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Anatomical and technical predictors of perioperative clinical outcomes after carotid artery stenting

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Abstract

Background—A few other studies have reported the effects of anatomical and technical factors on clinical outcomes of carotid artery stenting (CAS). This study analyzed the effect of these factors on perioperative stroke/myocardial infarction/death after CAS.

Methods—This was a retrospective analysis of prospectively collected data of 409 of 456 patients who underwent CAS during the study period. A logistic regression analysis was used to determine the effects of anatomical and technical factors on perioperative stroke, death, and myocardial infarction (major adverse events [MAEs]).

Results—The MAE rate for the entire series was 4.7% (19 of 409), and the stroke rate was 2.2% (9 of 409). The stroke rate for asymptomatic patients was 0.46% (1 of 218; $P = .01$). The MAE rates for patients with transient ischemic attack (TIA) were 7% (11 of 158) vs 3.2% (8 of 251) for other indications ($P = .077$). The stroke rates for heavily calcified lesions were 6.3% (3 of 48) vs 1.2% (4 of 332) for mildly calcified/noncalcified lesions ($P = .046$). Differences in stroke and MAE rates regarding other anatomical features were not significant. The stroke rate for patients with percutaneous transluminal angioplasty (PTA) before embolic protection device (EPD) insertion was 9.1% (2 of 22) vs 1.8% (7 of 387) for patients without ($P = .07$) and 2.6% (9 of 341) for patients with poststenting PTA vs 0% (0 of 68) for patients without. The MAE rate for patients with poststenting PTA was 5.6% (19 of 341) vs 0% (0 of 68) for patients without ($P = .0536$). The MAE rate for patients with the ACCUNET (Abbott, Abbott Park, Ill) EPD was 1.9% (3 of 158) vs

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AUTHOR CONTRIBUTIONS

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Data collection: TD, NH, EA, JA
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6.7% (16 of 240) for others (P = .029). The differences between stroke and MAE rates for other technical features were not significant. A regression analysis showed that the odds ratio for stroke was 0.1 (P = .031) for asymptomatic indications, 13.7 (P = .014) for TIA indications, 6.1 (P = .0303) for PTA performed before EPD insertion, 1.7 for PTA performed before stenting, and 5.4 (P = .0315) for heavily calcified lesions. The MAE odds ratio was 0.46 (P = .0858) for asymptomatic indications, 2.1 for PTAs performed before EPD insertion, 2.2 for poststent PTAs, and 2.2 (P = .1888) for heavily calcified lesions. A multivariate analysis showed that patients with TIA had an odds ratio of stroke of 11.05 (P = .029). Patients with PTAs performed before EPD insertion had an OR of 6.15 (P = .062). Patients with heavily calcified lesions had an odds ratio of stroke of 4.25 (P = .0871). The MAE odds ratio for ACCUNET vs others was 0.27 (P = .0389).

Conclusions—Calcific lesions and PTA before EPD insertion or after stenting were associated with higher stroke or MAE rates, or both. The ACCUNET EPD was associated with lower MAE rates. There was no correlation between other anatomical/technical variables and CAS outcome.

Carotid artery stenting (CAS) has become an alternative to carotid endarterectomy (CEA) and minimally invasive therapy for significant carotid stenosis, particularly in high-risk surgical patients. Currently, ~9000 CAS procedures are performed annually in the United States.[1] With the publication of the Carotid Revascularization Endarterectomy vs stenting Trial (CREST),[2] a significant proportion of patients with severe carotid artery stenosis have undergone CEA or CAS with somewhat similar results.[2] Better selection of high-risk surgical patients or high-risk CAS patients is critical in providing the best therapy for each individual patient.

Because percutaneous transluminal angioplasty (PTA) is a key component of CAS procedures and carries significant embolic risk,[3–7] its use before or after stent deployment is being scrutinized. Only a few studies have reported the effects of anatomical and technical factors on the clinical outcomes of CAS. This study analyzed the effect of these factors on perioperative stroke and on stroke, myocardial infarction (MI), and death (major adverse events [MAEs]) after CAS.

METHODS

Between January 2004 and October 2015, 456 CAS procedures were performed at our institution, and this was a retrospective analysis of prospectively collected data of 409 of these CAS patients. We excluded 47 CAS patients because of missing data or no embolic protection device (EPD) was used. This study was approved by the Institutional Review Board of West Virginia University/Charleston Area Medical Center. Informed consent was not necessary because all data were anonymous and no personal patient information was identified.

