Simultaneous Bilateral Carotid Stenting under the Circumstance of Neuroprotection Device

A Retrospective Analysis

Y.H. LEE, T.-K. KIM, S.-I. SUH, B.J. KWON*, T.H. LEE**, O-KI KWON***, M.H. HAN*, N.J. LEE, J.H. KIM, H.Y. SEOL

Department of Diagnostic Radiology, Korea University, College of Medicine, Seoul, Korea

*Department of Radiology, Seoul National University College of Medicine, Seoul, Korea

**Department of Diagnostic Radiology, Pusan National University Hospital, Pusan, Korea

***Neuroscience Center, Seoul National University Bundang Hospital, Seoul, Korea

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Summary

In this study, in order to evaluate the feasibility and outcomes of simultaneous bilateral carotid artery stenting (CAS) with the use of neuroprotection in symptomatic patients, we conducted a retrospective analysis of 27 patients (19 men, eight women; median age, 69.2 years), all of whom had been scheduled to undergo bilateral CAS in a single setting. All patients presented with severe atherosclerotic bilateral carotid stenosis (>50% for symptomatic side, >80% for asymptomatic side), exhibiting symptoms of either a cerebrovascular accident or of a transient ischemic attack on at least one side. 48 arteries were treated with self-expandable stents. Neuroprotection devices were utilized for bilateral CAS in 11 patients, and in 16 unilateral CAS patients. We did not perform the second procedure in six patients, in cases in which a patient exhibited (a) hemodynamic instability, (b) a new neurological impairment, or (c) restlessness after a prolonged time for the first CAS. The second procedure was postponed in a staged manner.

We achieved a mean residual stenosis of 8.1 ± 5.0 % in the treated lesions. The mean procedural time for bilateral CAS was three hours and 18 minutes. 17 patients (63%) developed transient bradycardia during the balloon dilatation of one or both of the relevant arteries. Three patients

(11%) exhibited persistent bradycardia and hypotension, which required the administration of intravenous vasopressors for several days (2~7 days). None of the patients ultimately required pacemakers, or any further therapy. Two of the patients (7%) developed transient ischemic attack during the periprocedural period, but recovered completely. One patient developed a new minor stroke after the first procedure, and the second procedure was delayed in a staged manner. We observed no periprocedural deaths, major strokes, or myocardial infarctions, nor did we detect any cases of hyperperfusion syndrome within 30 days.

In summary, simultaneous bilateral CAS with neuroprotection can be performed in a single setting without increased concerns with regard to hyperperfusion syndrome, hemodynamic instability, thrombo-embolism, or procedure time, when the first CAS has been safely completed with no evidence of complications in a well-managed procedure time.

Introduction

Carotid artery stenting (CAS) is a therapy for carotid bifurcation atherosclerotic disease, which has been utilized increasingly since its inception¹⁻⁵. In recent studies, CAS coupled with distal emboli neuroprotection evidenced re-

sults superior to those associated with CAS without neuroprotection⁶⁻¹². Although the benefits of routine use of cerebral protection have not been confirmed by level one evidence¹³, it appears that the use of neuroprotection devices may become widely accepted in the treatment of carotid stenosis.

Patients suffering from bilateral carotid stenosis generally have not undergone the stenting of both arteries within a single session, due to concerns regarding the risk of severe hypotension resulting from carotid sinus baroreflex stimulation, as well as the risk of cerebral hyperperfusion¹⁴⁻¹⁷. However, bilateral CAS performed in separate procedures is also plagued by disadvantages, including delays in the definitive treatment of carotid stenosis, higher costs, and inconvenience to the patients, due to prolonged hospital admission or separate incidences of hospitalization. Recent reports concerning the feasibility of simultaneous bilateral CAS have been published, and tend to cite favorable results, although none of these studies have involved a sufficiently large population¹⁸⁻²³. In addition, concerns also exist regarding the feasibility of simultaneous bilateral CAS coupled with the use of neuroprotection^{23,24}. The primary concern here is that the use of neuroprotection necessarily means extending procedure time, especially in cases of simultaneous bilateral CAS. Although another treatment option to be considered for bilateral carotid stenosis is carotid endarterectomy (CAE), bilateral CAE in a single stage also has potential shortcomings, including longstanding cerebral hypoperfusion during the CEA, extended operation time, and/or hemodynamic instability²⁵. Here, we have retrospectively evaluated the feasibility and outcomes of simultaneous bilateral CAS under the circumstance of neuroprotection, in symptomatic patients.

