Strategy of carotid artery stenting as first-line treatment and carotid endarterectomy for carotid artery stenosis: A single-center experience

Takanari Okamoto¹, Yasuo Inoue², Yuta Oi¹, Ichita Taniyama¹, Takashi Houri², Satoshi Teramukai³, Naoya Hashimoto¹

¹Department of Neurosurgery, Kyoto Prefectural University of Medicine Graduate School of Medical Science, Kyoto; ²Department of Neurosurgery, National Hospital Organization Maizuru Medical Center, Maizuru; ³Department of Biostatistics, Kyoto Prefectural University of Medicine Graduate School of Medical Science, Kyoto, Japan.

INTRODUCTION

Carotid endarterectomy (CEA) and carotid artery stenting (CAS) are well-established treatments for symptomatic or asymptomatic stenosis of the carotid artery.⁶ Since some randomized controlled trials have failed to show that CAS is not inferior to CEA, Japanese guidelines for stenosis of the carotid artery suggest CEA as the first choice.⁶ However, despite this...
recommendation, CAS procedures far outnumber CEA procedures in Japan due to advantages of convenience, reduced invasiveness, and availability of devices to prevent embolic stroke.\[15,16\]

There are opposing opinions on CEA and CAS, but, in recent years, this discussion has focused on selection of appropriate treatment based on the characteristics of both procedures. The unique feature of surgical treatment for carotid artery stenosis in Japan is that both CAS and CEA can be performed by the same team or the same neurosurgeon. Therefore, treatment options tend to be selected more evenly than in other countries and the outcomes of both treatments are likely to be good. In this study, we analyzed data from a single center to examine the strategy of CAS as first-line treatment and CEA for carotid artery stenosis.

**MATERIALS AND METHODS**

**Study design and patients**

Patients with carotid artery stenosis who underwent CAS or CEA at our hospital between January 2012 and May 2020 were reviewed retrospectively in a single-center study. Surgical treatment was performed for patients with (1) angiographically symptomatic stenosis of at least 50% of the luminal diameter, (2) asymptomatic stenosis of at least 70%, and (3) symptomatic stenosis with intraluminal ulceration. Patients who were unable to complete the surgical treatment and those with occlusion or dissection were excluded from the study. Demographic information (age, sex, and race), symptomatic stenosis, stroke risk factors (smoking status, hypertension, diabetes mellitus, hyperlipidemia, history of stroke, and history of heart disease), renal function (estimated glomerular filtration rate), plaque parameters (degree of stenosis and lesion length), pre- and postoperative modified Rankin Score (mRS), and postoperative complications were collected for each patient. Carotid stenosis was deemed symptomatic if neurologic symptoms attributable to the ipsilateral hemisphere were present within a year before presentation.

**Preoperative evaluation and patient selection**

For patients with carotid artery stenosis diagnosed by ultrasonography (US), either CTA or diagnostic angiography was performed before intervention to identify anatomical factors that could affect the treatment decision. Preoperative US, MRI, and CTA were used to evaluate internal carotid artery (ICA) plaques. In addition to carotid imaging, blood pressure monitoring, heart rhythm monitoring, electrocardiogram, and echocardiography were performed. If there were any abnormalities in cardiac function, the patient was examined by a cardiologist and prioritized for cardiac treatment. At our hospital, CAS is the first choice for patients with carotid artery stenosis. CEA was chosen in cases with vulnerable plaques; a relatively low risk for general anesthesia, such as cardiopulmonary insufficiency; and no anatomical features disadvantageous for endarterectomy, such as high or low cervical lesions, previous radiotherapy, and contralateral internal carotid stenosis [Figure 1].

