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Stent Coning Induces Distal Stent Edge Stenosis

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Abstract

Purpose: Stent coning is conular morphological changes at the distal end of wire-braided closed-cell stents. We discuss its incidence, predictors, and outcomes.

Materials and Methods: We reviewed data on 178 carotid arteries (172 patients) that were treated by carotid artery stenting (CAS) with wire-braided stents. All patients were followed-up by carotid duplex ultrasound (DUS) studies to detect in-stent restenosis (ISR) and stent-edge stenosis. In patients manifesting stent coning, we also obtained neck radiographs.

Results: Stent coning was detected in 11 arteries (6.2%). Internal carotid artery/common carotid artery ratio and use of a post dilation balloon were associated with coning ($p<0.05$). On radiographs obtained at 3-months follow-up, all instances of coning had disappeared spontaneously. Follow-up DUS detected 5 instance of stent-edge stenosis at 3- or 6 months. Stent-edge stenosis occurred only in arteries with coning. At 6 months post-CAS, Kaplan-Meier analysis revealed a cumulative freedom from stent-edge stenosis of 54.5% in vessels with coning; stent-edge stenosis was not observed in arteries without coning ($p<0.05$). At DUS follow-up performed a mean of 32.8 months post-CAS, 2 arteries without coning manifested ISR >50% ($p=0.72$). At clinical follow-up carried out a mean of 38.4 months after the procedure, none of our patients had developed new neurologic ischemic symptoms.

Conclusions: While stent coning is self-curing, it may be associated with the late development of stent-edge stenosis.

Keywords: Carotid artery stenting; Coning; Stenosis; Stent; Stent edge

Introduction

Self-expanding stents are now used routinely in carotid artery stenting (CAS) because of their superior conformability and resistance to deformation during neck movement or compression. In Japan, two types of carotid self-expanding stents, closed-cell stents (Wallstent; Boston Scientific Corp., Natick, MA) and open-cell stents (Precise; Cordis Endovascular, Miami, FL) are commercially available. The Wallstent, a wire-braided stent has been tested in clinical trials [1-5]; among stents it is the most flexible in the unemployed state and it has an intrinsically greater potential to scaffold and support fractured plaques and debris during CAS than open-cell stents [6-9].

Coning is the conular morphological change at the distal end of the deployed stent. Although it is often encountered with wire-braided stents, the clinical effects of coning have not been examined in detail. Here we document Wallstent coning in the ICA of patients treated by CAS and discuss its incidence, predictors, and outcomes.

Materials and Methods

Patient population

We retrospectively reviewed the records of 172 patients (178 arteries) who underwent elective CAS with Wallstent between January 2006 and December 2010 a tour institute. Indications for intervention were >50% stenosis in symptomatic- and >80% stenosis in asymptomatic patients. Exclusion criteria were occlusion of the ipsilateral common carotid or internal carotid artery, stenosis due to external compression, stenosis due to dissection, recurrent stenosis after CAS, and floating thrombus. The degree of stenosis was determined angiographically using NASCET measurement criteria [10]. All patients provided prior written informed consent. The study protocol was approved by the ethics committee of our hospital.

All patients received cilostazol (200 mg per day) or clopidogrel (75 mg per day) before intervention. Aspirin (100 mg per day) was administered concomitantly. A baseline angiogram and an angiogram obtained immediately after CAS were available in all patients. CAS was performed in standard fashion; it included placement of a protection device, pre dilation angioplasty, placement of a Wallstent, and post dilation angioplasty if necessary. We selected stents whose diameter was 1 to 2 mm larger than the diameter of the ipsilateral common carotid artery (CCA); they were deployed to cover the entire lesion from the distal internal carotid artery (ICA) to the CCA. Post dilation was with a 6- or 7 x 20 mm Sterling balloon catheter (Boston Scientific); the inflation pressure was 6atm for 10 sec. In cases with insufficient stent expansion the balloon was additionally inflated up to 10atm. At the end of the procedure, an intravascular ultrasound scan was performed to confirm complete coverage of the lesion by the stent (normal-to-normal from the distal ICA to the CCA). Immediately after CAS, the lesions were evaluated angiographically in the anteroposterior and lateral directions to evaluate residual stenosis and stent coning. The patients were transferred to the neurointensive care unit for overnight observation.

All stenotic lesions were accessed via the transfemoral approach.

