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Relevance of Distal Arterial Collapse in Stenting of Atherosclerotic Near-Occlusion of the Carotid Artery

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ABSTRACT

BACKGROUND AND PURPOSE: Carotid near-occlusion has been subclassified into near-occlusion with and without collapse. We aimed to compare the technical success and perioperative complication rates of carotid artery stent placement with special attention to these subtypes to see whether there is a clinical relevance of this subclassification.

MATERIALS AND METHODS: From January 2014 to January 2018, we retrospectively evaluated all patients with atherosclerotic extracranial carotid stenosis treated by carotid artery stent placement. Patients with near-occlusion were identified based on DSA findings. Patient characteristics, the presence of criteria for near-occlusion and collapse, arterial diameters, technical success rate, and perioperative (≤30 days) complications were analyzed.

RESULTS: We identified 59 near-occlusions in 58 (46 men, 11 with collapse) patients. Forty-one patients (70.7%) were symptomatic. Technical success rate was 98.3% (58 of 59 procedures). In 1 case of near-occlusion with collapse, we were not able to pass through the stenosis. Compared with patients without collapse (4.2% of 48 cases), those with collapse (30% of 10 stented patients) had significantly higher rates of postintervention hyperperfusion syndrome ($P = .032$). In the whole cohort, the permanent morbidity and mortality rate was 3.4% (1.7% permanent morbidity and 1.7% mortality). For asymptomatic and symptomatic near-occlusion groups, the rates were 0% and 4.9%, respectively. The composite risk of stroke, death, and myocardial infarction was similar between the groups with and without collapse ($P = .682$). Rate of hyperperfusion syndrome (with or without permanent deficit) was similar ($P = 1$) in preoperatively symptomatic patients versus asymptomatic patients (9.8% vs 5.9%). Internal carotid artery diameter consistently increased after carotid artery stent placement in patients with collapse and was not related to the development of hyperperfusion syndrome.

CONCLUSIONS: Care should be taken to minimize hyperperfusion risk in patients with near-occlusion undergoing CAS, especially in the subgroup of patients with collapse and in patients with both symptomatic and asymptomatic carotid stenosis.

ABBREVIATIONS: CEA = carotid endarterectomy; CAS = carotid artery stent placement; ECA = external carotid artery; TCD = transcranial Doppler

Carotid near-occlusion is defined as very severe carotid artery stenosis with a reduced lumen diameter distal to the stenotic segment. Ischemic stroke is one of the leading causes of morbidity and mortality worldwide. Carotid artery stenosis is responsible for 18%–29% of these cases[1]. It has been well demonstrated that stroke risk increases with an escalating degree of carotid stenosis.[2,3] Therefore, it would be expected that carotid near-occlusion would be associated with the greatest stroke risk. However, stroke risk in carotid near-occlusion is not well defined. The post hoc analyses of the North American Symptomatic Carotid Endarterectomy Trial (NASCET) and European Carotid Surgery Trial (ECST) reported a low stroke risk with medical therapy in near-occlusion compared with severe carotid artery stenosis without near-occlusion.[2,3] In contrast, some authors have reported that symptomatic patients with near-occlusion have a high stroke risk with medical treatment,[4-7] and one of the latest publications on carotid near-occlusion treatment suggests that there is a higher 30-day stroke and death rate after medical therapy than after carotid endarterectomy (CEA) or carotid artery stent placement (CAS) because the 1-year stroke-free or death-free survival rates were 96.1% for CEA, 94.4% for CAS, and 81.2% for medical therapy.[8] Thus, these results favor carotid revascularization in this subset of patients with severe carotid stenosis.

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One would assume that the presence of a collapsed distal carotid artery is a major risk for CAS. Whether near-occlusion with collapse is associated with a higher risk of failure or complications compared with near-occlusion without collapse remains to be shown.[9,10] We aimed to compare the technical success and perioperative (≤30 days) complication rates of CAS for the 2 near-occlusion subtypes to determine whether there is a clinical relevance of this subclassification in endovascular treatment.