Treatments were discussed among the surgical team. The surgery was performed mainly by two neurosurgeons with expertise in interventional radiology and open surgery. The study was conducted to evaluate whether patients were correctly assigned to CEA using a risk index for myocardial infarction, stroke, or death. The risk index predictors are age, living in a nursing home, cardiovascular disease, congestive heart failure, chronic obstructive pulmonary disease, diabetes mellitus, and degree of contralateral stenosis.\[5\]

**Definitions of preoperative variables**

Plaque was judged to be vulnerable in a case with a signal intensity ratio (SIR) of carotid plaque against sternocleidomastoid muscle on black-blood fat-suppressed (FS) T1-weighted magnetic resonance imaging (T1-MRI) >1.5 or a high-intensity signal on time-of-flight-MR angiography. In addition, if preoperative US of the ICA plaque showed a mobile component that was not synchronized with the heartbeat, the plaque was also judged to be vulnerable.\[17,21\] Severely calcified plaque was defined as four-quadrant calcification (cross-sectional calcium ≥270°) on CTA. High cervical stenosis was defined as that higher than the second cervical vertebra or mastoid mandibular

| Significant carotid artery stenosis with indication for surgery |
|---|
| Plaque vulnerability |
| No |
| Yes |
| Risk of general anesthesia |
| High |
| Low |
| Unfavorable anatomies for CEA |
| Yes |
| No |
| CAS |
| CEA |

**Figure 1:** Algorithm for surgical management of carotid artery stenosis. CEA: carotid endarterectomy, CAS: carotid artery stenting.
line.\textsuperscript{[11]} Unfavorable anatomies for CEA were considered to be (1) carotid lesions located at or above the level of the second cervical vertebra, (2) lesions below the clavicle, (3) prior radical neck surgery or radiation, (4) contralateral carotid occlusion, (5) preoperative CEA for recurrent stenosis, (6) contralateral laryngeal nerve palsy, and (7) tracheostomy in accordance with the USA Guideline on the Management of Patients with Extracranial Carotid and Vertebral Artery Disease.\textsuperscript{[1]}

**Surgical procedures**

CAS was performed under local anesthesia. Patients who underwent CAS received perioperative dual antiplatelet therapy (usually aspirin and clopidogrel) and intraoperative systemic heparinization. An embolic protection device was utilized in all cases, and dual protection (proximal balloon protection of the common carotid artery and the external carotid artery, and distal protection) was used whenever possible. Distal protection was mainly performed with filter devices. An open- or closed-cell stent was selected depending on plaque vulnerability and flexion of the lesion. For example, we primarily chose a closed-cell stent for linear lesions with vulnerable plaque. Systemic heparinization was continued for 5 min after the final angioplasty and not reversed.

CEA was performed under general anesthesia. Preoperative single antiplatelet therapy (usually aspirin) was continued, and systemic heparin was administered before blood flow was blocked. Patients underwent monitoring with continuous electroencephalography and near-infrared spectroscopy. Three-way internal shunts were placed in almost all cases. No patch angioplasty was performed.

**Postoperative evaluation**

Complications associated with CEA or CAS and those common to both, such as hyperperfusion, were assessed. Postoperative stroke or asymptomatic hyperintensities on diffusion-weighted imaging (DWI) were examined within 48 h after surgery. Major adverse events were defined as major stroke, myocardial infarction, and death after surgical treatment. Stroke was defined as a rapidly developing syndrome of focal disturbance of cerebral function that lasted more than 24 h. Minor and major strokes were defined as new neurological deficits that completely resolved within 30 days or lasted for more than 30 days of follow-up, respectively. The mRS score for disability was used to evaluate the preoperative and 1-year postoperative neurological conditions of the patients.

**Statistical analysis**

For patient characteristics, continuous and ordinal variables are summarized as median and range and compared by Mann–Whitney U-test, and categorical variables are summarized as frequencies (percentages) and compared by Fisher’s exact test. Relationships between the occurrence of postoperative ischemic lesions, including symptomatic and asymptomatic lesions positive on DWI, and patient characteristics were examined by multivariate logistic regression analysis according to the surgical procedures (CAS and CEA). \( P < 0.05 \) was considered to be statistically significant. All statistical analyses were performed with R version 4.0.0 (The R Foundation for Statistical Computing).