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All CAS procedures were successful (technical success rate 100%). The mean degree of stenosis treated was 81.5 ± 8.4%. Table 1 shows the baseline clinical, angiographic and procedural variables derived from the medical records of our patients.

Data collection

We recorded clinical, radiological and procedural data to identify those that may play a role in coning. The clinical variables included the patient age and sex, the presence or absence of symptoms, risk factors, and comorbidities (hypertension, diabetes, hypercholesterolemia, coronary artery disease, and smoking). We compared baseline angiograms and angiograms obtained immediately after CAS to assess radiological differences in the grade and length of the stenosis, the diameter of the ICA and CCA, and the presence of calcification. Procedural variables were the application of balloon post dilation, the diameter of the stent, and the degree (%) of residual post-CAS stenosis. All medical progress notes taken during hospitalization and at follow-up were reviewed for neurologic or cardiac events and death. All neurologic and cardiac examinations were made by independent expert neurologists and cardiologists.

Follow-up protocol and criteria for restenosis

All patients were followed at our outpatient clinic 30 days- and, 3-, 6-, 9-, and 12 months after the procedure and every 6 months thereafter. During these routine post procedural visits, an independent neurologist examined each patient; carotid duplex ultrasound scans (DUS) were acquired at 3 and 6 months post-CAS and at 6-month intervals thereafter to detect in-stent restenosis (ISR) and distal stent-edge stenosis. The velocity criteria we used to evaluate carotid artery stenosis were modifications of the Japanese Academy of Neurosonology Guidelines for Neurosonology and were validated at our hospital. A peak systolic velocity >150 cm/s correlated with >50% stenosis [11]. Luminal reductions on grayscale images and color flow disturbances were evaluated. In patients that distal stent edge of the stent could not be evaluated by DUS due to limited sonographic window, we used the transoral carotid ultrasonography method [11]. ISR and stent-edge stenosis >50% identified on DUS were confirmed on carotid angiographs, and stenoses were measured geometrically on the basis of NASCET criteria [10]. When >50% ISR or stent-edge stenosis was recognized on angiographs, carotid angioplasty and stenting were considered. In patients demonstrating stent coning, plain neck radiographs were acquired at 3 and 6 months post-CAS and at 6-month intervals thereafter to examine morphological stent changes.

Clinical variables	Stent coning(+) (n=11)	Stent coning(-) (n=167)	*p*
Male sex, n (%)	11 (100)	151 (90.4)	0.28
Age, y (mean ± SD)	72.3 ± 6.1	74.1 ± 8.2	0.51
Symptomatic lesion	8 (72.7)	132 (79.0)	0.62
Hypertension, n (%)	9 (81.8)	142 (85.0)	0.77
Diabetes mellitus, n (%)	3 (27.3)	72 (43.1)	0.30
Hypercholesterolemia, n (%)	4 (36.4)	53 (31.7)	0.75
Coronary artery disease, n (%)	3 (27.3)	58 (34.7)	0.61
Smoking, n (%)	2 (18.2)	48 (28.7)	0.45
Radiological variables			
Carotid stenosis (%, mean ± SD)	83.2 ± 8.1	79.8 ± 8.6	0.09
Length of stenosis (mm, mean ± SD)	12.6 ± 5.7	13.8 ± 6.9	0.52
ICA diameter (mm, mean ± SD)	4.7 ± 2.1	4.8 ± 3.4	0.72
ICA/CCA ratio (%,mean ± SD)	0.47 ± 0.3	0.67 ± 0.6	<0.05
Calcification, n (%)	4 (36.4)	48 (28.7)	0.59
Procedural variables			
Stent diameter (mm, mean ± SD)	10.0 ± 0.0	9.6 ± 0.5	<0.05
Postdilation, n (%)	11 (100)	122 (73.1)	<0.05
Residual stenosis, (%, mean ± SD)	1.7 ± 4.8	4.4 ± 5.8	0.22
ICA/stent ratio (%, mean ± SD)	0.48 ± 0.2	0.62 ± 0.3	0.18

*SD = standard deviation

Table 1: Comparison of the baseline clinical, angiographic and procedural variables in 172 patients manifesting 11 lesions with- and 167 lesions without stent coning.

