의학석사 학위논문

증상성 경동맥 협착의 스텐트 시술 후 원위부 혈관 직경과 무증상 뇌경색 발생과의 관련성

Distal normal vessel diameter might be associated with silent brain infarcts after stenting for symptomatic carotid stenosis

울산대학교 대학원

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류 재 찬

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지도교수 장준영

이 논문을 의학석사 학위 논문으로 제출함

2023 년 07 월

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ABSTRACT

Background: Carotid artery stenting (CAS) has been the standard treatment for carotid stenosis because it is less invasive; however, the risk of periprocedural thromboembolism is high. We investigated the predictors for silent brain infarcts (SBIs), focusing on embolic protection in CAS.

Methods: We obtained baseline demographics and clinical, laboratory, and periprocedural variables of patients who underwent CAS. Distal normal vessel diameter was defined as the diameter of cervical internal carotid artery where the artery wall becomes parallel. Diffusion-weighted imaging was performed before and after procedure to detect SBIs. The primary outcome was stented territory SBIs, and the secondary outcomes were any territories SBIs and stented territory SBIs in cases with EPD.

Results: A total of 209 CAS procedures in 194 patients, with mean age 69.3 ± 10.0 years, were included. After CAS, stented territory SBIs occurred in 53 (25.4%) cases and any territories SBIs in 60 (28.7%) cases. Univariable analyses revealed that distal normal vessel diameter (odds ratio=1.59, 95% confidence interval=1.13–2.24, *P*=0.007) was associated with the occurrence of stented territory SBIs after CAS. After adjusting for potential variables, larger distal normal vessel diameter (1.54 [1.05–2.24], *P*=0.026) increased the occurrence of SBIs after CAS. Consistent results were obtained when the outcome was any territories SBIs or stented territory SBIs in cases with EPD.

Conclusions: Distal normal vessel diameter was a predictor for the occurrence of SBI after CAS. The passable pore size of EPDs may vary depending on vessel diameter, and may impact the occurrence of SBIs.

Keywords: carotid artery stenting, vessel diameter, embolic protection device, silent brain infarct

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INTRODUCTION

Extracranial carotid artery stenosis is a common atherosclerotic disease that predisposes to ischemic stroke.[1,2] Carotid endarterectomy (CEA) has been the most commonly performed surgical intervention for preventing stroke in carotid artery stenosis.[3,4] Carotid artery stenting (CAS) is used as an alternative to CEA because it is less invasive and could be used in patients with severe comorbidities, such as coronary artery disease. However, several clinical trials have showed the presence of a higher risk of post-procedural complications, including ipsilateral stroke, after CAS compared with CEA.[5-7]

Silent brain infarcts (SBIs) are one of the post-procedural thromboembolic complications after CAS. SBIs are defined as small, radiologically detected dot-like lesions in diffusion-weighted imaging (DWI), without symptoms.[8] Although these new ischemic lesions are asymptomatic, studies have suggested that SBIs could increase the risk of dementia, cognitive decline, and future cerebrovascular incidents.[9-11] Despite the development and wide use of embolic protection devices in CAS, 29%–51% of patients may develop SBIs after CAS.[12-14]

Several studies have reported the predictors for SBI after CAS; however, the results have been inconsistent, and most reports have focused on laboratory markers; nature of plaques; and stent characteristics, such as type, length, and diameter.[12-16] Although intraprocedural embolic protection using embolic protection devices (EPDs)is the most powerful method for preventing thromboembolism in CAS,[17] the association between EPD and vessel size have rarely been investigated. Therefore, we aimed to identify predictors of SBI after CAS, focusing on the relationship between vessel size and EPD in CAS.

METHODS

Study participants

We retrospectively reviewed patients who had CAS for symptomatic proximal carotid artery stenosis in our tertiary hospital between January 2017 and November 2022. We included patients who fulfilled the following inclusion criteria: 1) age ≥18 years; 2) symptomatic proximal carotid artery stenosis >50%. Symptomatic carotid artery stenosis was defined as focal neurological symptoms including ischemic stroke or transient ischemic attack within 6 months.[18] Exclusion criteria were as follows: 1) patients who received emergent procedures within 24 h from symptom onset; 2) patients who failed recanalization due to failure of guidewire passage; 3) patients who did not undergo pre- and post-stenting DWI. The ethics committee of our tertiary hospital approved this study (approval No. 2023-0565) and waived the need for informed consent due to the retrospective nature of this study.