**RESULTS**

**Patient background**

After a retrospective review of cases, three patients were excluded from the study due to pseudo-occlusion with the guidewire failing to pass through, complete occlusion with the guidewire failing to pass, and performance of CAS for an occlusion due to carotid artery dissection, respectively. Of the 140 cases enrolled in the study, 102 underwent CAS and 38 underwent CEA. All of the patients were Japanese. The characteristics of the 140 cases are shown in Table 1. CAS cases had a higher median age compared to CEA cases (75 vs. 70 years). Regarding risk factors, the rate of previous stroke or transient ischemic attack was higher in the CEA group (71.6% vs. 89.5%). Cardiac disease tended to be more common in CAS cases, and no CEA cases had cardiac failure. The risk index for CEA was higher in CAS cases (8 vs. 6 points). The CEA group had higher rates of unfavorable anatomies (2.6% vs. 37.3%) and vulnerable plaques (40.2% vs. 84.2%) and SIR (1.18 vs. 1.71). The only vulnerable plaques were three mobile plaques identified on US, and all were found in CEA cases. The CEA group also had only one case with a high cervical lesion.

**Postoperative clinical outcome**

Postoperative clinical outcomes after CAS or CEA are shown in Table 2. Major stroke occurred as a major adverse event within 30 days in 2 patients (2%) in the CAS group, giving an overall rate of 1.4%. Minor stroke occurred in 2 patients (2%) in the CAS group and in 1 patient (2.6%) in the CEA group, giving an overall rate of 2.1%. DWI showed asymptomatic hyperintensities in 29 (28.4%) CAS and 9 (23.7%) CEA cases, and hyperperfusion occurred in 4 (3.9%) and 1 (2.6%) cases, respectively. At 1 year postoperatively, mRS did not differ between the two groups, and follow-up angiography showed no restenosis requiring retreatment. Five CAS cases had puncture site hematoma and 26 had hypotension/bradycardia, while two CEA cases had postoperative wound hematomas and two had transient hoarseness (recurrent laryngeal nerve palsy). None of these adverse events resulted in permanent disability.
Table 1: Patient characteristics (n=140 cases).

| Item                          | CAS (n=102) | CEA (n=38) | P-value |
|-------------------------------|-------------|------------|---------|
| Age, years, median (range)    | 75 (49–87)  | 70 (60–88) | 0.011   |
| Male sex, n (%)               | 92 (90.2)   | 32 (84.2)  | 0.373   |
| Symptomatic stenosis, n (%)   | 51 (50.0)   | 25 (65.8)  | 0.127   |
| Risk factors                  |             |            |         |
| Hypertension, n (%)           | 83 (81.4)   | 29 (76.3)  | 0.487   |
| Diabetes mellitus, n (%)      | 43 (42.2)   | 11 (28.9)  | 0.176   |
| Dyslipidemia, n (%)           | 63 (61.8)   | 25 (65.8)  | 0.699   |
| Current smoker, n (%)         | 22 (21.6)   | 15 (39.5)  | 0.051   |
| Ex-smoker, n (%)              | 50 (49.0)   | 15 (39.5)  | 0.346   |
| Previous stroke/TIA, n (%)    | 73 (71.6)   | 34 (89.5)  | 0.027   |
| Cardiovascular disease, n (%) | 36 (35.3)   | 7 (18.4)   | 0.065   |
| Cardiac failure, n (%)        | 7 (6.9)     | 0 (0)      | 0.189   |
| Peripheral artery disease, n (%) | 17 (16.7) | 2 (5.3)    | 0.099   |
| High risk for CEA, n (%)      | 38 (37.3)   | 1 (2.6)    | <0.001  |
| Risk index for CEA, median (range) | 8 (2–19) | 6 (4–15)   | <0.001  |
| eGFR, mL/min/1.73 m², median (range) | 64.8 (7.1–132.6) | 63.4 (28.6–95.8) | 0.933 |
| Plaque parameters             |             |            |         |
| Degree of stenosis, %*, median (range) | 72.5 (22.4–95.2) | 66.0 (35.4–95.0) | 0.066 |
| Lesion length, mm, median (range) | 22.8 (10.2–38.3) | 23.0 (8.1–35.6) | 0.643 |
| Signal intensity ratio, median (range) | 1.18 (0.40–2.84) | 1.71 (0.62–2.99) | <0.001 |
| Vulnerable plaque, n (%)      | 41 (40.2)   | 32 (84.2)  | <0.001  |
| Severely calcified plaque, n (%) | 21 (20.6) | 9 (23.7)   | 0.817   |
| High cervical lesion, n (%)   | 34 (33.3)   | 1 (2.6)    | <0.001  |