Definitions

Coning was recorded if there was evidence of conular morphological changes at the distal end (distal tapering) of the stent on angiograms obtained immediately post-CAS (Figure 1A-1C). ISR was defined as recurrent stenosis >50% within stented segment on DUS images, stent-edge stenosis as stenosis >50% at the distal stent edge not associated with ISR. The diameter of the ICA and the CCA were recorded as its retrospectively measured diameter on preprocedural angiograms at the site of the distal stent edge and the proximal stent edge, respectively, referred to post procedural angiograms. Calcification was recorded when it involved >50% of the artery circumference. Neurologic events were categorized as TIA (neurologic deficit lasting <24 hr), minor stroke (neurologic deficit lasting >24 hr with a NIHSS <4), and major stroke (neurologic deficit lasting >24 hr, NIHSS >4). Cardiac morbidity was defined as myocardial infarction on the basis of cardiac enzymes, electrocardiography, or clinical evidence of congestive heart failure. Cardiac enzymes and electrocardiograms were checked immediately after- and on the morning after the procedure.

Statistical analysis

The time to stent-edge stenosis was recorded using the Kaplan-Meier analysis method. Continuous variables were expressed as the mean ± 1 standard deviation. Categorical variables were expressed in terms of percentages. For univariate comparisons of continuous data we applied the Mann-Whitney *U*-test. Categorical data were compared with the Yates χ^2 test. All p values were two-sided; $p<0.05$ was considered statistically significant. Calculations were with statistical software (SPSS, version 10.0, for Microsoft Windows; SPSS, Chicago, III).

Results

CAS outcomes at 30 days and incidence of coning was recognized in 11 of the 178 lesions (6.2%). There were no statistically significant differences in the clinical variables of lesions with- and without stent coning. Mean residual stenosis was 3.1 ± 5.3% (1.7 ± 4.8% in lesions with- and 4.4 ± 5.8% in lesions without coning, p=0.22). The overall 30-day stroke rate was 1.7% (n=3). Among the 11 lesions with stent coning, one (9.1%) produced TIA. No patients with stent coning suffered minor- or major strokes. Of the lesion without stent coning (n=167), one each (0.6%) produced TIA or induced a minor stroke. No patients without stent coning suffered a major stroke. None of the172 patients experienced myocardial infarction or died.The30-day incidence of post-CAS stroke was not significantly different between lesions with- and without coning (9.1% in lesions with- and 1.2% in lesions without coning, p=0.45).

Risk factors for coning

Univariate analysis showed that variables significantly associated with the development of stent coning were the ICA/CCA ratio and use of a post dilation balloon ($p < 0.05$) (Table 1).

Follow-up study

Follow-up DUS studies performed for mean of 32.8 ± 12.3 months (range 12-72 months), confirmed stent-edge stenosis in 3lesions at 3- and in 2 lesions at 6 months. Mean stent-edge stenosis and mean peak systolic velocity were 50.2 ± 3.8% and 185.4 ± 68.1 cm/s, respectively. All 5 lesions with stent-edge stenosis were asymptomatic and all instance of stent-edge stenosis occurred in lesions with stent coning. By Kaplan-Meier analysis the cumulative freedom from stent-edge stenosis at 3- and 6 months post-CAS was72.7% and 54.5%, respectively, for lesions with- and 100% for lesions without coning ($p<0.05$). Figure 2 shows the Kaplan-Meier estimated curves of patients with stent-edge stenosis>50% in lesions that did- and did not manifest coning. Only one of the 11 lesions (median 48.5%, range 41% - 52%) with coning manifested angiographic evidence of stent-edge stenosis>50% (Figure 1D and 1E). All 5 lesions with stent-edge stenosis were treated conservatively with dual anti platelet therapy and were monitored by serial clinical evaluations and DUS studies. On plain neck radiographs obtained 3 months post-CAS, all instances of stent coning had disappeared.

ISR was detected in 2 of the 178 lesions (1.1%) on DUS images and confirmed by angiography (61.0% and 51.8%, respectively); both lesions were free of stent coning and asymptomatic. There was no statistically significant difference with respect to ISR between lesions with- and without coning ($p=0.72$). One lesion with ISR underwent endovascular re intervention (balloon angioplasty), and the other was treated conservatively with dual anti platelet therapy and monitored. All lesions with stent-edge stenosis or ISR who were treated conservatively remained stable without stenotic progression in the course of follow-up mean 32.8 months. The patient requiring balloon angioplasty has remained free of recurrence during 21.4-months follow-up.