Every effort was made to identify missing data (anatomical/technical factors) by a review of the electronic medical records and progress notes at our Vascular Center of Excellence. Indications for CAS were asymptomatic patients with ≥80% carotid artery stenosis and symptomatic patients (transient ischemic attacks [TIAs] and stroke) with ≥50% ipsilateral carotid stenosis by conventional arteriography or ≥70% stenosis by duplex ultrasound

imaging in our Intersocietal Accreditation Commission-accredited vascular laboratory, computed tomography angiography, or magnetic resonance angiography.

All patients were treated with aspirin (325 mg) and clopidogrel (75 mg) for 48 to 72 hours before the procedure. Clopidogrel was continued for 30 days after the procedure, whereas aspirin therapy was continued indefinitely.

All CAS procedures were done according to their carotid trial protocol and using cerebral protection devices. Overall, CAS was selected for high-risk surgical patients, which included physiological high-risk (severe coronary artery disease, congestive heart failure with ejection fraction of <30%, cardiopulmonary disease, etc) or anatomical high-risk (post-CEA restenosis, high cervical lesions, tracheostomy, neck irradiation, or past cranial nerve injury).[8] It should be noted that the number of CEAs performed in our tertiary medical center (950 beds) averaged 300 to 400 annually during the past 10 years. All demographic and clinical characteristics were recorded.

Specific anatomical factors that may affect perioperative CAS outcomes were also recorded. These included lesion length, lesion location, severity, calcification, and aortic arch type. Lesion length was measured as the distance, in millimeters, from the proximal to the distal shoulder of the lesion. Lesion location was defined at the distal common carotid artery or proximal internal carotid artery (ICA), including the bulb, or a combination of the distal common carotid and ICA (bifurcational lesion). They were also classified as right carotid or left carotid lesions. Severity of carotid stenosis was classified as 50% to 69% or 70% to 99%. Target site calcification was defined as no or mild calcification vs heavily calcified lesions (>50% circumferential calcification), which was based primarily on computed tomography angiography or ultrasound imaging, or both.

Aortic arch types were defined as type I arch when the vessels arise from the top of the arch, type II when they arise between parallel planes delineated by the outer and inner curves of the arch, and type III when they arise caudal to the inner surface of the arch or the ascending aorta. CAS was done as a primary procedure or for post-CEA stenosis (the CEA in 44 of 147 patients was done in <24 months).

The following technical factors were also identified and recorded: stent type (RX Acculink [Abbott, Abbott Park, Ill], Xact [Abbott], and others), number of stents (1 vs ≥2), stent length, stent diameter, EPD type (RX ACCUNET [Abbott], Emboshield NAV6 [Abbott], and others), EPD insertion to recovery time, pre-PTA before deployment of the distal EPD, pre-PTA before stenting, PTA after stenting, or any combination of pre-PTA or post-PTA. Prestenting PTA was done using 3- to 4-mm Viatrac balloons (Abbott) and poststenting PTA using 5-mm balloons. No specific balloons were used for calcific lesions in this location.

Excluded from the analysis were 47 CAS in patients with significant stenosis in the proximal common carotid artery or patients without the use of EPD.

Study end points

The primary end point of the study was 30-day perioperative stroke and the composite outcome of MAEs, which included stroke, MI, or death, or a combination of these.

Periprocedural neurologic evaluations were performed before and immediately after CAS and at 24 to 48 hours after CAS. Minor stroke was defined as a neurologic deficit lasting >24 hours, resulting in a grade I or II Rankin scale, and a major stroke as grades III to V Rankin scale. Stroke was also referred to as an ipsilateral stroke if it affected the same cerebral hemisphere of the carotid intervention or a contralateral stroke if it affected the contralateral cerebral hemisphere.

Cardiac enzyme levels and electrocardiograms were obtained for all patients who were a part of carotid clinical trials before the procedure and at 24 to 48 hours postoperatively. MI was defined as electrocardiogram evidence of ischemia or patients with biomarker elevations with chest pain. Every effort was made to follow the Society for Vascular Surgery Reporting Standards for Carotid Intervention.[8]

Statistical analysis

A statistical analysis was performed using SAS 9.2 software (SAS Institute, Inc, Cary, NC). A univariate analysis was performed on the effect of patient clinical and demographic characteristics and anatomical and technical variables on perioperative outcome. Continuous data are reported as means with a standard deviation. The 30-day stroke and MAE rates were compared between various anatomical and technical variables. A logistic regression analysis was also used to examine the correlation between these factors and outcomes, adjusting for indications for CAS and various patient clinical and demographic characteristics. The threshold for significance was 0.05.