TIA: Transient ischemic attack, eGFR: Estimated glomerular filtration rate. CAS: Carotid artery stenting. CEA: Carotid endarterectomy. *Based on North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria

Postoperative ischemic lesions after CAS or CEA

Symptomatic or asymptomatic lesions positive on DWI within 48 h after surgery were observed in 33 cases (32.4%) in the CAS group and 10 cases (26.3%) in the CEA group. The adjusted odds ratio (OR) with 95% confidence intervals (CIs) for positive DWI after CAS is shown in Table 3. Occurrence of postoperative ischemic lesions was independently associated with age (OR = 1.13, 95% CI: 1.01–1.26, P = 0.026) and vulnerable plaque (OR = 5.54, 95% CI: 1.48–20.70, P = 0.011). Proximal protection was weakly correlated with a risk of postoperative ischemic lesions, but strongly with aortic arch type. It was difficult to assess whether proximal protection is an independent effect because of the small number of patients. Similarly, adjusted ORs with 95% CIs for positive DWI after CEA are shown in Table 4. Neither age nor other factors were significantly correlated with a risk of postoperative ischemic lesions in CEA.

Illustrative case: A patient with major stroke

The patient was a 78-year-old man with asymptomatic right ICA stenosis who was indicated for surgical treatment because the stenosis was >70%, and further, progression was observed. Preoperative fluid-attenuated inversion-recovery MRI showed chronic ischemic white matter changes and no acute changes [Figure 2a]. The carotid plaque was judged to be vulnerable because the SIR on black-blood FS T1-MRI was >1.5 [Figure 2b], but we chose CAS due to old age and a relatively high cervical lesion. A right common carotid angiogram revealed severe stenosis of the cervical ICA before CAS [Figure 2c]. Under dual protection, predilation was
performed with a 4 × 30 mm angioplasty catheter [Figure 2d]. A 10 × 24 mm self-expanding stent (Carotid Wallstent; Boston Scientific, Natick, MA, USA) was deployed and postdilation was performed with a 4.5 × 20 mm angioplasty catheter. A poststenting angiogram showed sufficient dilatation of the right ICA stenosis [Figure 2e]. The presence of debris was confirmed in aspirated blood. Postoperatively, the patient developed major ipsilateral stroke and presented with the left hemiparesis and left spatial neglect [Figure 2f].

DISCUSSION

The characteristics of the patients show that the results of this study reflect the treatment algorithm used at our facility, indicating that triage is accurate. In the CAS group, adequate selection of protection devices and surgical procedure can produce satisfactory results, and combined with CEA, the rate of major adverse events was very low. However, vulnerable plaques were associated with increased risk of postoperative stroke after CAS, but not after CEA. These results are consistent with the high incidence of cerebral ischemic complications in CAS for vulnerable plaques with high signal intensity on MRI. In CEA, carotid plaque is removed under occlusion and the plaque characteristics are unlikely to be related to embolic risk, consistent with the finding that asymptomatic ischemic lesions are more frequently seen on DWI after carotid stenting than after endarterectomy.

All patients with major stroke in this study had preoperative findings of possible vulnerable plaque, but some also had factors that discouraged CEA under general anesthesia, such as advanced age and a high cervical lesion, so CAS was chosen. CAS for vulnerable plaques requires stringent embolic protection, and combined proximal and distal protection might be more useful and safer than single protection alone. At our center, flow arrest (proximal protection) and distal filter protection are mainly used. Construction of a flow reversal system is not used, but is considered to be a good option. A previous retrospective study found that a combination of dual protection and blood aspiration provides effective distal embolic protection, suggesting that CAS using this approach is safe in patients with vulnerable plaques. CAS with a dual layer stent has a low incidence of perioperative complications and restenosis and this may be a solution for protection from cerebral embolic events.

Multivariate logistic analysis revealed that proximal protection was weakly correlated with a risk of postoperative ischemic lesions, but strongly correlated with the aortic arch type. Instrumentation of an atherosclerotic aortic arch may release plaque debris to the brain during carotid catheterization. A balloon-guided catheter has a larger diameter and higher rigidity than a nonballoon-guided catheter, and proximal balloon protection involves more complex surgical procedures. Increased shear stress in the artery walls during carotid catheterization and the longer procedure time may affect the results. For this reason, it is important to evaluate the approach route for using proximal protection.