MATERIALS AND METHODS

After ethical approval was obtained from our institutional review board, we retrospectively evaluated all patients with atherosclerotic extracranial ICA stenosis treated with CAS in a single medical institution between January 2014 and January 2018. Patients with near-occlusion were identified based on cerebral DSA findings. The DSA images were evaluated by 2 interventional radiologists independently based on the DSA criteria; in case of disagreement, consensus was reached. Two of the 4 following criteria described by Fox et al[11] were sought for a near-occlusion diagnosis: 1) delayed arrival of contrast medium in the distal ICA, 2) evidence of intracranial collaterals, 3) reduced ICA diameter compared with the contralateral ICA diameter, and 4) reduced ICA diameter compared with the ipsilateral external carotid artery (ECA) diameter. Collapse was defined as a stringlike lumen distal to the stenotic segment. Patients who were unable or unwilling to stay on antiplatelet agents, those with total occlusion of the cervical carotid artery on the initial diagnostic angiogram, patients with occlusion of the intracranial carotid artery, and patients with suspected vasculitic involvement of the cervical carotid artery were not offered CAS as a treatment technique. Patient characteristics, the presence of criteria for near-occlusion and collapse, arterial diameters, technical success rate, and perioperative (≤30 days) complications were analyzed.

Before carotid revascularization was performed, all patients were evaluated by 2 stroke neurologists. Baseline neurologic status was evaluated based on the modified Rankin scale before the procedure. Patients were defined as symptomatic if they had a transient ischemic attack (ocular or hemispheric) or stroke without severe disability (mRS score ≤3) up to 6 months before the procedure. Disabled patients (mRS of 4 or more) were not treated except for a single patient with a baseline mRS score of 4. This patient was treated because the patient also had contralateral ICA occlusion and insufficient collateral flow from the posterior circulation. Hyperperfusion syndrome was diagnosed based on clinical findings, which included seizures, focal neurologic deficits, or a deterioration of consciousness without evidence of a new stroke on cross-sectional imaging. In patients with subtle or equivocal findings, the presence of cerebral edema, hemorrhage, or both in the ipsilateral carotid territory without an acute territorial stroke led to the diagnosis of hyperperfusion syndrome.

The angiograms of the patients with collapse were evaluated for pre- and postprocedure ICA diameters. The measurement was made at the same level based on bony landmarks. We assumed the diameter of the ECA was constant during the procedure, and the ratio of the postprocedure diameter of the ICA over the preprocedure diameter was calculated with the following formula: (postprocedure ICA diameter/postprocedure ECA diameter) divided by (preprocedure ICA diameter/preprocedure ECA diameter). Angiograms were also scrutinized for dissection, thrombus formation, and intracranial embolization.

All of the patients referred for treatment of carotid stenosis by stent placement were initially evaluated by CTA. Those who had an ultrasound evaluation as an initial diagnostic technique were also verified by CTA. In case of equipoise, a contrast-enhanced head and neck MRA was performed. All patients who had noninvasive confirmation of the stenosis first underwent a cerebral DSA. None of the patients were denied CAS based on anatomy of the aortic arch on CTA or plaque morphology on ultrasound imaging. Stent placement was universally performed in a second session.

Interventional Procedure

Written informed consent was obtained from all patients before the procedure. Dual antiplatelet therapy (75 mg of clopidogrel and 300 mg of aspirin per day) was orally initiated at least 5 days before the procedure for elective cases. For acute cases, patients were preloaded with 6 tablets of clopidogrel. Although the value of intraprocedural monitoring studies such as transcranial Doppler sonography (TCD) during CAS has been delineated well in the literature,[12] we were unable to use this technique intraprocedurally in our major tertiary referral center, because of logistical reasons such as scheduling issues and unavailability of a full-time specialist dedicated to TCD in the angiography suite. After insertion of the femoral vascular sheath under monitored anesthesia, common carotid artery catheterization was performed with a long sheath. The stenosis was crossed with a microguidewire. When a distal protection device was used, it was either advanced directly over this guidewire, or alternatively, a small-bore microcatheter was used to cross the stenosis, and it was exchanged with the protection device. Then, under distal protection (Spider FX, Medtronic or Emboshield, Abbott Vascular), proximal protection (Mo.Ma system, Medtronic), or dual (proximal and distal) cerebral protection, angioplasty was performed with a 2- to 3-mm balloon catheter to dilate the stenotic segment. Then, a self-expandable stent (Protégé, Medtronic) was deployed. If there was suspicion for suboptimal plaque coverage, a second stent was deployed telescopically. Postdilation was performed with a monorail angioplasty balloon catheter after stent deployment.