RESULTS

Demographic and clinical predictors of perioperative stroke/MAE

Of 456 CAS procedures that were performed, 409 were analyzed. The perioperative stroke rate for the entire series was 2.2% (9 strokes: 8 ipsilateral, 1 contralateral; 7 minor and 2 major) and the MAE rate was 4.7% (19 MAEs; 4 deaths, 9 strokes, and 6 MIs). The mean age was 71.1 years (range, 51–84; median, 74 years) and 68.4 years for those without a perioperative stroke (range, 40–88; median, 69; $P = .345$). The mean age was 71.5 years (range, 51–84; median, 73 years) for patients who had perioperative MAEs and 68.3 years for patients without perioperative MAEs (range, 40–88; median, 69 years; $P = .1469$).

Table I summarizes the demographic and clinical characteristics in the entire series (409 CAS procedures). Table II summarizes the correlation between demographic and clinical characteristics and perioperative stroke and MAE rates. As noted, the stroke rate for asymptomatic patients was 0.46% vs 4.2% for symptomatic patients ($P = .0144$). Meanwhile, the MAE rate for TIA patients was 7% vs 3.2% for other indications ($P = .0774$) and was also higher for nonsmokers ($P = .0297$).

Because this study reported our 12-year experience during which new devices were introduced, we analyzed the results according to three periods: 2001 to 2005, 2006 to 2010, and 2011 to 2015. The perioperative stroke rates were 2.6% (2 of 77), 3.2% (5 of 156), and 1.1% (2 of 176), respectively ($P = .4145$), and MAE rates were 2.6% (2 of 77), 4.5% (7 of 156), and 5.7% (10 of 176), respectively ($P = .5586$).

Anatomical predictors of perioperative stroke/MAEs

Table III summarizes the anatomical predictors of peri-operative stroke and MAE rates. As noted, the stroke rate for heavily calcified lesions was 6.3% vs 1.2% for non-calcified (or mildly calcified) lesions ($P = .046$). Forty-three of 250 patients (17.2%) with primary CAS had heavily calcified lesions vs 4 of 129 (3.1%) for CAS for post-CEA restenosis ($P < .0001$). There were no significant differences in stroke and MAE rates in regards to other anatomical features.

The distribution of lesion length was 46 lesions (11.4%) were <10 mm, 165 (41%) were between 10 and <15 mm, 19 (4.7%) were between 15 and <20 mm, 116 (29%) were between 20 and <30 mm, and 57 (14.1%) were ≥30 mm. The mean lesion length for patients with perioperative strokes was 16.8 mm (range, 10–30 mm) vs 17.2 mm for patients who had no strokes (range, 5–65 mm; $P = .8792$). The mean lesion length for patients with MAEs was 15.5 mm (range, 5–39 mm) vs 17.2 mm (range, 5–65 mm) for patients without MAEs ($P = .2616$). The mean percentage of stenosis for patients with peri-operative strokes was 84% (range, 60%–99%) vs 82.2% (range, 50%–99%; $P = .4877$) for patients without strokes. Meanwhile, the mean percentage of stenosis for patients with MAE was 81% (range, 60%–99%) vs 82.3% (range, 50%–99%; $P = .6522$) for patients without MAE.

There were also no significant differences in perioperative stroke and MAE rates according to lesion location (right vs left carotid); however, left ICA lesions had a trend toward higher stroke rates (3.1% vs 1.5%, $P = .4584$) and MAE rates (6.2% vs 2.9%, $P = .1808$).

Technical predictors of perioperative stroke/MAEs

Table IV summarizes various technical factors that may affect perioperative stroke/MAE rates. As noted, the stroke rate for patients with PTA before EPD insertion was 9.1% vs 1.8% for patients without ($P = .0791$) and 2.6% for patients with poststenting PTA vs 0% for patients without ($P = .3666$). The MAE rate for patients with poststenting PTA was 5.6% vs 0% for patients without ($P = .0536$). MAE rates for patients with ACCUNET EPDs was 1.9% vs 6.7% for all other EPDs combined ($P = .0291$). There were no significant differences in stroke and MAE rates in regards to stent type, number of stents used, stent diameter, stent length, and time from EPD insertion to recovery time.