Table 3: Multivariate logistic regression analysis of potential risk factors for postoperative ischemic lesions after CAS (n=102).

| Variable                                      | OR   | 95% CI     | P-value |
|-----------------------------------------------|------|------------|---------|
| Age (1-year increment)                        | 1.13 | 1.01–1.26  | 0.026   |
| Degree of stenosis (1% increment)             | 0.98 | 0.95–1.01  | 0.234   |
| Lesion length (1 mm increment)                | 1.10 | 0.98–1.24  | 0.102   |
| Degree of contralateral stenosis (1% increment) | 0.99 | 0.98–1.01  | 0.246   |
| Symptomatic lesion                            |      |            |         |
| No                                            |      |            |         |
| Yes                                           | 0.32 | 0.09–1.11  | 0.073   |
| Vulnerable plaque                             |      |            |         |
| No                                            |      |            |         |
| Yes                                           | 5.54 | 1.48–20.70 | 0.011   |
| Aortic arch type                              |      |            |         |
| II                                            |      |            |         |
| III                                           | 2.88 | 0.80–10.40 | 0.107   |
| Distal protection                             |      |            |         |
| No                                            |      |            |         |
| Yes                                           | 0.99 | 0.031–31.60| 0.996   |
| Proximal protection                           |      |            |         |
| No                                            |      |            |         |
| Yes                                           | 2.25 | 0.48–10.60 | 0.302   |
| Stent type                                    |      |            |         |
| Closed cell                                   |      |            |         |
| Open cell                                     | 2.81 | 0.72–11.00 | 0.137   |

Table 4: Multivariate logistic regression analysis of potential risk factors for postoperative ischemic lesions after CEA (n=38).

| Variable                                      | OR   | 95% CI     | P-value |
|-----------------------------------------------|------|------------|---------|
| Age (1-year increment)                        | 0.92 | 0.80–1.05  | 0.211   |
| Degree of stenosis (1% increment)             | 0.98 | 0.93–1.03  | 0.432   |
| Lesion length (1 mm increment)                | 0.98 | 0.86–1.10  | 0.695   |
| Degree of contralateral stenosis (1% increment) | 1.00 | 0.96–1.04  | 0.857   |
| Clamp time (1 min increment)                  | 1.00 | 0.97–1.03  | 0.965   |
| Symptomatic lesion                            |      |            |         |
| No                                            |      |            |         |
| Yes                                           | 1.10 | 0.18–6.57  | 0.917   |
| Vulnerable plaque                             |      |            |         |
| No                                            |      |            |         |
| Yes                                           | 2.45 | 0.18–33.80 | 0.502   |

OR: Odds ratio, CI: Confidence interval, CAS: Carotid artery stenting.
In some CEA cases, viscous fluid flowed out from the carotid plaque after cutting the adventitia of the common carotid artery [Figure 3]. It is important to extract the liquid plaque in soft plaques correctly because viscous fluid can pass through a distal protection device, which may result in severe cerebrovascular embolism. A retrospective study found that MRI categories based on a combination of SIR in black-blood FS T1-MRI and T2-MRI could differentiate liquid from solid components with high accuracy. Thus, if preoperative assessment shows high SIR on both FS T1-MRI and T2-MRI, the indication for CEA should be reconsidered or more stringent embolic protection such as dual protection and blood aspiration should be used if CAS is the only choice because of other characteristics.

The Carotid Revascularization Endarterectomy versus Stent Trial demonstrated superiority of CEA in elderly patients. Outcomes were slightly better after CAS for patients aged <70 years and better after CEA for patients >70 years. In addition, Macdonald et al. found a significant increase in complications of CAS in cases with tortuous access routes and...
in patients aged >80 years. In our analysis, aging was not significantly correlated with a risk of postoperative ischemic lesions in CEA, whereas the occurrence of these lesions was independently associated with aging in CAS. Similar results were found in a meta-analysis. Thus, in our treatment algorithm, it is important not to hesitate to perform CEA just because of old age, with due consideration of the risks of general anesthesia, such as cardiac and respiratory function. Furthermore, although we chose CAS in the illustrative case, we should not hesitate CEA because of a slight anatomical risk such as a relatively high-level lesion which does not strictly meet with the definition of high cervical stenosis.