Blood pressure and heart and expiratory rates were continuously monitored during the procedure by anesthesiologists. After the procedure, all patients were admitted to an intensive care unit for at least 24 hours. During the procedure and the postoperative period, the mean blood pressure was kept at around 100 mm Hg, preferably by using esmolol or nitroglycerin infusion as needed for 24–48 hours.

Patients were advised to use 300 mg/day of aspirin and 75 mg/day of clopidogrel for at least 6 months after the procedure and were asked to return for a 1-month clinical follow-up and a 6-month cervical Doppler sonography study.

Statistical Analysis

The SPSS 20.0 (IBM) program was used for statistical analysis. Continuous data were presented as mean ± SD and categoric data as frequency and percentage. Categoric variables compared were with use of the χ-square test (Fisher exact test if required), and mean values were compared with use of the independent-samples *t*

Table 1: Angiographic findings in carotid near-occlusion

Findings	*n* (%)
Near-occlusion with collapse	11 (18.6)
Delayed arrival of contrast medium in the distal ICA	53 (89.8)
Evidence of intracranial collateral	47 (79.7)
Reduced ICA diameter compared with the contralateral ICA diameter[a]	56 (100)
Reduced ICA diameter compared with the ipsilateral ECA diameter	53 (89.8)

[a]Not applicable in 3 patients because of contralateral total occlusion or near-occlusion with collapse.

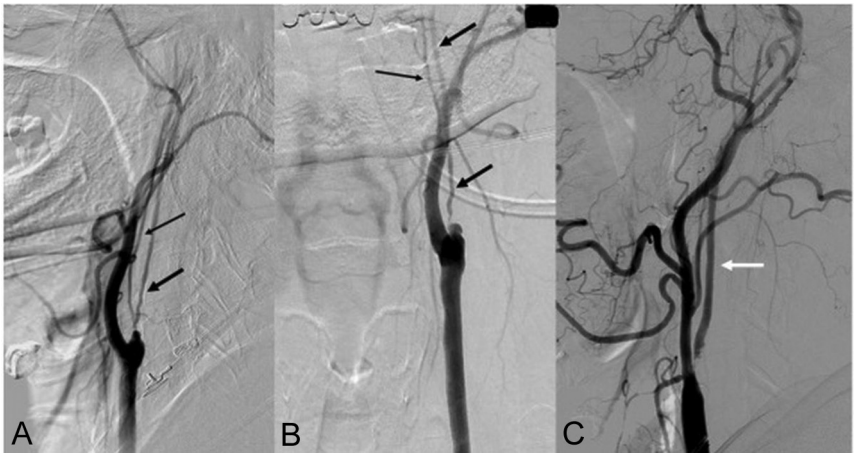

FIG 1. *A* and *B*, Near-occlusion with collapse. Cervical angiograms in lateral and anteroposterior projections, respectively. *Thick black arrows* indicate the string sign in near-occlusion with collapse. *Thin black arrows* indicate ascending pharyngeal artery. The ascending pharyngeal artery should not be confused with near-occlusion with collapse, especially in the setting of total ICA occlusion. *C*, Near-occlusion without collapse. Lateral cervical angiogram shows tight stenosis of ICA origin and diameter reduction in distal cervical ICA (*white arrow*) compared with the ipsilateral external carotid artery.