The mean time between the deployment of the EPD and recovery was 14.7 minutes (range, 9–23 minutes) for patients who had perioperative strokes vs a mean of 14.9 minutes (range, 4–51 minutes; $P = .8518$) for patients without perioperative strokes. Meanwhile, the mean recovery time for patients who had MAEs was 13 minutes (range, 5–23 minutes) vs 14.9 minutes (range, 4–51 minutes; $P = .3548$) for patients without MAEs.

To be noted, the technical factors for primary CAS and CAS for post-CEA restenosis were similar, except that ACCUNET EPDs were used in 32.4% of patients for primary CAS vs 52.1% for CAS for post-CEA restenosis ($P < .001$), and stent length of ≥30 mm was used in 78.5% in primary CAS vs 56.2% for CAS for post-CEA restenosis ($P < .001$). There were no significant differences between both groups in PTA before EPD insertion or post-PTA stenting.

Table V summarizes the correlation of early stroke/MAE rates and various PTAs, whether before the deployment of the EPD, prestenting insertion, poststenting, or a combination of the above.

Regression analysis

Table VI summarizes univariate and multivariate analyses for predictors of early stroke and MAE rates. Regression univariate analysis showed that the odds ratio for stroke was 0.1 (P = .031) for asymptomatic indications, 13.7 (P = .014) for TIA indications, 6.1 (P = .0303) for PTAs performed before EPD insertion, 1.7 (P = .4413) for PTA performed before stenting, and 5.4 (P = .0315) for heavily calcified lesions. It also showed that the MAE odds ratio was 0.46 (P = .0858) for asymptomatic indications, 2.1 (P = .35) for PTAs performed before EPD insertion, 2.2 (P = .2232) for poststent PTAs, and 2.2 (P = .1888) for heavily calcified lesions. Multivariate analysis showed that TIA indications had an odds ratio of stroke of 11.05 (P = .029), and PTA performed before EPD insertion had an odds ratio of stroke of 6.15 (P = .062), whereas heavily calcified lesions had an odds ratio of stroke of 4.25 (P = .0871). The MAE odds ratio for ACCUNET EPD vs other filters was 0.27 (P = .0389).

DISCUSSION

A comprehensive search of MEDLINE from January 1, 1996, through December 31, 2011, by Khan and Qureshi[1] showed that clinical factors, including age >80 years, symptomatic status, diabetes mellitus, chronic renal failure, procedures done ≤2 weeks of symptoms, and hemispheric TIA were associated with 30-day perioperative stroke and death. They also found that certain angio-graphic factors, including left carotid artery intervention, ulcerated/calcified plaques, lesion length >10 mm, stenosis >90%, ostial involvement, aortic arch type III, aortic arch calcification, and ICA/common carotid artery angulation >60° were predictors of increasing perioperative stroke. Other technical factors that were associated with increasing perioperative stroke include the use of multiple stents, PTA without EPDs, and PTA before stent placement. Meanwhile, intraprocedural use of EPDs and closed-cell vs open-cell stent design were not associated with 30-day perioperative stroke or death, or both.[1]

Our present study showed that the 30-day perioperative stroke rate for asymptomatic patients was significantly lower than for symptomatic patients (0.46% vs 4.2%; P = .0144). Several other carotid stent trials have shown that patients with symptomatic disease have higher 30-day perioperative stroke or MAE rates, or both, compared with asymptomatic patients.[2,9–12] However, a few studies have reported anatomical and technical factors and their effect on the clinical outcomes of CAS. Our study analyzed the effect of these factors on perioperative stroke and MAEs of CAS.

Anatomical predictors of CAS outcome

Naggara et al[13] reported the results of a pooled analysis of 34,398 CAS patients and showed that CAS for left ICA stenosis was associated with higher 30-day perioperative stroke/death rates compared with CAS for right carotid artery stenosis (7.5% vs 6%). They suggested that

this higher rate was secondary to the difficult access from the aortic arch to the left common carotid artery. However, other studies have not found a significant difference in 30-day stroke or death rates, or both, between right-and left-sided CAS.[14,15]

Our present study showed that the 30-day perioperative stroke rate for all left-sided lesions was 2.6% vs 1.7% for right carotid lesions ($P = .7371$) and that the 30-day MAE rate was 6.1% for left carotid lesions vs 2.8% for right carotid lesions ($P = .1164$). The 30-day stroke rate for left ICA lesions was 3.1% vs 1.5% for right ICA lesions ($P = .4584$) and the 30-day MAE rate was 6.2% for left ICA lesions vs 2.9% for right ICA lesions ($P = .1808$). As noted, there was a trend toward lower stroke and MAE rates in right carotid lesions compared with left carotid lesions; however, the P value was not statistically significant, which can be explained by the smaller sample size.