In recent years, the use of CAS has increased markedly due to advantages of convenience, less invasiveness, and advances in devices. In 2019, 8471 CAS and 4053 CEA procedures were performed in Japan, and the proportion of CAS is increasing yearly. In real-world experience of CAS, data from the Japanese Registry of NeuroEndovascular Therapy 3 (JR-NET3) showed that 9.8% of patients had major stroke and 33.4% had minor stroke. The second asymptomatic carotid surgery trial (ACST-2), an international multicenter randomized trial of CAS versus CEA, suggested that serious complication is similarly uncommon after both procedures, and that the long-term effects on fatal or disabling stroke are also comparable, with overall rates of 1% for disabling stroke or death and 2% for nondisabling procedural stroke.

Two of our patients (1.4%) had major stroke and 3 (2.1%) had minor stroke, indicating that our results are not inferior to those of JR-NET3 or ACST-2. Our algorithm reflects the treatment situation in Japan, where the proportion of CAS procedures is increasing yearly, and we obtained relatively good outcomes. There are conflicting views of CAS and CEA, but the key issue for good outcomes seems to be use of an appropriate treatment algorithm. A strategy using CAS as first-line treatment and CEA based on individual factors, with performance of the two procedures by the same team, appears to be reasonable.

This study has several limitations. First, it was a single-center retrospective study with no control group, and the statistical power was low due to the small number of patients. Second, there was no uniform algorithm used for medical treatment and the algorithm for surgical treatment was not sufficiently rigorous, especially in determining the risks of general anesthesia. In addition, the follow-up period was short and evaluation of long-term outcomes for events such as restenosis and recurrent stroke is required.

CONCLUSION

CAS as first-line treatment and CEA are effective and safe, and this strategy reflects the treatment situation in Japan. It is beneficial to assign cases to CEA based primarily on plaque vulnerability and anatomical risk, but the indications and procedures can be modified and it seems to be important to not hesitate to perform CEA just because of old age.