Table 2: Patient characteristics

Characteristics	Near-Occlusion with Collapse (*n* = 11) (%)	Near-Occlusion without Collapse (*n* = 47) (%)	*P* Value
Age (mean)	65.73 ± 11.6	67.81 ± 8.7	.509
Male sex	9 (81.8)	37 (78.7)	1
Left side	6 (54.5)	29 (60.4)	.745
Symptomatic	8 (72.7)	33 (70.2)	1
Hypertension	7 (63.6)	36 (76.6)	.450
Diabetes	6 (54.5)	26 (53.3)	1
Active smoker	4 (36.4)	24 (51.1)	.380
Hypercholesterolemia	5 (45.5)	25 (53.2)	.644
Coronary heart disease	4 (36.4)	15 (31.9)	1
Contralateral severe stenosis (≥70)/ near-occlusion/total occlusion)	1[a] (9.1)	8[b] (16.7)	1

[a]Near-occlusion without collapse.
[b]One near-occlusion with collapse, 2 total occlusions, and 5 severe stenoses.

test (Mann-Whitney *U* test if required). Statistical significance was set at $P = .05$.

RESULTS

A total of 58 patients (46 men, mean age 67.41 ± 9.2 years) with 59 near-occlusions were treated with CAS. One patient had bilateral near-occlusion. Near-occlusion with collapse was diagnosed in 12 cases by the first interventional radiologist and in 11 by the second interventional radiologist. In 11 of these, the final diagnosis was near-occlusion with collapse. By consensus, the last patient was finally diagnosed with near-occlusion without collapse.

The angiographic findings in the patients with near-occlusion are listed in Table 1. Angiographic examples of near-occlusions with and without collapse are demonstrated in Fig 1. There was no significant difference between the near-occlusion with collapse and near-occlusion without collapse groups based on patient characteristics (Table 2). The near-occlusions were symptomatic in 41 patients (70.7%, including 23 patients with stroke, 16 patients with TIA, and 2 patients with acute ICA occlusion after the diagnosis of near-occlusion) and asymptomatic in 17 patients (29.3%). Two patients (1 with collapse and 1 without collapse) were treated emergently for acute ICA occlusion, which developed as the patient was waiting for a CAS procedure after the elective cerebral DSA. Otherwise, CAS was performed electively.

Fifty-eight of the 59 CAS procedures were successful. In 1 case of near-occlusion with collapse, we were not able to pass through the stenosis because of patient motion under conscious sedation; the patient was advised to undergo stent placement under general anesthesia during the procedure, but he refused to have any type of procedure under general anesthesia. Examples of the procedures in 2 patients with and without collapse are provided in Figs 2 and 3. Additional stents were used in 12 of the 58 cases. In 10 cases, the second stent was deployed because the operator's threshold to deploy a second stent in case of the slightest suspicion of plaque prolapse is low. In 2 cases, the second stent was deployed as the position of the first stent was deemed to be less than ideal by the operator. Overall, 17 patients were asymptomatic, and the permanent morbidity and mortality rate, excluding transient symptoms related to hyperperfusion syndrome, in this group was 0%. One patient of 17 in this group had an episode of a seizure related to hyperperfusion syndrome and was treated with antiepileptics promptly and without any clinical consequences. Forty-one patients were symptomatic. Of these, 1 died as a result of stent occlusion, and 1 had residual