It has also been noted that patients with aortic arch class III had a higher 30-day perioperative stroke rate of 17.2% vs 8.1% for patients with aortic arch classes I and II in the Endarterectomy vs Angioplasty in Patients With Symptomatic Severe Carotid Stenosis (EVA-3S) trial.[13] Our study did not show any significant difference according to aortic arch type; however, most of our patients were done for aortic arch type I and II; with only 32 patients done for aortic arch type III.

Target site calcification has also been correlated with a higher 30-day stroke rate. A single-center study by Setacci et al[16] noted that the presence of target site calcification was associated with a higher 30-day perioperative stroke rate of 6.5% in contrast to 2.3% in patients without calcification. Similar findings were noted in our present study, with a stroke rate for heavily calcified lesions of 6.3% vs 1.2% for noncalcified or mildly calcified lesions ($P = .046$).

A few studies have reported the correlation of the severity of carotid stenosis and perioperative CAS outcomes. Gray et al[14] and Chaturvedi et al[15] both reported no difference in the mean severity of carotid stenosis and perioperative outcome. Similar findings were noted in our study. However, a single-center study report by Mathur et al[17] showed that CAS performed for lesions with >90% stenosis was associated with a higher 30-day stroke rate of 14.9%, compared with 3.5% in patients with lesion severity of <90%.

A few other studies[15,17,18] have analyzed the correlation of CAS outcome and the target lesion length and concluded that longer lesions were associated with a higher 30-day perioperative stroke rate, particularly in octogenarians, as seen in the Carotid ACCULINK/ACCUNET Post- Approval Trial to Uncover Unanticipated or Rare Events (CAPTURE) 2 trial.[15] Mathur et al[17] reported a 30-day stroke rate of 11.4% for lesions longer than 10 to 15 mm vs 3.8% for lesions shorter than 10 mm; whereas Sayeed et al[18] reported a stroke rate of 17% vs 2.1%, and Setacci et al[16] reported a stroke rate of 5.6% vs 2.6% for these lesions, respectively.

Long lesions have been postulated to have a higher atherosclerotic burden, which leads to a higher risk of dislodgement of emboli during PTA or stent placement, or both. Recently, Moore et al[19] reported the carotid angiographic characteristics in the CREST trial and the major contributors to periprocedural stroke and death differences between CAS and CEA.

They found that lesion length and lesions that were contiguous or sequential and noncontiguous extending remote from the bulb were identified as influencing CAS to CEA stroke and death treatment difference. For longer lesion lengths (≥12.85 mm), the risk of CAS was higher than that of CEA (odds ratio, 3.42). They concluded that a higher stroke and death rate for those treated with CAS appeared to be largely isolated to those with longer lesions or those with sequential and remote lesions, or both. Our present study did not show any significant correlation according to lesion length, which may be attributed to the small sample size or because most of the treated lesions were <20 to 30 mm in length.

Technical predictors of CAS outcome

Number, length, and types of stents—Stent design or the type of stent and its effect on CAS outcome has been controversial. A multicenter study of 3179 patients concluded that a free-cell area of >7.5 mm was associated with a higher 30-day stroke rate of 1.3% vs 3.4%, suggesting that closed-cell designed stents may be associated with a lower rate of stroke[20]; however, in contrast, data from the Society for Vascular Surgery Registry did not show any significant difference in CAS outcome, whether using open-cell or closed-cell stent designs.[21] Timaran et al,[22] in a recent randomized controlled trial of 40 CAS patients treated by closed-cell or open-cell design, found no significant difference in embolic events, as documented by diffusion-weighted magnetic resonance imaging and transcranial Doppler.

Our present study showed no significant differences in stroke and MAE rates in regards to stent type, number of stents used, stent diameter, and stent length. To be noted, only 24 patients in our series had more than one stent. However, the CAPTURE registry study reported that the use of multiple carotid stents was associated with a higher 30-day perioperative stroke rate of 9.7%, compared with 4.5% in patients with only one stent placement.[13] Theoretically, that can be explained by the fact that multiple stents are a marker of lesion length, which is generally associated with a higher rate of stroke events.