Clinical, laboratory, and procedural data collection

We obtained data on baseline demographics; vascular risk factors; laboratory findings, including antiplatelet resistance assay, angiographic findings (stenosis degree before CAS, distal and proximal normal vessel diameter, stenotic vessel diameter, presence of contralateral severe carotid stenosis, and residual stenosis after CAS); and procedure-related factors (type and size of devices, methods of balloon angioplasty, and embolic protection). We administered aspirin and clopidogrel to patients at least 5 d before the procedure. We used the VerifyNow Aspirin and P2Y12 assays (Accumetrics, San Diego, CA, U.S.A.) to measure the extent of platelet inhibition using aspirin reaction unit (ARU), P2Y12 reaction unit (PRU), and percentage platelet inhibition (%PI). ARU > 550, PRU > 235, and %PI < 20% were defined as resistance to antiplatelet treatment.[19,20]

CAS was performed by three neurointerventionists with an average of 15 years of experience in endovascular treatment. According to the North American Symptomatic Endarterectomy Trial (NASCET) criteria, distal normal vessel diameter, proximal normal

vessel diameter, and most stenotic diameter were measured.[4,18] Stenosis degree before CAS was categorized into moderate (50%–70%) and severe (>70%) stenosis. The use of guiding catheters, microwires, ballooning angioplasty, embolic protection devices, and stents was at the discretion of the three neurointerventionists. The methods of balloon angioplasty (no balloon angioplasty, pre-stent balloon angioplasty, post-stent angioplasty, or pre- and post-stent angioplasty), maximum balloon diameter, balloon length, and total procedure time were collected. Moreover, methods used for embolic protection (no protection, distal EPD, or proximal balloon guiding catheter) during CAS were obtained. CAS was performed using Acculink (Abbott Laboratories), Precise (Cordis), or Protégé (Covidien), and the diameters and total lengths of stents were obtained. After CAS procedure, residual stenosis was defined as >30% stenosis remaining compared with distal normal vessel diameter.[13,21]

Outcomes and neuroimaging analyses for SBIs

Magnetic resonance imaging (MRI), including diffusion-weighted imaging (DWI), was performed within 7 d before CAS procedure, and within 48 h after CAS procedure. By comparing pre- and post-stenting DWIs, we evaluated the occurrence and location of SBIs. The occurrence of SBIs was defined as a newly detected dot-like lesion in post-stenting DWI after CAS compared with pre-stenting DWI. The primary outcome was the occurrence of stented territory SBIs, which means newly detected dot-like lesions in the stented vascular territory. Regarding secondary outcomes, the occurrence of any territories SBIs and stented territory SBIs in cases with EPD were investigated. All MRIs were analyzed by two stroke neurologists (JCR and JYC) and one neuroradiologist separately, blinded to clinical data.

Statistical analysis

We first analyzed the baseline demographics, vascular risk factors, and procedure-related factors of study participants. Continuous variables are presented as mean ± standard deviation, whereas categorical variables are presented as frequency and percentage. We used univariable logistic regression analyses to identify factors that are potentially associated with stented territory SBIs. Then, variables with potential association ($P < 0.10$) in univariable logistic regression analyses and baseline demographics (age and sex) were included in multivariable logistic regression analyses. For sensitivity analyses, univariable and multivariable logistic regression test were performed for any territories SBIs, and for stented territory SBIs only in cases with EPDs. *P*-value <0.05 was considered statistically significant. Odds ratios (ORs) and their 95% confidence intervals (CIs) were calculated. All statistical analyses were performed using R Software (version 4.2.3; R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

During the study period, 249 patients underwent CAS for symptomatic carotid artery stenosis (Figure 1). We excluded 27 (10.8%) patients who received emergent procedures, 8 (3.2%) who failed CAS for recanalization, and 20 (8.0%) who did not perform pre- or post-stenting DWI. Finally, 209 CAS procedures in 194 patients were included in the study (15 patients underwent bilateral stent insertions for bilateral symptomatic carotid artery stenoses). A comparison of baseline characteristics between included and excluded cases revealed no significant differences between the two groups (Table 1).