Material and Methods

Patient Population

We retrospectively reviewed a prospectively collected database of CAS cases in our department, to find patients who had been scheduled to undergo bilateral CAS in a single setting for the treatment of bilateral carotid stenosis. Between July 2002 and December 2004, 27 patients were slated to undergo simultaneous bilateral CAS. Patients with severe bilateral carotid stenosis (according to NASCET crite-

ria; 50% for symptomatic side, 80% for asymptomatic side), exhibiting the symptoms of either a minor cerebrovascular accident or a transient ischemic attack (TIA) on at least one side, all of whom required carotid revascularization therapy, constituted the study group. In general, patients with 70% stenosis on the symptomatic side were enrolled in the study group. But patients with 50% and <70% stenosis on the symptomatic side also underwent CAS, in cases in which those patients had a history of recurrent symptoms of either a minor cerebrovascular accident or a TIA, which were presumed by thromboembolic propagation of carotid stenosis although the patient had been on the proper antiplatelet medication, or in cases in which the carotid stenosis evidenced a significant ulceration which could easily rupture into an embolus. All cases were assumed to be atherosclerotic disease. Patients who had experienced a stroke within one week and patients with 100% occlusion of either carotid artery were excluded. The mean time interval between the last ischemic episode (either a minor cerebrovascular accident or a TIA) and the CAS was 17.4 days. We explained the pros and cons of simultaneous bilateral CAS, in comparison with the performance of the procedure in a staged manner, in detail to all patients, and written consent was obtained from all patients prior to the procedure. Patients who were unwilling to undergo simultaneous bilateral CAS underwent staged bilateral CAS in separate procedures, and were not included in the study population.

The clinical and angiographic characteristics of the patients are summarized in table 1. The mean patient age was 69.2 ± 8.6 years, and 70% of the study patients were male. Hypertension was detected in 18 patients (67%), and eight patients (30%) had coronary diseases. None of the patients had histories of prior carotid artery angioplasty or stenting. The mean stenosis of the lesions was $78.3 \pm 10.3\%$. An independent neurologist neurologically evaluated all patients, both prior to and following the procedure.

Procedure

All patients were premedicated with 75 mg of clopidogrel and 325 mg of aspirin daily, for at least three days prior to the procedure. Patients received their regular blood pressure medications on the morning of the procedure.

Figure 1 64-year-old man with a history of recurrent TIAs. Lateral view of right (A) and left (B) CCA injection angiography shows bilateral severe degree stenosis of the carotid bulb and the proximal ICA. Recombined AP view of the bilateral carotid angiography before (C) and after (D) the angioplasty and stenting in a single session.

A single bolus of intravenous heparin (3,000~5,000 units, according to patients' body weights) was administered at the beginning of the procedure, and additional doses were administered when required for the maintenance of an activated clotting time (ACT) of between 250 and 300 seconds. During the procedure, atropine (0.6 mg) was routinely used prior to balloon dilatation. A systolic blood pressure of less than 130 mm Hg was strictly maintained throughout the procedure, and for at least 1 week after the procedure. Patients were permitted to take their regular antihypertensive medicines orally both prior to and after the procedure. Intravenous antihypertensive medications were administered during and after the procedure, if needed to maintain blood pressure.

Percutaneous access was achieved through the femoral artery in all patients. As a rule, we approached the symptomatic lesions first. Guiding catheters were positioned in the common carotid artery, using a 0.035" guidewire. A neuroprotection device was then advanced, employing dedicated catheters or sheaths for both deployment and retrieval. Neuroprotection devices were utilized for bilateral CAS in eleven patients (52%), and for unilateral CAS in 16 patients (48%). Six patients, all of whom exhibited one or more unfavorable signs after the first CAS, underwent only unilateral CAS. We did not employ a neuroprotection system for the asymptomatic side in ten patients who underwent simultaneous bilateral CAS. The decision to use a neuroprotection system for the asymptomatic side or not was predicated on the morphology of the lesions. In patients in whom the carotid lesions appeared smooth,

and evidenced no surface irregularity or ulceration prior to the procedure on ultrasound and/or angiography, the decision not to use the neuroprotection system was explained in detail to patients, and written consent was obtained before the procedure, as well.