PTA before EPD insertion, pre-PTA or post-stenting PTA, or both—Our study showed that the stroke rate for patients with PTA before EPD insertion was 9.1% vs 1.8% for patients without and was 2.6% for patients with post-stenting PTA vs 0% for patients without. The MAE rate for patients with post-stenting PTA was 5.6% vs 0% for patients without ($P = .0536$). As noted, these results showed a significant trend toward a higher rate of stroke in patients who underwent PTA before filter insertion and in MAE rates in patients who had poststenting PTA. Our study also showed that the MAE rate for patients with the ACCUNET filter was 1.9% vs 6.7% for all other filters combined ($P = .0291$).

A regression analysis model showed that the odds ratio of stroke was 6.1 ($P = .0303$) for PTA performed before filter insertion, and 1.7 for PTA performed before stenting. It also showed a MAE odds ratio of 2.1 for PTA before filter insertion and 2.2 for poststent PTA.

A multivariate analysis also showed that PTA performed before EPD insertion had an odds ratio of stroke of 6.15 ($P = .062$). These findings only echo the fact that PTA before filter insertion or before stenting, or both, and after stenting was associated with a significant adverse event rate.

The CAPTURE registry study[14] noted that pre-PTA without EPD was associated with a higher 30-day stroke rate of 15.4% compared with a stroke rate of 4.3% in patients without pre-PTA. Similarly, Theiss et al[23] showed that the pro-CAS registry data indicated that pre-PTA led to a higher periprocedural stroke rate of 4.1% vs 3%. Obeid et al[7] recently completed a retrospective analysis of CAS patients between 2005 and 2014 in the Vascular Quality Initiative Database of 3772 patients and reported that poststent PTA was associated with an increased periprocedural stroke and death rate of 3% overall, with a 2.4-fold increase in perioperative stroke and death rates when prestent PTA only technique was compared with prestenting PTA and poststenting PTA. Poststenting PTA is generally believed to result in increased embolic showering and hemodynamic depression that may persist into the postoperative period.

Vos et al[3] demonstrated that microembolization can occur during all phases of the CAS procedure, starting with the wiring, the EPD insertion, prestenting PTA, stenting, and poststenting PTA, as demonstrated by transcranial Doppler. It has also been suggested that the most showering occurs during the poststenting PTA, because PTA fractures the plaque and PTA may facilitate pushing some of the embolic material through the stent mesh.[7]

Our study has some limitations, including being a single-center study, which limits the sample size. It is also a retrospective data analysis of prospectively collected data, and reporting bias could lead to some errors. As indicated in the Results, the stroke rate was four times higher in patients with primary CAS vs CAS for post-CEA restenosis, which may be partially explained by the fact that there were more patients with calcific lesions in primary CAS; however, this stroke rate was not statistically significant, which can be explained by the sample size.

CONCLUSIONS

Calcific lesions and PTA before EPD insertion or post-stenting PTA were associated with a higher stroke or MAE rate, or both, after CAS. The ACCUNET filter was associated with lower MAE rates. No correlation was found between other anatomical and technical variables and CAS outcome in our study.

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HIGHLIGHTS

- **Type of Research:** Retrospective analysis of a prospectively collected single-center database
- **Take Home Message:** In 409 patients, including 191 with symptoms, carotid artery stenting resulted in a stroke rate of 2.2%. It was 6.3% for heavily calcified lesions, 9.1% in those who received angioplasty before embolic protection device placement and 2.6% in those who had angioplasty after stenting.
- **Recommendation:** This study suggests that heavily calcified carotid lesions and angioplasty before embolic protection device placement or after stent placement are risk factors for stroke and major adverse events after carotid artery stenting.

Table I

Demographic and clinical characteristics in the entire series (409 patients)

Variable	No. (%) or mean (range) (N = 409)
Age, years	68.5 (40–88)
Male gender	241 (59)
Coronary artery disease	301 (74)
Hypercholesterolemia	321 (78)
Hypertension	365 (89)
Diabetes	182 (45)
Congestive heart failure	104 (25)
Chronic renal insufficiency	93 (23)
Smoking	248 (61)
Indication	
Asymptomatic	218 (53)
TIA	158 (39)
Stroke	45 (11)

TIA, Transient ischemic attack.