Baseline characteristics of cases included

The clinical and procedure-related factors of this study are summarized in Table 2. The mean age of patients in 209 cases was 69.3 ± 10.0 years old, and 172 (82.3%) were male. In total,

145 (69.4%) cases were CAS for severe degree, and 71 (34.0%) cases had contralateral carotid stenosis >70%. The types of stents used in the procedure were: Precise® (77.5%), Acculink® (11.5%), and Protégé® (11.0%).

In this study, 53 (25.4%) cases had stented territory SBIs after CAS, and 60 (28.7%) had any territories SBIs. ARUs were tested in 204 cases, and 39 (19.1%) cases had resistance to aspirin. PRU and %PI were tested in 201 cases. In total, 50 cases in (24.9%) in PRU and 100 (49.8%) in %PI had clopidogrel resistance.

Predictors for stented territory SBIs after CAS

Univariable logistic regression analysis showed that stented territory SBIs after CAS were potentially associated with age, hyperlipidemia, coronary artery disease, distal normal vessel diameter, and aspirin resistance (Table 3). In multivariable analysis adjusted for age, sex, and variables with potential associations, distal normal vessel diameter was significantly associated with the occurrence of stented territory SBIs after CAS (OR=1.54, 95% CIs=1.05–2.24, *P*=0.026).

For sensitivity analyses, we analyzed any territories or stented territory SBIs in cases with EPDs as secondary outcomes. When the outcome was any territories SBIs, distal normal vessel diameter (1.53 [1.07–2.18], *P*=0.021) was significantly associated with any territories SBIs in CAS after adjusting for other potential variables (Table 4). These relationships were maintained when the outcome was stented territory SBIs after CAS only with the use of EPDs (Table 5).

DISCUSSION

The findings of this study showed that approximately one-quarter of patients developed stented territory SBIs after CAS despite the use of EPDs. As distal normal vessel diameter

increased in CAS, the occurrence of stented territory SBI increased. These relationships were maintained even when the outcomes were any territories SBIs and stented territory SBIs in cases with EPD. Larger distal normal vessel diameter, aspirin resistance, hyperlipidemia, and coronary artery disease increased the occurrence of SBIs in the stented territory. These findings were maintained even when the outcomes were any territories SBIs and stented territory SBIs in cases with EPD.

Our study focused on the relationship between distal normal vessel diameter and stented territory SBIs in patients with CAS. Studies have demonstrated that age, diabetes, specific biomarkers, cerebrovascular reserves, nature of plaque, and stent length are associated with the occurrence of SBI risk;[12,14,16,22] however, reports on the relationship between distal normal vessel diameter and stented territory SBI in CAS are lacking. Two possible mechanisms can be proposed for the association between distal normal vessel size and SBIs. First, the embolus can bypass the EPD when the EPD does not fully appose the wall (Figure 1A). The malapposition between EPD and distal normal vessel can lead to the embolus bypassing the EPD.[23] Second, the pore size of EPDs ranges from 70 to 200 μm, varying depending on EPD type.[24] Typically, pore size remains consistent within a single type of EPD. However, passable pore size can vary depending on how much EPD is expanded according to vessel size (Figure 1B). If the EPD is deployed in small-diameter vessels, the angle between the side of distal filter and the cross-section of the vessel is likely to be large. In this scenario, considering blood flow and direction of emboli, passable pore size would be smaller. Therefore, the effect of EPD in preventing thromboembolism would be better. However, in the case of deploying an EPD in a patient with large-diameter vessels, the angle between the side of the EPD and the cross-section of the vessel would be small, resulting in a relatively larger passable pore size. Consequently, the efficacy for preventing thromboembolism in CAS might be somewhat decreased, and SBIs are more likely to occur.

In this study, other factors, including aspirin resistance, hyperlipidemia, and coronary artery disease, were also associated with the occurrence of stented territory SBIs. Studies have shown that insufficient platelet inhibition increased the occurrence of SBIs in coronary angiography,[25] and aspirin resistance increased the development of SBIs on follow-up DWI by approximately twofold.[26] Moreover, proximal carotid atherosclerosis shares many similarities with coronary artery disease, and hyperlipidemia is an important risk factor for both diseases.[27,28] Coronary artery atherosclerosis is an important risk factor for SBI, and the presence of coronary artery disease was shown to be associated with the occurrence of SBI in CAS.[29,30] Therefore, hyperlipidemia and coronary artery disease might increase the occurrence of SBI in CAS for symptomatic carotid stenosis.