None of our 172 patient developed new neurologic ischemic symptoms in the course of 18 – 78-month follow-up (mean 38.4 ± 13.2 months).

Discussion

Although the natural course of stent coning remains unknown, our study demonstrated that it is associated with the development of stent-edge stenosis. Coning-induced stent-edge stenosis was mild and did

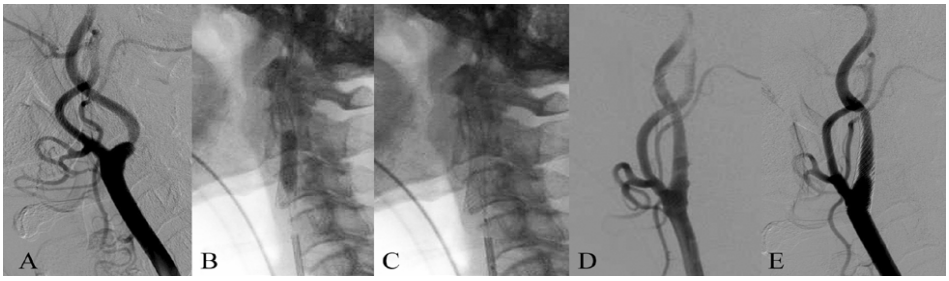

Figure 1: Preoperative right carotid angiogram (A) demonstrating severe carotid stenosis. Intra procedural plain radiograph obtained at post dilation balloon inflation (B) and radiograph at the end of the procedure (C) showing stent coning. Right carotid angiogram obtained immediately after CAS (D) demonstrating excellent dilation of the stenosis. Right carotid angiogram obtained at 3-month follow-up (E) shows stent edge stenosis.

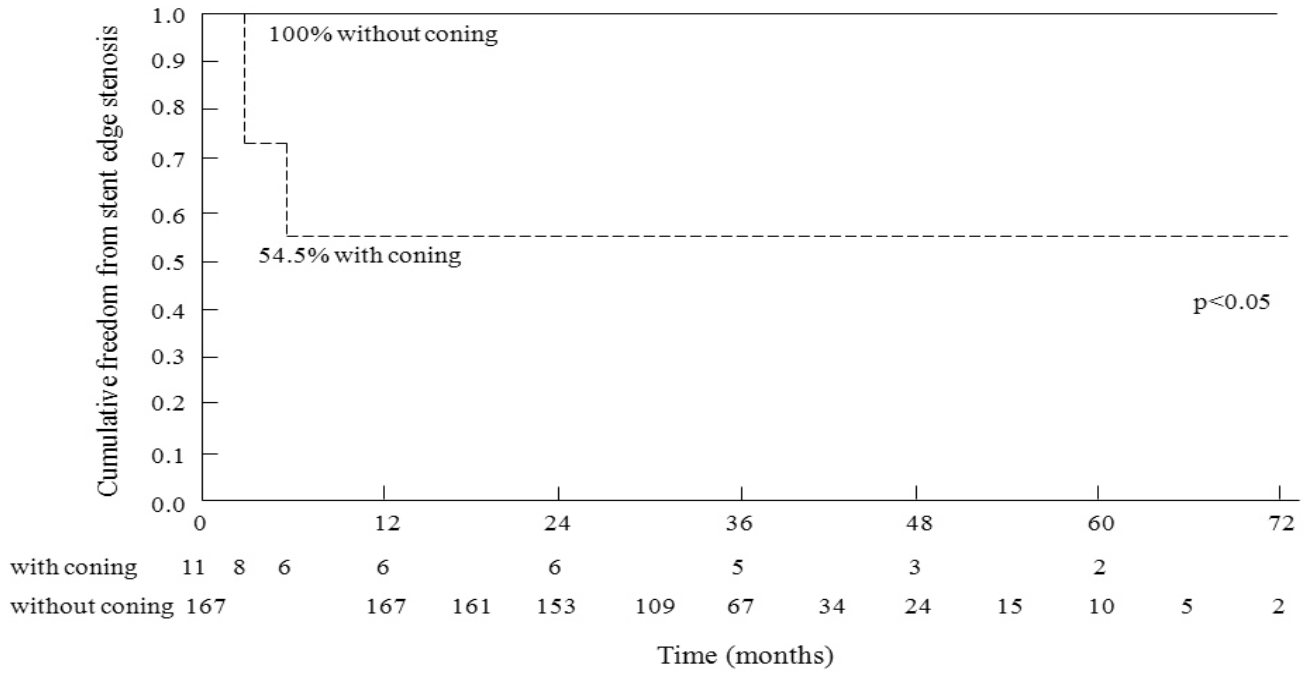

Figure 2: Kaplan-Meier analysis comparing the cumulative freedom from stent edge stenosis in lesions who did (dashed line) and did not (solid line) manifest coning.

not affect the clinical outcome. However, coning may result in severe stent-edge stenosis and elicit ischemic events.