We used NeuroShield (MedNova, Horsham, UK) in 17 arteries, Filter Wire EX (Boston Scientific, Natick, MA) in 15 arteries, and Percutaneous GuardWire Plus (Medtronic, Minneapolis, MN) in six of the arteries. In cases in which the protection device was not able to traverse a given lesion without predilatation, we used a 0.014-in floppy wire to cross the lesion, which was then dilated with a coronary balloon (2~4 mm) prior to crossing with the protection device. This was necessary in ten arteries (21%) as the result of very tight stenosis. A protection balloon or filter was positioned 2 to 3 cm above the stenosis in a straight region of the internal carotid artery. After the protection device had been deployed, the lesion was predilated with a coronary balloon (2~4 mm) in 34 arteries (71%). We implanted self-expandable stents in all of the patients. We implanted 26 Johnson & Johnson-Cordis Smart Stent (Johnson & Johnson-Cordis, Warren, NJ), 14 Boston Scientific Carotid Wallstent, and 6 Cook Zilver Stent (Cook, Bloomington, IN) devices. After stent deployment, 26 of the lesions (54%) were postdilated with 5 or 6 mm peripheral angioplasty balloons, at the operator's discretion. The neuroprotection devices were then retrieved. After the completion of the first procedure, we evaluated the patients meticulously with complete neurological checks and measurement of vital signs. Anteroposterior and lateral cerebral angiograms were ob-

tained. We did not perform the second procedure in 6 patients who had been scheduled to undergo simultaneous bilateral CAS, in cases in which a patient exhibited any of the aforementioned unfavorable signs or angiographic findings after the first CAS, including (a) unstable blood pressure or heart rate (four patients), (b) development of a new neurological impairment (one patient), or (c) likelihood of restlessness after a prolonged procedure time (one patient). The second procedure was simply postponed in a staged manner.

For the contralateral carotid artery lesions, we followed an identical procedure (figure 1). The procedural data are provided in table 2.

After the procedure, the patients were monitored in the intensive care unit for an additional 24 hours. Careful hemodynamic monitoring, including meticulous blood pressure control, was conducted, in addition to frequent neurological examinations. Heparin was physiologically tapered off. Patients received clopidogrel for three months, and aspirin therapy continued for an indefinite period.

Results

We achieved a mean stenosis of $8.1 \pm 5.0\%$ in the treated lesions. The mean procedural time (beginning at the time that femoral arterial access was obtained, and ending with the attainment of hemostasis in the access site) was

three hours and 18 minutes in patients undergoing simultaneous bilateral CAS. 20 patients (74%) developed bradycardia and/or hypotension during the balloon dilatation of one or both of the relevant arteries. Most of the patients, however, exhibited normal cardiac rhythms and pressure after balloon deflation, with the help of an additional dose of atropine in some patients. However, four patients (15%) exhibited unstable blood pressure and/or heart rate, which persisted after completion of the first CAS. In these cases, the second procedure was postponed in a staged manner, and the patients exhibited normal cardiac rhythms and pressure within 24 hours. Three patients (11%) evidenced persistent bradycardia and hypotension after completion of the second procedure, which required the administration of intravenous vasopressors for several days (2~7 days) in order to maintain optimal blood pressure. None of these patients were believed to have experienced sufficient blood loss, either internally or externally, to account for the degree of observed hypotension. Hemoglobin levels were within normal range in all patients during the postprocedural period. All patients were restored to optimal blood pressure ranges uneventfully, with the meticulous application of blood pressure control. None of the patients ultimately required pacemakers, or further therapy.

We observed no periprocedural deaths, major strokes, or myocardial infarctions, nor were there any cases of hyperperfusion syndrome within 30 days. One patient developed a new neurological impairment after the first procedure, and the second procedure was delayed in a staged manner. Diffusion-weighted MRI of the patient revealed a new cortical embolic lesion in the ipsilateral hemisphere of the treated side. Two patients developed TIA in the immediate postprocedural period, but recovered completely. TIA occurred in the symptomatic side that had been treated in the first procedure in one patient, and in another patient TIA occurred in the asymptomatic side, which had been treated in the second procedure. The diffusion-weighted MRI of the patients was negative. Neuroprotection devices were used for a unilateral CAS patient with a minor stroke and for both carotid arteries in the two patients with TIA.

Technical results and complications are summarized in table 3.

Table 1 Clinical and Angiographic Characteristics of the Patients.