There are some limitations to our study. First, the nature or characteristics of plaque were not investigated. Calcification or ulceration in plaque might be associated with the occurrence of SBIs. Second, SBIs can occur during guidewire passage before the deployment of EPD. Continuous monitoring for microembolic signals by transcranial Doppler ultrasound might help in determining at which stage of the procedure SBIs occurred. Third, this study was performed retrospectively in a single center, and the number of cases was modest.

Despite these limitations, we have shown that distal normal vessel diameter was associated with the occurrence of SBIs in CAS after adjusting for other factors, such as aspirin resistance, hyperlipidemia, and coronary artery disease. Approximately 25% of patients had SBIs despite the use of an EPD. Therefore, considering vessel size before CAS may allow for predicting the occurrence of SBI after CAS. In real practice, considering the use of a slightly larger EPD to prevent SBIs might be considered during CAS, which requires further investigation with a larger prospective study.

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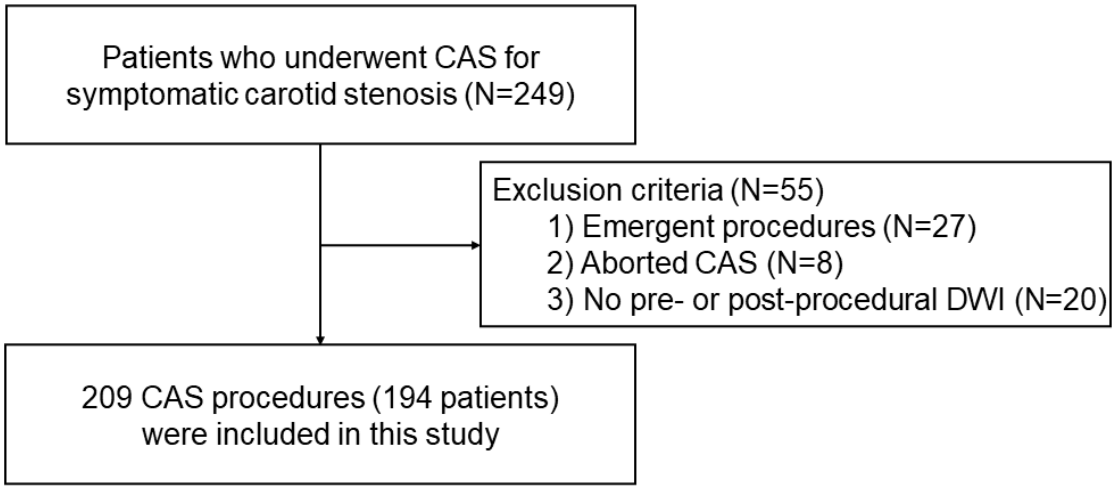
FIGURES

Figure 1. Flow diagram of the patients included in this study.



CAS, carotid artery stenting; DWI, diffusion-weighted imaging

Figure 2. Schematic illustration of passable pore size according to vessel diameter. A) The malapposition between EPD and distal normal vessel can lead to embolic materials bypassing the side of distal filter during CAS. B) If the EPD is deployed in small distal vessels, the angle (θ_1) between the side of distal filter and vessel is likely to be large. In this case, considering blood flow and direction of emboli, passable pore size would be smaller (D × cos75° = 0.26D). However, when EPD is deployed in large distal vessels, the angle (θ_2) between the side of distal filter and vessel is likely to be small. Therefore, passable pore size would be larger (D × cos60° = 0.5D), and EPD would be less effective in preventing thromboembolism.