Table II

Demographic and clinical characteristics, perioperative stroke, and major adverse events (*MAEs*)

Parameter	Stroke, No. (%)	*P* value	MAEs, No. (%)	*P* value
Gender				.568
Male	5/236 (2.1)	1	10/241 (4.2)	
Female	4/168 (2.4)		9/168 (5.4)	
Coronary artery disease		1		.9927
Yes	7/301 (2.3)		14/301 (4.7)	
No	2/108 (1.9)		5/108 (4.6)	
Hypercholesterolemia		1		1
Yes	7/321 (2.2)		15/321 (4.7)	
No	2/88 (2.3)		4/88 (4.6)	
Hypertension		1		.4444
Yes	8/365 (2.2)		16/365 (4.4)	
No	1/44 (2.3)		3/44 (6.8)	
Diabetes		1		.7966
Yes	4/182 (2.2)		9/182 (5)	
No	5/227 (2.2)		10/227 (4.4)	
Congestive heart failure		.6986		1
Yes	3/104 (2.9)		5/104 (4.8)	
No	6/305 (2)		14/305 (4.6)	
Chronic renal insufficiency		1		1
Yes	2/93 (2.2)		4/93 (4.3)	
No	7/316 (2.2)		15/316 (4.8)	
Smoker		.1636		.0297
Yes	3/248 (1.2)		7/248 (2.8)	
No	6/161 (3.7)		12/161 (7.5)	
Indication				
Asymptomatic		.0144		.1409
Yes	1/218 (0.46)		7/218 (3.2)	
No	8/191 (4.2)		12/191 (6.3)	
TIA		.0027		.0774
Yes	8/158 (5.1)		11/158 (7)	
No	1/251 (0.4)		8/251 (3.2)	
Stroke		1		1
Yes	1/45 (2.2)		2/45 (4.4)	
No	8/364 (2.2)		17/364 (4.7)	

TIA, Transient ischemic attack.

Table III

Anatomical predictors of perioperative stroke and major adverse events (*MAEs*)

Predictor	Stroke, No. (%)	*P* value	MAE, No. (%)	*P* value
Lesion location				
All lesions		.7371		.1164
Left side[a]	6/230 (2.6)		14/230 (6.1)	
Right side[a]	3/179 (1.7)		5/179 (2.8)	
ICA lesions only		.4584		.1808
Left side	5/161 (3.1)		10/161 (6.2)	
Right side	2/137 (1.5)		4/137 (2.9)	
Primary CAS	8/261 (3.1)	.1656	15/261 (5.8)	.1637
CAS for post-CEA	1/147 (0.7)		4/147 (2.7)	
Preoperative stenosis		.4626		.3616
50%–69%	1/27 (3.7)		2/27 (7.4)	
70%–99%	8/382 (2.1)		17/382 (4.5)	
Lesion length				
<15 mm	4/175 (2.3)	1	12/175 (6.9)	.0754
≥15 mm	5/228 (2.2)		7/228 (3.1)	
<20 mm	6/230 (2.6)	.7380	14/230 (6.1)	.1588
≥20 mm	3/173 (1.7)		5/173 (2.9)	
Target site calcification		.046		.2496
Heavy	3/48 (6.3)		4/48 (8.3)	
None to mild	4/332 (1.2)		13/332 (3.9)	
Aortic arch type		1		.4569
Type I	4/179 (2.2)		6/179 (3.4)	
Type II	3/170 (1.8)		10/170 (5.9)	
Type III	1/32 (3.1)		1/32 (3.1)	
Unknown	1/28 (3.6)		2/28 (7.1)	

CAS, Carotid artery stenting; *CEA*, carotid endarterectomy; *ICA*, internal carotid artery.

[a]Includes all common carotid artery, carotid bifurcation, and ICA lesions.

Table IV

Technical predictors of perioperative stroke and major adverse events (*MAEs*)

Predictor	Stroke, No. (%)	*P* value	MAE, No. (%)	*P* value
Type of EPD		.745		.0955[a]
Accunet[b]	3/158 (1.9)[c]		3/158 (1.9)	
Emboshield/NAV6[b]	4/198 (2)		13/198 (6.6)	
Others (including Emboshield)	5/240 (2)		16/240 (6.7)	.0291[d]
Type of stent		.7449		.3827
ACCULINK[b]	4/209 (1.9)		7/209 (3.4)	
Xact[b]	4/155 (2.6)		10/155 (6.5)	
Others	1/42 (2.4)		2/42 (4.8)	
Stents, No.		1		1
1	9/383 (2.4)		18/383 (4.7)	
>1	0/24		1/24 (4.2)	
Stent length		.426		.7871
≤30 mm	3/119 (2.5)[c]		4/119 (3.4)[c]	
>30 mm	4/284 (1.4)		13/284 (4.6)	
PTA before EPD				
Yes	2/22 (9.1)	.0791	2/22 (9.1)	.2721
No	7/387 (1.8)		17/387 (4.4)	
PTA before stenting				
Yes	6/244 (2.5)	.7451	12/244 (4.9)	.7501
No	3/165 (1.8)		7/165 (4.2)	
PTA after stenting				
Yes	9/341 (2.6)	.3666	19/341 (5.6)	.0536
No	0/68		0/68	

EPD, Embolic protection device; *PTA*, percutaneous transluminal angioplasty.

