age of the patients was 63.9 ± 7 (range 40–82) years; 23.5% were females and 76.5% were males. The mean follow-up duration was 15.7 (interquartile range 7–28) months. Of the patients, 19.4% were asymptomatic, whereas 80.6% were symptomatic, presenting with TIA (29.6%), hemispheric stroke (45.9%), or amaurosis fugax (5.1%). In the symptomatic group, 61.2% and 19.4% of patients had symptoms duration <6 and >6 months, respectively.

Pre-procedural modified Rankin Scale scores were 0, 1, 2, 3, and 4 in 31 (31.6%), 46 (46.9%), 15 (15.3%), 5 (5.1%), and

Table 1: Demographic characteristics of symptomatic and asymptomatic patients*.

	Asymptomatic group	Symptomatic group	Total
Number of cases	19 (19.4)	79 (80.6)	98 (100)
Age	64±6	63±8	63.9±7
Female/Male	4 (21.1)/15 (78.9)	19 (24.1)/60 (75.9)	23 (23.5)/75 (76.5)
Hypertension	17 (17.3)	65 (66.3)	82 (83.6)
Diabetes mellitus	8 (8.2)	28 (28.6)	36 (36.8)
Hyperlipidemia	8 (8.2)	27 (27.6)	35 (35.8)
Smoking	11 (1.1)	42 (42.9)	53 (54.0)
Coronary artery disease	12 (12.2)	28 (28.6)	40 (40.8)
Peripheral artery disease	3 (3.1)	5 (5.1)	8 (8.2)

Values are expressed as *n* (%) or mean±SD. *The results are based on 112 vessels. SD: Standard deviation

1 (1.0%) patients, respectively. During mid- to late-term follow-up, modified Rankin Scale scores were 0, 1, 2, 3, 4, and 5 or 6 in 32 (32.7%), 50 (51.1%), 7 (7.1%), 1 (1.0%), 1 (1.0%), and 7 (7.1%) patients, respectively.

Post-procedure medications included ASA (100–300 mg) in 46 patients (46.9%), clopidogrel in 14 (14.3%), ASA + clopidogrel in 19 (19.4%), ASA + dipyridamole in 1 (1.0%), clopidogrel + dipyridamole in 1 (1.0%), warfarin in 4 (4.1%), and ASA + warfarin in 1 (1.0%). Type 1 (Protege/open-cell) stents were implanted in 57 vessels (50.9%), whereas type 2 (Xact/closed-cell) stents were implanted in 55 vessels (49.1%). Pre-dilation was performed in 87 vessels (77.7%) and omitted in 25 (22.3%). Filters were used in 107 vessels (95.5%), spiders in 2 (1.8%), and microglide in 1 (0.9%); no filters were used in two vessels. Post-dilation was performed in 110 vessels (98.2%). Notably, 14 patients underwent bilateral carotid stenting (10 in a single session and 4 in separate sessions).

Over a 30-day period, five patients (5.1%) experienced TIA, four (4.1%) suffered ipsilateral strokes, two (2.0%) had intracranial hemorrhages, and one (1.0%) experienced gastrointestinal bleeding. In total, four patients (4.1%) died during this period, including three from stroke and one from intracranial hemorrhage. At three-year follow-up, eight patients (8.5%) had experienced TIAs, two (2.1%) had ipsilateral strokes, one (1.1%) had restenosis requiring balloon revascularization, and six patients (6.4%) died during follow-up including two from stroke and four from other causes. Notably, no patients in the asymptomatic group experienced TIA, stroke, or death (P = 0.03). Therefore, 88 patients underwent follow-up imaging (101 vessels; [Table 2]). Ipsilateral CTA revealed normal stent patency in 88 vessels (87.1%), neointimal hyperplasia in 12 (11.8%), and 50–69% stenosis in 1 (1%). Contralateral CTA revealed normal stent patency in 24 vessels (23.8%), neointimal hyperplasia in 2 (1.9%), no stenosis in 13 (12.9%), 0–49% stenosis in 34 (33.7%), 50–69% stenosis in 9 (8.9%), 70–99% stenosis in 1 (1%), and occlusion in 18 (17.8%).

Table 2: Follow-up CTA examination of the vessels*.

	Ipsilateral CTA	Contralateral CTA
Normal stent patency	88 (87.1)	24 (23.8)
Neointimal hyperplasia	12 (11.8)	2 (1.9)
No stenosis	-	13 (12.9)
0–49% stenosis	-	34 (33.7)
50–69% stenosis	1 (1)	9 (8.9)
70–99% stenosis	-	1 (1)
Occlusion	-	18 (17.8)

Values are expressed as *n* (%).*The results are based on 101 vessels. CTA: Computed tomography angiography

The development of neointimal hyperplasia was not significantly associated with age, sex, antiplatelet use, symptom duration, smoking, hypertension, diabetes mellitus, hyperlipidemia or coronary artery disease (P > 0.05). However, a significant association was observed between neointimal hyperplasia and stent geometry, with a higher incidence observed in open-cell stents compared to closed-cell stents (P = 0.03).