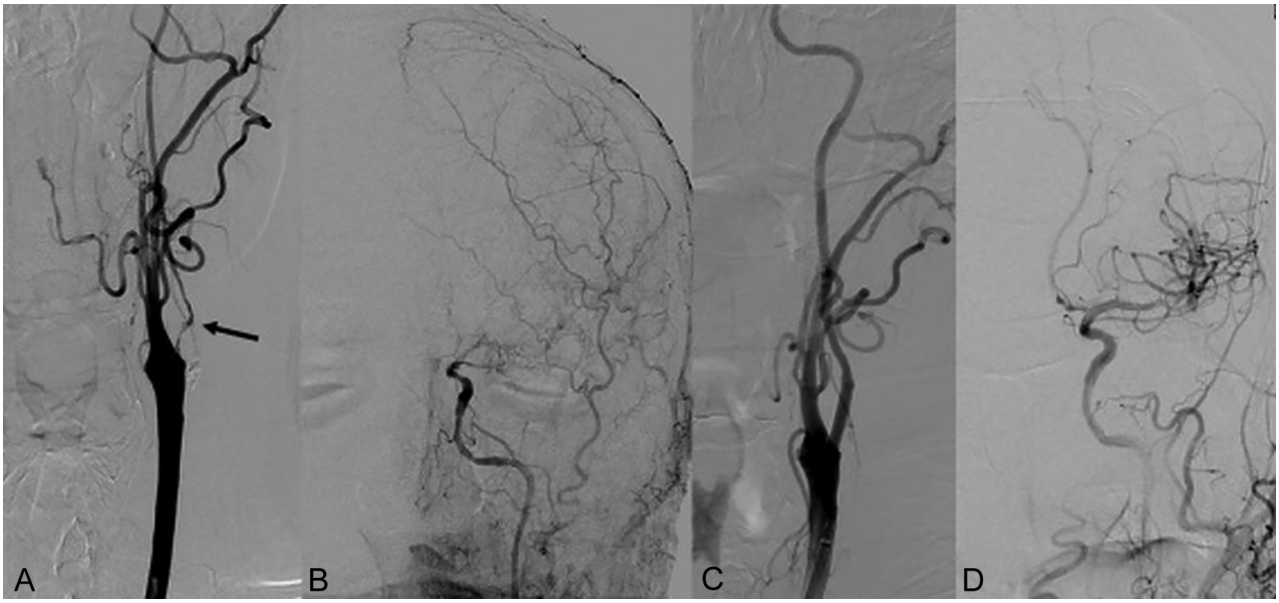

FIG 2. *A* and *B*, Preoperative cervical and cranial angiograms in patient with near-occlusion with collapse (*arrow* in *A*), respectively. *C* and *D*, Postoperative cervical and cranial angiograms show improvement of diameter of ICA and restoration intracranial blood flow, respectively.

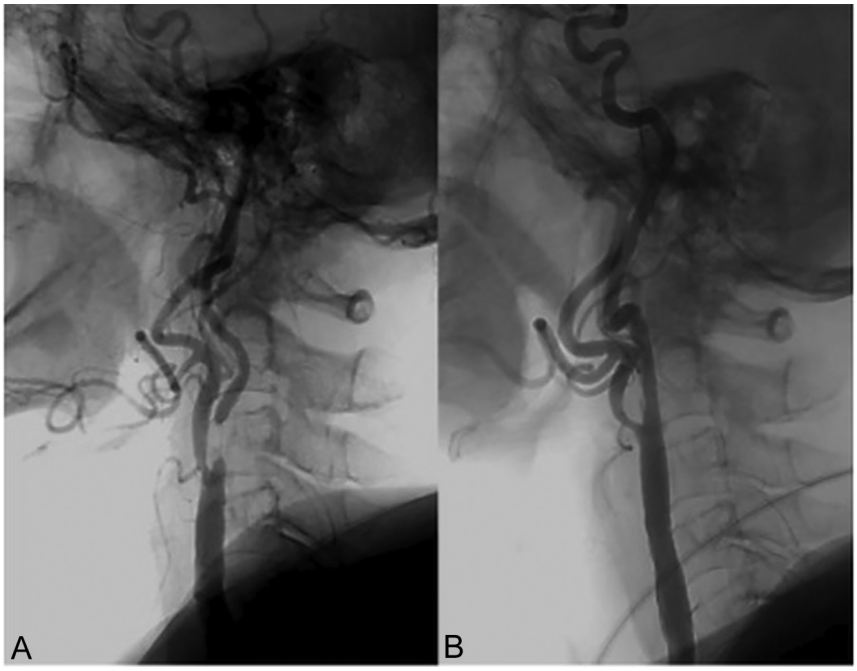

FIG 3. Pre- and postoperative cervical lateral angiograms in patient with near-occlusion without collapse, respectively.

hemiparesis after hyperperfusion syndrome, resulting in a permanent morbidity and mortality rate of 4.9%. In this group, 1 patient complained of headache, and head CT revealed minor intracranial hemorrhage (Fig 4). The patient's initial mRS score was 3 and remained 3 on follow-up.