In our study, the ICA/CCA ratio and use of a post dilation balloon was the predictor of coning. We used Wallstents that consist of a single cobalt alloy wire woven into a tubular structure; the single wire continues along the entire length of the stent [12]. The cells of these stents are affected by adjacent cells because unlike open-cell stents that feature a small number of connecting bridges [6], they are not independent. Wire-braided stent deployed in the vessel with discrepancy of the diameter between the ICA and the CCA may tend to have larger size of cells at the CCA and smaller size at the ICA. Under this condition, when the cells of in some part of the Wallstent expand, adjacent cells are compressed. Such cell compression at the distal edge of the Wallstent may result in coning.

Wallstents tend to stretch tortuous carotid artery resulting in acute angulation (kinking) of the stent-deployed carotid artery at the distal edge [12,13]. This stent-induced kinking may develop the stent-edge stenosis, because the stent distal edge dent into the vessel wall and cause the intimal injury. However, different mechanisms may exist in the development of stent-edge stenosis in the stent coning, as the stent is not adapted to the vessel wall at the distal edge in the vessel with the coning. In the presence of coning at the stented ICA, the distal end of the stent is under-expanded and not in contact with the vessel wall. Stent under-expansion and low or reversed wall shear stress have been found to be associated with a higher risk for restenosis [14-16]. Also, clots may form at coning site due to blood flow stasis in the space between the stent and the vessel wall. Clots outside the stent result in decreased blood flow velocity at the stenotic lesion and this may induce restenosis [17,18]. Delayed arterial healing with incomplete endothelialization and the persistence of fibrin have been observed at the site of stent malpositioning [19]. Coning also results in an increase in the metal-to-tissue ratio, and this may lead to neointimal hyperplasia [15,20].Thus, many factors may contribute to the development of stent-edge stenosis in the presence of coning (Figure 3).

As we deployed Wallstents to cover the entire lesion (normal-to-normal from the distal ICA to the CCA), the instances of coning-induced stent-edge stenosis we observed may reflect a de novo lesion not associated with intimal hyperplasia related to the plaques of the stenotic lesion or their protrusion.

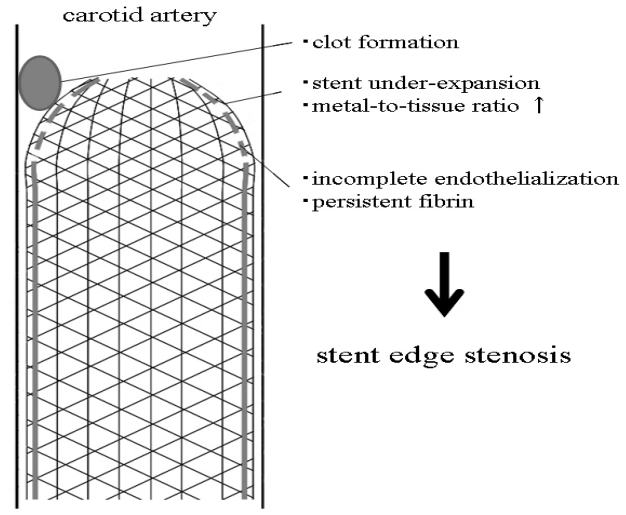

Figure 3: Schematic drawing of coning and possible mechanism of stent edge stenosis.

In ours study, all coning disappear by 3 months post-CAS and stent-edge stenosis was observed at 3- and 6 months after CAS. Although self-expanding stents may facilitate the self-cure of coning [2,21], because it may result in the late development of stent-edge stenosis, coning must be ruled out during the final stage of CAS.

Conclusion

Although stent coning is a self-curing lesion, it is associated with the late development of stent-edge stenosis.

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