Declaration of patient consent

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

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Brott TG, Halperin JL, Abbara S, Bacharach JM, Barr JD, Bush RL, et al. 2011 ASA/ACCF/AHA/AANN/AANS/ACR/ANSR/CNS/SAIP/SCAI/SIR/SNIS/SVM/SVS guideline on the management of patients with extracranial carotid and vertebral artery disease. A report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, and the American Stroke Association, American Association of Neuroscience Nurses, American Association of Neurological Surgeons, American College of Radiology, American Society of Neuroradiology, Congress of Neurological Surgeons, Society of Atherosclerosis Imaging and Prevention, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of NeuroInterventional Surgery, Society for Vascular Medicine, and Society for Vascular Surgery. Circulation 2011;124:e54-130.
2. Brott TG, Hobson RW 2nd, Howard G, Roubin GS, Clark WM, Brooks W, et al. Stenting versus endarterectomy for treatment of carotid-artery stenosis. N Engl J Med 2010;363:11–23.
3. Castro-Afonso LH, Abud LG, Rolo JG, Santos AC, Oliveira LD, Barreira CM, et al. Flow reversal versus filter protection: A pilot carotid artery stenting randomized trial. Circ Cardiovasc Interv 2013;6:552–9.
4. Flach HZ, Oublous M, Hendriks JM, Van Sambeek MR, Veenland JF, Koudstaal PJ, et al. Cerebral ischemia after carotid intervention. J Endovasc Ther 2004;11:251–7.
5. Gates L, Botta R, Schlosser F, Goodney P, Fokkema M, Schermerhorn M, et al. Characteristics that define high risk in carotid endarterectomy from the Vascular Study Group of New England. J Vasc Surg 2015;62:929–36.
6. Halliday A, Bulbulia R, Bonati LH, Chester J, Craddock-Bamford A, Peto R, et al. Second asymptomatic carotid surgery trial (ACST-2): A randomised comparison of carotid artery stenting versus carotid endarterectomy. Lancet 2021;398:1065–73.
7. Howard G, Roubin GS, Jansen O, Hendriks J, Halliday A, Fraedrich G, et al. Association between age and risk of stroke or death from carotid endarterectomy and carotid stenting: A meta-analysis of pooled patient data from four randomised
trials. Lancet 2016;387:1305-11.
8. Howie BA, Witek AM, Hussain MS, Bain MD, Toth G. Carotid endarterectomy and carotid artery stenting in a predominantly asymptomatic real-world patient population. World Neurosurg 2019;127:e722-6.
9. Iihara K, Saito N, Suzuki M, Date I, Fujii Y, Houkin K, et al. The Japan neurosurgical database: Statistics update 2018 and 2019. Neurofl Med Chir (Tokyo) 2021;61:675-710.
10. Kajihara Y, Sakamoto S, Kiura Y, Mukada K, Chaki T, Kajihara S, et al. Comparison of dual protection and distal filter protection as a distal embolic protection method during carotid artery stenting: A single-center carotid artery stenting experience. Neurosurg Rev 2015;38:671-6.
11. Kubota H, Sanada Y, Yoshioka H, Tasaki T, Shiroma J, Miyauchi M, et al. C1 transverse process-hyoid bone line for preoperative evaluation of the accessible internal carotid artery on carotid endarterectomy: Technical note. Acta Neurochir (Wien) 2015;157:43-8.
12. Macdonald S, Lee R, Williams R, Stansby G, Delphi Carotid Stenting Consensus Panel. Towards safer carotid artery stenting: A scoring system for anatomic suitability. Stroke 2009;40:1698-703.
13. Moody AR, Allder S, Lennox G, Gladman J, Fentem P. Direct magnetic resonance imaging of carotid artery thrombus in acute stroke. Lancet 1999;353:122-3.
14. Mutzenbach JS, Griessnueter CJ, Broussalis E, Pikija S, Moscote-Salazar LR, Milieski K, et al. Follow-up after carotid stenting with the CASPER stent system: A duplex ultrasound evaluation. J Vasc Surg 2020;72:2054-60.e2.
15. Park JH, Lee JH. Carotid artery stenting. Korean Circ J 2018;48:97-113.
16. Report of Japan Neurosurgery Registry (2015-2017). Neurol Med Chir (Tokyo) 2019;59(Spec):13-81.
17. Sakamoto S, Kiura Y, Okazaki T, Shinagawa K, Ishii D, Ichinose N, et al. Carotid artery stenting for vulnerable plaques on MR angiography and ultrasonography: Utility of dual protection and blood aspiration method. J Neurointerv Surg 2016;8:1011-5.
18. Tokuda R, Yoshimura S, Uchida K, Yamada K, Satow T, Iihara K, et al. Real-world experience of carotid artery stenting in Japan: Analysis of 8458 cases from the JR-NET3 nationwide retrospective multi-center registries. Neurofl Med Chir (Tokyo) 2019;59:117-25.
19. Watanabe Y, Nagayama M, Suga T, Yoshida K, Yamagata S, Okumura A, et al. Characterization of atherosclerotic plaque of carotid arteries with histopathological correlation: Vascular wall MR imaging vs. color Doppler ultrasonography (US). J Magn Reson Imaging 2008;28:478-85.
20. Yoshida K, Endo H, Sadamasa N, Narumi O, Chin M, Inoue K, et al. Evaluation of carotid artery atherosclerotic plaque distribution by using long-axis high-resolution black-blood magnetic resonance imaging. J Neurosurg 2008;109:1042-8.
21. Yoshimura S, Yamada K, Kawasaki M, Asano T, Kanematsu M, Takamatsu M, et al. High-intensity signal on time-of-flight magnetic resonance angiography indicates carotid plaques at high risk for cerebral embolism during stenting. Stroke 2011;42:3132-7.

How to cite this article: Okamoto T, Inoue Y, Oi Y, Taniyama I, Houri T, Teramukai S, et al. Strategy of carotid artery stenting as first-line treatment and carotid endarterectomy for carotid artery stenosis: A single-center experience. Surg Neurol Int 2022;13:513.

Disclaimer

The views and opinions expressed in this article are those of the authors and do not necessarily reflect the official policy or position of the Journal or its management. The information contained in this article should not be considered to be medical advice; patients should consult their own physicians for advice as to their specific medical needs.