Population study	27 patients
Age (years)	69.2 ± 8.6 (52-84)
Age >80 years	6 (22%)
Male	19 (70%)
Hypertension	18 (67%)
Coronary disease	8 (30%)
Angiographic evaluation	54 lesions
Severity of lesion	78.3 ± 10.3 %
Lesion length	15.1 ± 4.9 mm
Ulcerated lesion	26 (48%)
Calcified lesion	44 (81%)

Discussion

Although we do not know accurately the incidence of bilateral carotid stenosis which is eligible for bilateral CAS, hemodynamically significant bilateral carotid stenosis is not infrequent in clinical practice. According to Henry et Al.²³, 115 procedures in their series of 701 CAS procedures were bilateral CAS. Generally, a majority of patients exhibiting severe bilateral carotid stenosis undergo two separate procedures of unilateral CAS, despite the disadvantages associated with such an approach, including the delay of definitive treatment for the carotid stenosis, the higher costs, and the considerable inconvenience to the patient due to prolonged hospital admission or separate hospitalizations^{19,22,23}. The principal concerns regarding simultaneous bilateral CAS are related to the risk of severe hypotension resulting from carotid sinus baroreflex stimulation, as well as the risk of cerebral hyperperfusion^{14-17,24}. However, recent reports of simultaneous bilateral CAS have clearly indicated both the safety and the efficacy of this procedure¹⁸⁻²³. Chen et Al.²² reported the reliable performance of simultaneous bilateral CAS in ten high-risk patients, with no procedural deaths, myocardial infarctions, or strokes. More recently, Henry et Al.²³ reported the safety of simultaneous bilateral CAS in 17 patients, in which one patient experienced a hyperperfusion cerebral hemorrhage that resulted in the patient's death, and one patient experienced a myocardial infarction, which also resulted in death. In the work presented here, we also report no periprocedural deaths, major strokes, or myocardial infarctions, nor did we witness any hyperperfusion syndrome in our patients.

With regard to cerebral hyperperfusion, we observed no cases of hyperperfusion in our study group. In addition, we found only one case of hyperperfusion syndrome with simultaneous bilateral CAS in our literature review, which encompassed five original reports¹⁹⁻²³. Thus, the incidence of hyperperfusion syndrome for simultaneous bilateral CAS was quite low as compared to that reported in large-series unilateral CAS studies - 3.8% in the report of Morrish²⁷ et Al. and 5% in the report of Meyers et Al.¹⁷. The results of simultaneous bilateral CAS take on even more importance when we consider that these were all patients at extremely high risk of hyperperfusion

Table 2 **Procedural Data.**

(*48 arteries underwent CAS)	
Pre-dilatation before neuroprotection	10 arteries (21%).
Pre-dilatation after neuroprotection	34 arteries (71%).
Post-dilatation after stent deployment	26 arteries (54%)
Protection Device	38** arteries (79%)
NeuroShield	17 arteries (45%)
Filter Wire Ex	15 arteries (39%)
Percutaneous GuardWire	6 arteries (16%)
*Six patients who had any of unfavorable signs after the first CAS underwent unilateral CAS. **Neuroprotection devices were utilized for bilateral carotid artery stenting in 11 patients, and for unilateral carotid artery stenting in 16 patients.	

syndrome, much higher than is typically associated with the subjects of unilateral CAS studies. Abou-Chebl et Al.²⁸ reported five cases (1.1%) of hyperperfusion syndrome out of 450 cases of unilateral CAS. In that study, it was al-

Table 3 **Technical Results and Complications (n= 27).**

Immediate residual stenosis after the procedure	8.1 ± 5.0%
Mean procedure time	3 hour 18 minutes*
Periprocedural Complications	(within 30 days)
Bradycardia and/or Hypotension	
Transient	17 patients (63%)
Persistent	3 patients (11%)
Neurological complications	
Transient ischemic attack	2 patients (7%)
Stroke, Hyperperfusion syndrome	1 patient (4%)
Myocardial infarction, Death	none
* For bilateral carotid stenting	

so suggested that patients suffering from severe stenosis of the contralateral carotid artery or occlusion might be somewhat predisposed to hyperperfusion syndrome, particularly in cases in which concurrent arterial hypertension also exists. However, we could not conclude that simultaneous bilateral CAS is superior to staged bilateral CAS for treatment of hemodynamically significant bilateral carotid stenosis, with regard to the safety of hyperperfusion syndrome. This is because there have been no randomized large prospective studies conducted to directly compare simultaneous bilateral CAS with staged bilateral CAS. Although the results of the work presented showed favorable outcomes, simultaneous bilateral CAS was completed only in selected patients without randomization, and even during the procedure several patients with unfavorable signs after the first procedure were excluded from the bilateral CAS population. However, our report and previous reports indicated that the completion of bilateral CAS in single setting simply does not increase the risks of hyperperfusion syndrome on its own, contrary to general concerns.