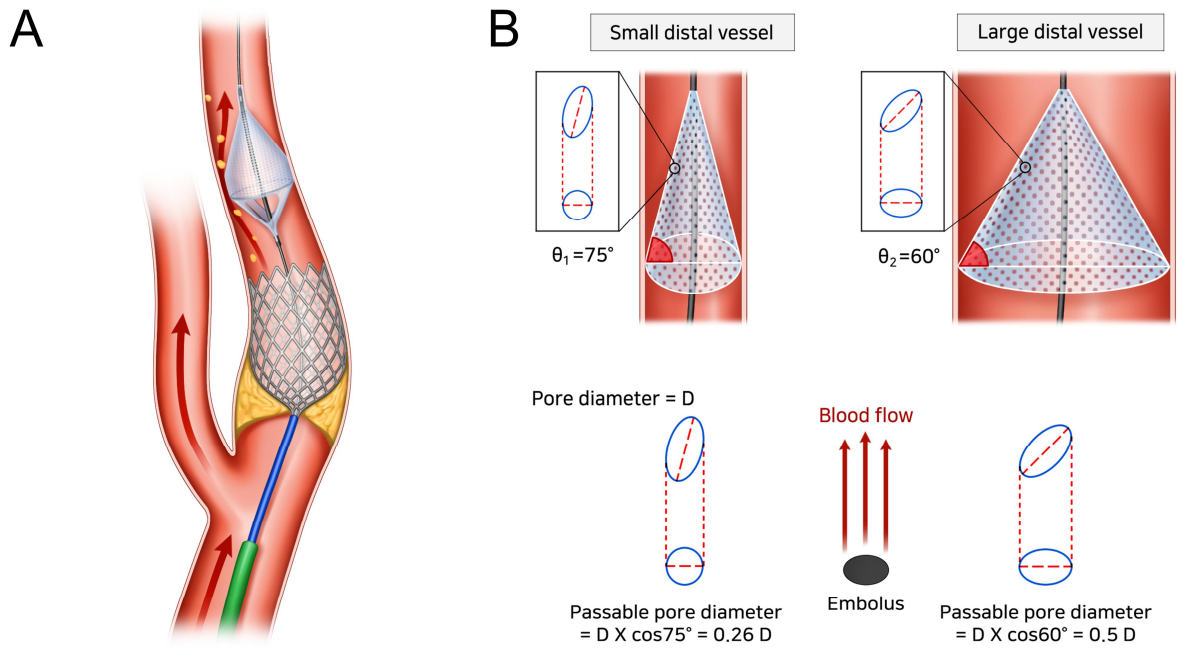


Table 1. Comparison of baseline characteristics between included and excluded cases.

Variables	Included (N = 209)	Excluded (N = 55)	*P*-value
Age, years	69.3 ± 10.0	68.4 ± 10.0	0.567
Male sex	172 (82.3)	42 (76.4)	0.420
Hypertension	152 (72.7)	33 (60.0)	0.095
Diabetes	86 (41.1)	23 (41.8)	>0.999
Hyperlipidemia	117 (56.0)	24 (43.6)	0.139
Coronary artery disease	66 (31.6)	13 (23.6)	0.328
Atrial fibrillation	13 (6.2)	3 (5.5)	>0.999
Smoking	92 (44.0)	27 (49.1)	0.603
Contralateral severe stenosis (>70%)	71 (34.0)	13 (23.6)	0.193

Table 2. Baseline characteristics, clinical variables, and procedure-related factors of CAS procedure in symptomatic carotid artery stenosis

Variables	Total vessels treated with CAS (*N* = 209)
Age, years	69.3 ± 10.0
Male sex	172 (82.3)
Hypertension	152 (72.7)
Diabetes	86 (41.1)
Hyperlipidemia	117 (56.0)
Coronary artery disease	66 (31.6)
Atrial fibrillation	13 (6.2)
Smoking	92 (44.0)
Contralateral severe stenosis (>70%)	71 (34.0)
Distal normal vessel diameter, mm	4.5 ± 1.0
Proximal normal vessel diameter, mm	7.5 ± 4.6
Stenotic vessel diameter, mm	1.2 ± 0.6
Stenosis degree before CAS*	74.6 ± 11.2
Moderate stenosis (50%–70%)	64 (30.6)
Severe stenosis (>70%)	145 (69.4)
Stent diameter, mm	7.2 ± 0.8
Stent length, mm	36.0 ± 6.2
Stent type	
Acculink®	24 (11.5)
Precise®	162 (77.5)
Protégé®	23 (11.0)
Balloon angioplasty	
No	14 (6.7)
Pre-stenting	48 (23.0)
Post-stenting	29 (13.9)
Pre- and post-stenting	118 (56.5)
Maximum balloon diameter, mm (*N* = 195)	4.4 ± 0.5
Balloon length, mm (*N* = 195)	31.2 ± 5.1
Embolic protection	
No	26 (12.4)
Distal embolic protection device	168 (80.4)
Proximal balloon guiding catheter	15 (7.2)
Procedure time, min	43.1 ± 14.4
Residual stenosis (≥30%) after CAS	18 (8.6)
Platelet inhibition	
ARU >550 (*N* = 204)	39 (19.1)
PRU >235 (*N* = 201)	50 (24.9)
%PI <20% (*N* = 201)	100 (49.8)