DISCUSSION

The CEA remains the recommended first-line treatment for carotid artery stenosis.[12] However, CAS is a viable alternative, particularly with technological advancements and increasing procedural expertise. The CAS offers advantages over CEA, including its minimally invasive nature, shorter hospital stays, and applicability for high-risk patients. Although early CAS studies have demonstrated higher rates of emboli and associated ischemia compared to CEA, the introduction of newer devices and techniques has significantly reduced the risk of procedure-related embolic events.[13]

The CAS can be associated with early-term complications, including stroke, myocardial infarction, and death. The CAVATAS and CREST studies, including patients with symptomatic and asymptomatic stenosis >50%, reported 30-day complication rates of 10% and 5.2%, respectively.[14] Our study demonstrated a lower 30-day stroke or death rate of 4.1%, with

no observed cases of myocardial infarction. Hyperperfusion syndrome, a rare but significant complication of carotid stenting, can lead to severe morbidity and mortality in the early stages.[15,16] A healthy brain possesses an autoregulation system that balances intracranial pressure during blood pressure fluctuations.[16] Chronic stenosis can impair cerebral autoregulation, leading to uncontrolled blood flow when the stenosis is suddenly relieved.[17] Hypertension, often a response to hypotension triggered by balloon stimulation of carotid baroreceptors, can contribute to hypoperfusion syndrome.[18] Risk factors for hypoperfusion syndrome include high-degree carotid artery stenosis, poor collateral blood flow, post operative hypertension, hyperperfusion, and impaired cerebrovascular reserve.[16] This syndrome can result in subarachnoid or intracerebral hemorrhage, presenting with symptoms, such as ipsilateral headache, seizure, focal neurological deficit, and hypertension. A meta-analysis reported a 1.2% incidence of hyperperfusion syndrome in CAS patients, with a 0.7% rate of intracranial hemorrhage.[17] Consistent with prior studies, we found a 2% incidence of hyperperfusion syndrome and related intracranial hemorrhage.

Ipsilateral stroke is the most frequent mid- to late-term complication of CAS. Several studies have demonstrated a higher stroke risk in the initial years after stenting, followed by a gradual decrease; the CAVATAS study reported a three-year stroke risk of 14.3%,[19] the CREST study a four-year risk of 7.2%,[20] and the ACST-2 study a five-year risk of 5.3%.[21] Conversely, we observed a lower ipsilateral stroke rate of 2.1% over a 15-month follow-up; this may be attributable to appropriate patient selection, effective patient care support, and procedural expertise. Restenosis is another complication, with reported rates of 6% at 2 years and 12.2% at 10 years in the CREST study,[22] 12.5% at 3 years in the EVA-3S study,[23] and 6.9% at 1 year and 10.8% at 5 years in the international carotid stenting study.[20] Compared to other studies, we noted a significantly lower restenosis rate of 1%; this might be attributable to the shorter duration of our study. Early stent restenosis (in the first few years) arises from vascular damage, neointimal hyperplasia, and vascular remodeling, whereas late restenosis primarily results from the progression of carotid atherosclerosis.[24] We observed neointimal hyperplasia, indicative of smooth muscle cell proliferation and migration after stenting, in only two vessels; this suggests a significant association between stent geometry and restenosis risk. Open-cell stents, with less atherosclerotic material coverage, are more prone to restenosis compared to closed-cell stents. This can lead to material prolapse from stent struts, increasing the risk of embolization and in-stent restenosis.[10] Several studies have demonstrated a significant association between open-cell stents and late-term complications,[25,26] whereas others have found no association between stent geometry and complications.[10,27]

The large free cell area of open-cell stents (free cell area Xact = 2.74, Protege = 10.71 mm^2) can contribute to their increased susceptibility to restenosis by providing insufficient scaffolding support to the vessel wall.[28]

Our study had several limitations, including its retrospective design, relatively small sample size, unequal sex distribution, and short follow-up period that limits our assessment of long-term outcomes.

CONCLUSION

We found comparable early-term and lower mid- to late-term complication rates compared to prior studies. There was an association between neointimal hyperplasia and stent geometry, with a higher incidence in open-cell stents than in closed-cell stents. A multidisciplinary approach with meticulous technique, appropriate materials, and careful patient selection can optimize CAS outcomes.

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Ethical approval

The research/study was approved by the Institutional Review Board at The Medical Research Ethics Committee of Uludag University Faculty of Medicine, number 2013–11/26, dated 2013–11/26.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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