In 1 patient without collapse, plaque rupture and acute thrombus formation were encountered immediately after predilation. Thrombectomy and stent placement were performed without any clinical adverse outcomes. In 3 patients (2 without collapse and 1 with collapse) following postdilation, antegrade slow flow was encountered proximal to the distal protection device as previously described in the literature.[13] After aspiration of free debris and removal of the distal filter, antegrade flow was restored without clinical outcomes in 2 patients without collapse. In the patient with collapse, intracranial severe stenosis was discovered after postdilation and was unrecognizable before angioplasty. Aspiration of free debris and removal of the distal filter did not restore the optimum antegrade flow in this case. Asymptomatic stent occlusion occurred without clinical outcomes within 1 month after the procedure.

Five patients (8.6%) experienced hyperperfusion syndrome during the perioperative period. Three patients experienced a seizure within 24 hours after the procedure. One patient experienced left upper extremity paresis without any signs of stroke on MR imaging 3 weeks after the procedure, consistent with late hyperperfusion syndrome. There was a decline in the patient's baseline mRS score from 1 to 2. One of the patients who presented with acute ICA occlusion experienced small patchy cortical and subarachnoid hemorrhagic foci without a deterioration in baseline neurologic status (Fig 4). Compared with patients without collapse (2 of 48 patients [4.2%]), those with collapse (3 of 10 stented patients

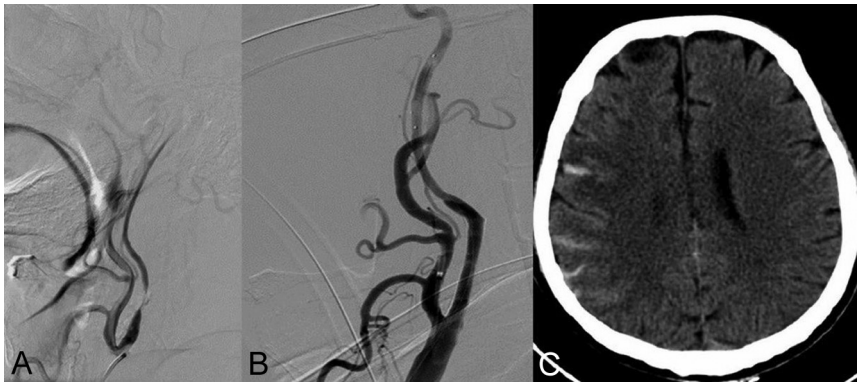

FIG 4. Right carotid angiogram (*A*) before and (*B*) after deployment of a carotid stent. *C*, Postprocedure cranial CT image of the same patient shows right-sided hemorrhage related to hyperperfusion syndrome after carotid artery stent placement.

[30%]) had significantly higher rates of postintervention hyperperfusion syndrome (Fisher exact test, $P = .032$). In 2 of the 3 patients with seizures, seizure control was achieved with antiepileptic drugs. In a patient (without collapse and contralateral occlusion) with intractable seizures, clopidogrel was stopped because of the possibility of an intracranial hemorrhage. The patient was discharged with aspirin and low-molecular-weight heparin after the seizures were controlled. The patient failed to comply with his medication, presented with stent occlusion, and eventually died. One patient (1.7%) developed perioperative minor ischemic stroke without a change in the patient's baseline mRS score (preoperative mRS score, 3). There was no myocardial infarction in either of the groups. In the whole cohort, the overall permanent morbidity and mortality rates were 1.7% and 1.7%, respectively. The ratio of postprocedural ICA diameter divided by preprocedural ICA diameter ranged from 1.16 to 3.69 (mean, 1.76). There was no residual stenosis in the collapse group per NASCET criteria postprocedurally, mainly because the distal artery was small to start with, and it did not expand fully immediately after stent placement (maximum postprocedure carotid artery diameter, 3.69 mm). However, the diameters of all of the stented arteries in the collapse group remained below the nominal size range of the carotid artery immediately after stent placement. There was no difference in the occurrence of hyperperfusion syndrome in patients who had symptomatic carotid near-occlusion versus those who had asymptomatic carotid near-occlusion ($P = 1$).