Outcomes	
Stented territory SBIs	53 (25.4)
Any territories SBIs	60 (28.7)

Values are expressed as number (%) and mean ± standard deviation.

*Stenosis degree before CAS was calculated by the North American Symptomatic Endarterectomy Trial (NASCET) criteria.

CAS, carotid artery stenting; ARU, aspirin reaction unit; PRU, P2Y12 reaction unit; %PI, percent platelet inhibition; SBI, silent brain infarct

Table 3. Univariable and multivariable logistic analyses for stented territory SBIs ($N = 53$) after CAS in symptomatic carotid artery stenosis

Variables	cOR (95% CIs)	*P*-value	aOR (95% CIs)	*P*-value
Age	1.03 (1.00–1.07)	0.089	1.03 (0.99–1.07)	0.143
Male sex	1.07 (0.47–2.44)	0.873	1.17 (0.46–2.99)	0.749
Hypertension	1.39 (0.67–2.88)	0.382		
Diabetes	1.13 (0.60–2.13)	0.700		
Hyperlipidemia	2.20 (1.13–4.27)	0.020	2.31 (1.11–4.82)	0.025
Coronary artery disease	2.79 (1.46–5.34)	0.002	3.09 (1.54–6.20)	0.002
Atrial fibrillation	1.33 (0.39–4.52)	0.644		
Smoking	0.64 (0.33–1.21)	0.167		
Contralateral stenosis (>70%)	0.79 (0.41–1.56)	0.501		
Distal normal vessel diameter, mm	1.59 (1.13–2.24)	0.007	1.54 (1.05–2.24)	0.026
Proximal normal vessel diameter, mm	1.13 (0.89–1.43)	0.314		
Stenotic vessel diameter, mm	1.49 (0.92–2.42)	0.107		
Stenosis degree before CAS				
Moderate stenosis	Reference			
Severe stenosis	0.91 (0.47–1.79)	0.791		
Stent diameter, mm	1.22 (0.82–1.82)	0.328		
Stent length, mm	0.96 (0.90–1.01)	0.127		
Stent type				
Acculink®	Reference			
Precise®	1.86 (0.60–5.76)	0.279		
Protégé®	1.39 (0.32–5.99)	0.659		
Balloon angioplasty				
Pre-stenting	Reference			
Post-stenting	2.06 (0.75–5.63)	0.161		
Pre- and post-stenting	1.20 (0.54–2.64)	0.652		
Maximum balloon diameter, mm ($N = 195$)	1.49 (0.82–2.71)	0.191		
Balloon length, mm ($N = 195$)	0.97 (0.91–1.03)	0.304		
Embolic protection				
No	Reference			
Distal EPD	0.96 (0.38–2.45)	0.937		
Proximal BGC	0.42 (0.07–2.34)	0.320		
Procedure time, min	1.00 (0.98–1.02)	0.841		
Residual stenosis (≥30%)	1.15 (0.39–3.38)	0.805		
Platelet inhibition				
ARU >550 ($N = 204$)	2.24 (1.06–4.71)	0.034	2.62 (1.16–5.89)	0.020
PRU >235 ($N = 201$)	1.16 (0.56–2.37)	0.692		

%PI <20% (*N* = 201)	1.54 (0.81–2.91)	0.185

SBI, silent brain infarct; CAS, carotid artery stenting; cOR, crude odds ratio; aOR, adjusted odds ratio; EPD, embolic protection device; BGC, balloon guiding catheter; ARU, aspirin reaction unit; PRU, P2Y12 reaction unit; %PI, percent platelet inhibition

Table 4. Univariable and multivariable logistic analyses for any territories SBIs ($N = 60$) after CAS in symptomatic carotid artery stenosis