With regard to hemodynamic instability, we observed three patients (11%) who experienced persistent hypotension for up to 1 week after the procedure. The frequency with which we encountered persistent hemodynamic instability was slightly higher than what was reported in other studies of simultaneous bilateral CAS. Henry et al²³ reported that 44% of their patients exhibited transient bradycardia and/or hypotension, but that all of these patients promptly recovered, without requiring vasopressors or pacemakers. Chen et al²² reported three patients (30%) in whom vasopressors were required, due to persistent hypotension. However, two of these episodes were attributed to hemorrhages that generated hypovolemic hypotension. Therefore, the actual incidence of persistent hemodynamic instability due to carotid baroreflex stimulation was determined to be 10%, in their report. The reason as to the relatively high incidence of persistent hypotension in our study is not completely clear. However, when compared to the results of the unilateral CAS study conducted by Dangas et al²⁹ or the results of the study of Leisch et al²⁶, our study, as well as previous studies of simultaneous bilateral CAS, generated fairly favorable results with regard to hemodynamic

instability. Also, we determined that the incidence of periprocedural or postprocedural complications did not increase as the result of hemodynamic instability in either simultaneous bilateral CAS, or unilateral CAS^{22,23,26,27}.

Concerns also existed with regard to the use of neuroprotection devices during simultaneous bilateral CAS²⁴. The goal of our study was to evaluate not the value of neuroprotection devices, but the feasibility of simultaneous bilateral CAS with the use of them. The studies with data from several large trials, series, and registries provided considerable evidence that the use of an embolic protection device reduces the incidence of cerebral embolic events during CAS, in both symptomatic and asymptomatic patients⁶⁻¹². However, the benefits of routine use of cerebral protection devices have yet to be confirmed by level one evidence¹³, and should still be established in more randomized prospective studies. Although it appears that neuroprotection devices do, indeed, reduce the rate of periprocedural strokes, they also add substantial time and cost to the procedure. In addition, they may cause unwanted device-related complications, such as vasospasm or dissection. As the technology of neuroprotection device manufacturing has progressed, and as the experience of practitioners has increased, the actual risk of potential problems associated with the protection devices has been substantially attenuated, and additionally, procedure time has not expanded sufficiently to hinder the performance of simultaneous bilateral CAS as a general clinical procedure. In cases in which the first procedure is excessively long, and/or the patient exhibits poor cooperation, we can simply delay the second procedure for a few days. In our study, ten patients received neuroprotection combined with unilateral CAS. This was not related to an increase in procedure time, but rather to the cost of neuroprotection. As a rule, neuroprotection is not fully covered by insurance in most countries. Thus, we did not perform neuroprotection for the CAS in cases in which the lesions appeared smooth without ulcerations, or when it was assumed that a low possibility of distal emboli migration existed, according to the operator's discretion. The mean procedural time was three hours and 18 minutes in patients who underwent simultaneous bilateral CAS in our study. It certainly takes longer to perform a bilateral CAS in one session, and a certain amount of evaluation

time after completion of the first procedure is required as well. However, all procedures were completed in a reasonable time for the majority of patients to endure, and only one patient postponed the second procedure due to the long procedure time necessary for the completion of the first CAS.

When we evaluated the overall complication rate at the 30-day follow-up, no significant inferiority was demonstrated in comparison to the previous studies in either the symptomatic or asymptomatic patients, with or without protection devices⁶⁻¹². On the contrary, with regard to concerns about complications, the incidence in our study was rather low. However, the number of patients in the study is too small to place a great deal of emphasis on the superior safety of our procedure. In addition, because the cohort of simultaneous bilateral CAS didn't appear to be the same as the cohort of the staged bilateral CAS, we did not evaluate the long-term results concerning complications between them.

Although the primary objective of this study was to assess the clinical impact of simultane-

ous bilateral CAS coupled with the use of neuroprotection, this study has several limitations: in addition to the usual limitations associated with any retrospective study, we also had a small study group, and conducted no direct comparison between simultaneous CAS and staged CAS. However, this report reflects the favorable results of simultaneous bilateral CAS performed with improved equipment with neuroprotection devices, a standardized technique, and a careful selection of eligible patients. In summary, simultaneous bilateral CAS with neuroprotection can be performed in a single setting without increased concerns regarding hyperperfusion syndrome, hemodynamic instability, thrombo-embolism, or procedure time, given that the first CAS has been safely completed with no evidence of complications, in a well-managed procedure time. However, in order to more accurately determine the efficacy and safety of the methods described in the present study, more randomized prospective studies with larger sample sizes will be necessary.

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Taik-Kun Kim, M.D.
 Department of Diagnostic Radiology
 Korea University College of Medicine
 Ansan Hospital, #516 Kojan-Dong
 Ansan City, Kyungki-Do 425-020
 Korea
 E-mail: tkkim@kumc.or.kr)