DISCUSSION

Carotid near-occlusion has been described with the use of various terms in the literature, such as string sign, pseudo-occlusion, slim sign, critical stenosis, and preocclusive stenosis.[14] Carotid near-occlusion was subclassified as near-occlusion with and without string sign in the NASCET.[2] Carotid near-occlusion's definition and its subclassification were revised by Fox et al[11] to include near-occlusion with collapse and the rare subtype[9] of near-occlusion without collapse. Similarly, Johansson et al[14] also advocated the use of near-occlusion with collapse and near-occlusion without collapse in their review. The definition of collapse is described clearly in the literature, yet there may be discrepancies between radiologists as to the presence of collapse, particularly when DSA is used for diagnosis. In the routine clinical setting, patient discomfort and subsequent motion during DSA may add to the degradation of images to an extent that may compromise the validity of measurements. The lack of specific criteria for patients with contralateral carotid occlusion (removing 1 of the 4 diagnostic criteria in this subset of patients) may also lead to discrepancies in the evaluation of the DSA images. Nevertheless, in real-world practice, no angiographic definition is ideal. With relatively clear-cut angiographic criteria[14] and a good-to-excellent interrater agreement of the definition of collapse or near-occlusion,[11] the diagnosis of collapse appears reliable in most patients. Although subclassification has been advocated for near-occlusion, the actual risk of treatment failure and perioperative complication rate related to CAS in the 2 different near-occlusion subgroups are unknown.

The use of invasive treatment in patients with near-occlusion is still controversial. Nevertheless, CAS performed for near-occlusion constitutes 1.72% to 28.9% of all CAS procedures.[10] Many previous CAS studies have reported high technical success with near-occlusion treatment,[15-21] with perioperative complication rates ranging between 3.3% and 17.4%. Interestingly, in studies with low perioperative complication rates, most of the complications were TIA or minor strokes. In contrast, in studies with relatively higher complication rates, the reported complications tended to be major complications, such as major stroke, hyperperfusion syndrome, and death.[16-19,22] Our overall perioperative complication rate was 10.3%. The permanent morbidity and mortality rate was 4.2% in the group without collapse and 0% in the collapsed group. Although the overall complication rate in this study appears to be high, we would like to stress the fact that the permanent morbidity and mortality rate in this cohort is within acceptable range. The overall complication rate is increased by additional inclusion of minor stroke without clinical consequences as well as hyperperfusion syndrome cases discovered by imaging in patients with symptoms such as headache, dizziness, or stiff neck without a clinical sign.

We were unable to find a previous report comparing the outcomes of CAS in the 2 near-occlusion subtypes (with collapse versus without collapse). In our cohort, although there was no significant difference in the composite risk of stroke, death, and myocardial infarction between near-occlusions with and without collapse (Fisher exact test, $P = .682$), we found a significant difference in hyperperfusion syndrome rates between these 2 groups.

In total, 5 of our patients (8.6%) experienced hyperperfusion syndrome perioperatively. Three of the 5 patients were patients with collapse. Compared with patients without collapse, those with collapse had significantly higher rates of postintervention hyperperfusion syndrome ($P = .032$). Previous studies on near-

occlusion treatment with CEA reported a trend toward a higher hyperperfusion syndrome rate in patients with collapse (0% without collapse vs 3.8% to 5.9% with collapse).[9,23] However, studies evaluating CAS in the setting of near-occlusion merged patients both with and without collapse into a single near-occlusion group without comparing these groups.[16-19,22] Hence, the actual perioperative complication rates of near-occlusions with and without collapse are unknown. To the best of our knowledge, Neves et al.[7] are the only authors who reported outcomes of CAS specifically in patients with symptomatic near-occlusion with collapse. They reported no periprocedural complications other than immediate upper limb monoparesis with complete recovery in 19 patients. These authors did not include near-occlusion without collapse in their series, so a comparison was not available.