Variables	cOR (95% CIs)	*P*-value	aOR (95% CIs)	*P*-value
Age	1.02 (0.99–1.05)	0.260	1.01 (0.98–1.05)	0.492
Male sex	0.94 (0.43–2.05)	0.880	0.92 (0.38–2.23)	0.854
Hypertension	1.33 (0.66–2.67)	0.418		
Diabetes	0.94 (0.51–1.72)	0.831		
Hyperlipidemia	2.56 (1.34–4.90)	0.004	2.72 (1.34–5.52)	0.006
Coronary artery disease	2.83 (1.51–5.31)	0.001	3.05 (1.56–5.98)	0.001
Atrial fibrillation	1.11 (0.33–3.76)	0.865		
Smoking	0.65 (0.35–1.21)	0.176		
Contralateral stenosis (>70%)	0.70 (0.36–1.34)	0.276		
Distal normal vessel diameter, mm	1.58 (1.14–2.19)	0.006	1.53 (1.07–2.18)	0.021
Proximal normal vessel diameter, mm	1.09 (0.90–1.33)	0.366		
Stenotic vessel diameter, mm	1.22 (0.76–1.95)	0.413		
Stenosis degree before CAS, %				
Moderate stenosis	Reference			
Severe stenosis	1.17 (0.60–2.25)	0.649		
Stent diameter, mm	1.20 (0.82–1.77)	0.351		
Stent length, mm	0.97 (0.92–1.02)	0.204		
Stent type				
Acculink®	Reference			
Precise®	1.65 (0.58–4.66)	0.347		
Protégé®	1.34 (0.35–2.79)	0.671		
Balloon angioplasty				
Pre-stenting	Reference			
Post-stenting	2.12 (0.79–5.68)	0.136		
Pre- and post-stenting	1.27 (0.59–2.71)	0.546		
Maximum balloon diameter, mm ($N = 195$)	1.02 (0.57–1.83)	0.941		
Balloon length, mm ($N = 195$)	0.95 (0.90–1.01)	0.131		
Embolic protection				
No	Reference			
Distal EPD	0.95 (0.39–2.34)	0.917		
Proximal BGC	0.35 (0.06–1.91)	0.223		
Procedure time, min	1.00 (0.98–1.02)	0.919		
Residual stenosis (≥30%)	0.95 (0.32–2.79)	0.927		
Platelet inhibition				
ARU >550 ($N = 204$)	2.04 (0.98–4.22)	0.055	2.35 (1.06–5.22)	0.035
PRU >235 ($N = 201$)	0.95 (0.46–1.92)	0.878		

%PI <20% (*N* = 201)	1.23 (0.67–2.27)	0.505

SBI, silent brain infarct; CAS, carotid artery stenting; cOR, crude odds ratio; aOR, adjusted odds ratio; EPD, embolic protection device; BGC, balloon guiding catheter; ARU, aspirin reaction unit; PRU, P2Y12 reaction unit; %PI, percent platelet inhibition

Table 5. Univariable and multivariable logistic analyses for stented territory SBIs after CAS with EPDs (44/168 [26.2%] cases) in symptomatic carotid artery stenosis