[a] Accunet vs Emboshield vs others.

[b] Abbott, Abbott Park, Ill.

[c] Two patients with stroke had missing stent length and one patient had missing type of EPD.

[d] Accunet vs others.

Table V

Percutaneous transluminal balloon angioplasty (*PTA*) combinations and perioperative stroke and major adverse events (*MAEs*)

PTA	Stroke, No. (%)	MAE, No. (%)
None	0/26	0/26
Pre-EPD only	0/1	0/1
Prestent only	0/39	0/39
Poststent only	2/130 (1.5)	6/130 (4.6)
Pre-EPD and prestent	0/2	0/2
Pre-EPD and poststent	1/8 (12.5)	1/8 (12.5)
Prestent and poststent	5/192 (2.6)	11/192 (5.7)
Pre-EPD, prestent, and poststent	1/11 (9.1)	1/11 (9.1)

EPD, Embolic protection device.

Table VI

Logistic regression analysis

	Univariate: Early stroke		
Variable	**OR**	**95% CI**	***P* value**
Age	1.03	(0.96–1.11)	.4058
Gender (male vs female)	0.86	(0.23–3.24)	.8223
Hypertension	1.04	(0.13–8.52)	.9683
Coronary artery disease	1.33	(0.27–6.51)	.7215
Congestive heart failure	1.63	(0.4–6.61)	.4977
Hypercholesterolemia	1.10	(0.23–5.4)	.9026
Renal failure	1.00	(0.21–4.91)	.9971
Indication			
Asymptomatic	0.10	(0.01–0.81)	.031
TIA	13.69	(1.7–110.47)	.014
Stroke	1.10	(0.14–9.02)	.9273
Preprocedure stenosis (70%–99% vs 50%–69%)	0.56	(0.07–4.6)	0.5852
Lesion length (≥15 mm)	0.96	(0.25–3.62)	.9501
Pre-PTA performed before EPD	6.09	(1.19–31.2)	.0303
Pre-PTA performed before stent	1.73	(0.43–7.01)	.4413
Target site calcification-heavy	5.4	(1.16–24.66)	.0315
	Univariate: Early MI/stroke/death		
	OR	**95% UCL**	***P* value**
Age	1.02	(0.97, 1.06)	.5127
Gender (male vs female)	1.00	(0.42, 2.38)	.9913
Hypertension	0.42	(0.15, 1.19)	.101
Coronary artery disease	1.01	(0.39, 2.65)	.981
Congestive heart failure	0.94	(0.34, 2.62)	.9102
Hypercholesterolemia	0.66	(0.26, 1.66)	.3781
Renal failure	1.34	(0.51, 3.51)	.5558
Indication			
Asymptomatic	0.46	(0.19, 1.12)	.0858
TIA	2.04	(0.86, 4.82)	.1055
Stroke	0.88	(0.2, 3.87)	.8603
Preprocedure stenosis (70%–99% vs 50%–69%)	0.58	(0.13, 2.66)	.4833
Lesion length (≥15 mm)	0.43	(0.17, 1.12)	.0831
EPD (ACCUNET[a] vs other)	0.27	(0.08, 0.95)	.0413
Pre-PTA performed before EPD	2.07	(0.45, 9.45)	.35
Pre-PTA performed before stent	1.03	(0.44, 2.43)	.9463
Poststent PTA performed	2.16	(0.63, 7.43)	.2232

Target site calcification	2.18	(0.68, 6.98)	.1888
	Multivariate: Early stroke		
	OR	**95% CI**	***P* value**
TIA indication	11.05	(1.28–95.47)	.029
Pre-PTA performed before EPD	6.15	(0.91–41.44)	.062
Target site calcification: heavy	4.25	(0.81–22.32)	.0871
	Multivariate: Early MI/stroke/death		
	OR	**95% LCL**	***P* value**
EPD: ACCUNET vs other	0.27	(0.08, 0.94)	.0389

CI, Confidence interval; *EPD*, embolic protection device; *LCL*, lower confidence limit; *MI*, myocardial infarction; *PTA*, percutaneous transluminal balloon angioplasty; *TIA*, transient ischemic attack; *UCL*, upper confidence limit.

[a]Abbott, Abbott Park, Ill.