Hyperperfusion syndrome occurs in 0%–3% of patients with conventional extracranial ICA stenosis treated with carotid intervention.[24] Decreased cerebral autoregulation and postoperative hypertension are the most consistently reported risk factors for hyperperfusion syndrome in the literature, and it is well accepted that the greater extent of ipsilateral stenosis will cause a greater risk of perioperative hyperperfusion syndrome.[24-27] Oka et al[28] demonstrated that patients with near-occlusion were more hemodynamically compromised than those with severe stenosis without near-occlusion. This could be the explanation for the higher perioperative hyperperfusion syndrome rate (8.6%) in our study. Although CAS series with no hyperperfusion syndrome have been reported in patients with near-occlusion,[16,19] in literature defining near-occlusion based on strict angiographic criteria, hyperperfusion syndrome risk was higher. For instance, consistent with our results, Son et al[17] reported an 8.7% perioperative hyperperfusion syndrome risk in patients with near-occlusion treated with CAS. Additionally, Ruiz-Salmeron et al[18] reported a 5.5% perioperative mortality rate related to intracranial hemorrhage secondary to hyperperfusion syndrome in patients with near-occlusion, which was higher than the rate in the group without near-occlusion (perioperative mortality rate related to hyperperfusion syndrome, 0.6%). However, from these studies, it is not possible to extract any data regarding the role of collapse in the near-occlusion group.

Our 8.6% rate of hyperperfusion syndrome is high albeit still within the expected range based on a recent review of the literature.[29] The reported rate of hyperperfusion syndrome actually depends on the proposed definition of the syndrome. For instance, when strict imaging criteria such as TCD imaging criteria are used, the rate of hyperperfusion syndrome is higher compared with the diagnosis of hyperperfusion syndrome based only on clinical symptoms.[12,29] This is why our results, which rely not only on definite symptoms but also on clinical suspicion supported by cross-sectional imaging, are on the higher side of the expected range. Because hyperperfusion syndrome may result in grave consequences, TCD-based detection may allow operators to act earlier, taking additional measures to prevent development of full-blown hyperperfusion syndrome. Aside from vigorous hemodynamic monitoring, these measures include early detection of this syndrome via use of intraprocedural monitoring by TCD as described earlier, prophylactic use of antiepileptics in the perioperative period in high-risk cases, and avoidance of poststent angioplasty. Although the postoperative increase in the diameter of the carotid artery was not related to hyperperfusion syndrome in this study, a possible relation may have been overlooked because of the small number of hyperperfusion syndrome cases. Intuitively, one may put forward that gradual expansion of the ICA with the intrinsic force of a self-expanding stent, without the acute luminal expansion created by poststent angioplasty, may be beneficial in minimizing the risk of hyperperfusion syndrome. This CAS method has been adopted routinely by some authors[30] and may potentially decrease the complications and cost of CAS as well.

After finding an increased rate of hyperperfusion syndrome in the collapsed group, we wanted to determine whether the enlargement of the collapsed artery may be involved in the development of hyperperfusion syndrome. When ICA diameters were calibrated according to the ECA diameter and the proportion of increase in the diameter of the collapsed ICA was calculated after the procedure, we found that the diameter of the ICA consistently increased after the treatment. However, we could not find a significant difference between ICA diameter ratios in the collapse groups with and without hyperperfusion syndrome. This suggests that the higher hyperperfusion syndrome risk in the collapse group may not be related only to luminal diameter, and other factors such as decreased cerebral autoregulation or underlying blood–brain barrier damage may be involved.

The limitations of our study include the retrospective nature of the study and the lack of mid- and long-term follow-up periods for the treated patients. In addition, it would have been ideal to perform routine postprocedure cross-sectional imaging dedicated to hyperperfusion syndrome or perfusion[31] or to obtain postprocedure TCD measurements in all patients.

CONCLUSIONS

Because near-occlusions with and without collapse differ in terms of their natural history, it is imperative that CAS outcomes be discussed separately for these 2 groups. In our series, there was no difference in the overall complication rate, but there was a higher risk of hyperperfusion syndrome in the collapse group than in the noncollapse group, which is likely associated with a lower risk of ipsilateral stroke if left untreated.[7] Thus, there may be a clinical correlate of the subclassification of near-occlusion that needs to be further verified in larger studies or registries. Strict perioperative measures to decrease the likelihood of hyperperfusion syndrome and meticulous performance of the CAS technique may be vital in the collapse group to surpass the natural history in this subgroup.

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