Variables	cOR (95% CIs)	*P*-value	aOR (95% CIs)	*P*-value
Age	1.04 (0.99–1.08)	0.059	1.04 (0.99–1.09)	0.093
Male sex	1.02 (0.42–2.50)	0.957	1.24 (0.47–3.24)	0.661
Hypertension	1.53 (0.67–3.51)	0.316		
Diabetes	1.50 (0.75–2.99)	0.255		
Hyperlipidemia	2.64 (1.22–5.68)	0.013	2.36 (1.06–5.23)	0.035
Coronary artery disease	2.04 (1.00–4.14)	0.049	2.06 (0.97–4.35)	0.059
Atrial fibrillation	1.06 (0.27–4.19)	0.933		
Smoking	0.57 (0.27–1.17)	0.125		
Contralateral stenosis (>70%)	0.79 (0.37–1.67)	0.536		
Distal normal vessel diameter, mm	1.75 (1.16–2.63)	0.008	1.68 (1.09–2.59)	0.019
Proximal normal vessel diameter, mm	1.14 (0.88–1.48)	0.322		
Stenotic vessel diameter, mm	1.52 (0.87–2.66)	0.142		
Stenosis degree before CAS, %				
Moderate stenosis	Reference			
Severe stenosis	1.05 (0.49–2.27)	0.904		
Stent diameter, mm	1.05 (0.68–1.63)	0.822		
Stent length, mm	0.97 (0.91–1.03)	0.290		
Stent type				
Acculink®	Reference			
Precise®	1.77 (0.56–5.58)	0.329		
Protégé®	1.13 (0.21–5.95)	0.890		
Balloon angioplasty				
Pre-stenting	Reference			
Post-stenting	1.39 (0.40–4.82)	0.601		
Pre- and post-stenting	1.23 (0.50–3.03)	0.646		
Maximum balloon diameter, mm (N = 195)	1.38 (0.73–2.60)	0.327		
Balloon length, mm (N = 195)	1.00 (0.93–1.08)	0.923		
Procedure time, min	1.00 (0.98–1.02)	0.939		
Residual stenosis (≥30%)	1.67 (0.46–6.01)	0.432		
Platelet inhibition				
ARU >550 (N = 204)	1.48 (0.61–3.58)	0.386		
PRU >235 (N = 201)	1.13 (0.53–2.43)	0.755		
%PI <20% (N = 201)	1.37 (0.68–2.76)	0.386		

SBI, silent brain infarct; CAS, carotid artery stenting; EPD, embolic protection device; cOR, crude odds ratio; aOR, adjusted odds ratio; ARU, aspirin reaction unit; PRU, P2Y12 reaction unit; %PI, percent platelet inhibition

국문요약

연구 배경: 경동맥 스텐트는 경동맥 협착에 대한 표준적인 치료로서 널리 행해지고 있으며, 덜 침습적인 장점이 있지만, 시술 전후 혈전색전증의 위험이 높은 것으로 알려져 있다. 본 연구에서는 경동맥 스텐트 시술 후 발생할 수 있는 무증상 뇌경색의 예측인자에 대하여 분석하였으며, 특히 경동맥 스텐트에서의 색전 예방에 초점을 맞추어 연구하였다.

연구 방법: 우리는 경동맥 스텐트를 환자의 기본 정보, 임상적, 그리고 시술과 관련된 인자들을 수집하였다. 원위부 정상 혈관 직경은 경부분절 내경동맥이 서로 평행하게 되는 부분의 직경으로 정의하였다. 시술 후 무증상 뇌경색의 발생을 확인하기 위해 시술 전 후 확산강조영상을 시행하였다. 일차결과지표는 스텐트 시행 영역의 무증상 뇌경색, 그리고 이차결과지표는 모든 영역의 무증상 뇌경색과 원위부 색전 방지 기구를 사용한 환자들에서의 스텐트 시행 영역의 무증상 뇌경색 이었다.

결과: 총 194명의 환자에서 209건의 경동맥 스텐트가 시행되었으며, 평균 나이는 69.3±10.0세 였다. 경동맥 스텐트 이후 53 (25.4%) 건에서 스텐트 시행 영역에 무증상 뇌경색이 발생하였고, 60 (28.7%) 건에서 모든 영역의 무증상 뇌경색이 발생하였다. 단변량 로지스틱 회귀분석에서 원위부 정상 혈관 직경 (OR=1.59, 95% CI=1.13–2.24, P=0.007)은 스텐트 시행 영역의 무증상 뇌경색과 관련이 있었으며, 여러가지 잠재적 혼란 변수를 보정한 다변량 로지스틱 회귀분석에서도 원위부 정상 혈관 직경 (OR=1.54, 95% CI=1.05–2.24, P=0.026)의 증가는 스탠트 시행 영역의 무증상 뇌경색을 증가시켰다. 이러한 결과는 결과가 이차결과지표일 때에도 그 경향이 유지되었다.

결론: 경동맥 스텐트에서 원위부 정상 혈관 직경은 무증상 뇌경색의 발생과 연관이 있

었다. 색전 방지 기구의 통과 가능한 구멍의 크기는 원위부 정상 혈관 직경에 따라 변하며, 이는 무증상 뇌경색의 발생과 연관이 있었다.

중심단어: 경동맥 스텐트, 혈관 직경, 색전방지기구, 무증상 뇌경색