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Post-Stent Ballooning during TransCarotid Artery Revascularization

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Abstract

Background: Post-stent ballooning/angioplasty (post-SB) have been shown to increase the risk of stroke risk after Transfemoral CAS. With the advancement of TransCarotid Artery Revascularization (TCAR) with dynamic cerebral blood flow reversal, we aimed to study the impact of post-SB during TCAR.

Methods: Patients undergoing TCAR in the Vascular Quality Initiative between September 2016 and May 2019 were included and were divided into 3 groups: those who received pre-stent deployment angioplasty only (pre-SB, reference group), those who received post-stent deployment ballooning only (post-SB), and those who received both pre-stent and post-stent deployment ballooning (prepost-SB). Patients who did not receive any angioplasty during their procedure (n=367, 6.7%) were excluded as these represent a different group of patients with less complex lesions than those requiring angioplasty. Primary outcome was in-hospital stroke/death. Analysis was performed using univariable and multivariable logistic regression models.

Results: Out of 5,161 patients undergoing TCAR, 34.7% had pre-SB only, 25% had post-SB only and 40.3% had both (prepost-SB). No differences in the rates of in-hospital and 30-day stroke, death and stroke/death were observed among the three groups; in-hospital stroke/death in the pre-SB group was 1.4% (n=25), post-SB: 1.2% (n=16), and prepost-SB: 1.4% (n=29), p=0.92. However, patients undergoing post-SB and prepost-SB had higher rates of in-hospital transient ischemic attacks (TIA) (post-SB:0.9%, prepost-SB: 1% vs. pre-SB: 0.2%, p<0.01) and post-procedural hypotension (16.6% and 16.8% vs. 13.1%, respectively; p<0.001). Post-stent ballooning also had longer operative times, as well as flow reversal and fluoroscopy times. On

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multivariable analysis, no association was seen between post-stent ballooning and the primary outcome of in-hospital stroke/death [OR (95%CI): post-SB: 0.88 (0.44, 1.73), prepost-SB: 0.98 (0.57, 1.70)]. Similarly, no significant differences were noted in terms of post-procedural hemodynamic instability and 30-day outcomes. However, post-SB and prepost-SB were associated with four times the odds of in-hospital TIA compared to pre-SB alone [OR (95%CI): post-SB: 4.24 (1.51, 11.8); prepost-SB: 4.76 (1.53, 14.79), p=.01]. Symptomatic patients had higher rates of in-hospital stroke/death compared to their asymptomatic counterparts, however, there was no significant interaction between symptomatic status and ballooning in predicting the primary outcome.

Conclusion: Post-stent ballooning was used in 65.3% of TCAR patients. This maneuver seems to be safe without an increase in the odds of post-operative in-hospital stroke/death. However, the increased rates of TIA associated with post-SB requires further investigation.

Table of Contents Summary:

This VQI analysis demonstrated favorable outcomes with the use post-stent ballooning during TCAR. However, the increased rates of TIA with post-SB mandates its use in cases with severe residual stenosis.

Keywords

post-stent ballooning; angioplasty; transCarotid artery revascularization; TCAR; flow reversal

Introduction:

Carotid artery stenting (CAS) is a minimally invasive alternative to carotid endarterectomy (CEA) in patients who are at a high-surgical risk. Nonetheless, despite years of improvement, the increased risk of stroke after CAS in the peri-operative period was one of the main reasons that limited the use of CAS as the gold standard procedure for the treatment of carotid artery stenosis and affected reimbursement for this procedure. [1] The increased stroke rates observed with CAS can be attributed to certain technical factors such as the need to cross the arch and the lesion before deployment of a protection device, in addition to the possibility of malalignment of the distal protection device[2]. In an aim to optimize its outcomes, much variability exists in CAS technique today. One common variation in technique among providers is the timing of percutaneous transluminal angioplasty (PTA).

PTA is a key step during CAS that is frequently performed, after establishing distal or proximal protection. Pre-stent ballooning (Pre-SB) is commonly used to prepare a stenosed vessel for stent placement. This is achieved via slow and gradual balloon inflations to low atmosphere pressure followed by an immediate gradual deflation to prevent negative pressure formation and minimize showering of emboli to the brain[3]. While the CAPTURE study showed an increase in the risk of peri-operative stroke in patients with pre-SB versus without pre-SB (Odds Ratio: 3.68, 95%CI: 2.26–6.0, P<.001)[2], a study from the Vascular Quality Initiative (VQI) database showed a similar risk of stroke and death after primary

CAS without angioplasty compared with conventional CAS with angioplasty, as long as an embolic protection device is used[4].

On the other hand, post-stent ballooning (post-SB) may be more operator-dependent and is used to closely appose the stent and intima and to mitigate any residual stenosis after stent deployment by visually creating a greater luminal diameter[3,5]. Post-SB is associated with a dramatic increase in the risk of intra- and post-operative hemodynamic depression, as well as stroke or death after transfemoral CAS (TFCAS), which might be secondary to fracturing of the atheromatous plaque and the liberation of a large amount of particulate debris[3,5].

TransCarotid artery revascularization (TCAR) is a novel procedure designed to overcome the limitations of TFCAS[7–9]. TCAR obviates the need to cross and manipulate the aortic arch via direct access of the common carotid artery. Moreover, the high-rate temporary and dynamic cerebral blood flow reversal applied during TCAR carries embolic debris, released during or immediately after PTA and stent placement, distally from the cerebral circulation[9–11]. With the promising initial outcomes of this procedure[7–13], we aim to assess whether post-SB would affect the outcomes of TCAR.

METHODS

We performed a retrospective analysis of the Society for Vascular Surgery (SVS) Vascular Quality Initiative (VQI) CAS registry. Patients undergoing TCAR between September 2016 and May 2019 were included. Patients who did not receive any angioplasty during their procedure (n=367, 6.7%) were excluded as these represent a different group of patients (primary stenting) with less severe lesions compared to the ones requiring angioplasty. Moreover, they might represent a different technique that is practiced by few interventionalists and surgeons. Carotid stents placed in conjunction with planned intracranial procedures and patients with unknown presenting symptom status were also excluded.

Patients were then divided into three groups (Figure 1)

1. Patients receiving pre-stent deployment angioplasty only (pre-SB, reference group)
2. Patients receiving post-stent deployment angioplasty only (post-SB), and
3. Patients undergoing both pre-stent and post-stent deployment angioplasty (prepost-SB)

The VQI Patient Safety Organization research committee approved this study. Only de-identified information from participating institutions in the VQI was used for this analysis, thus the need for individual informed consent is waived and the study is exempt from Institutional Review Board approval.

Outcomes:

The primary outcome was in-hospital stroke and death after TCAR. Secondary outcomes of interest included post-procedural hypotension or hypertension requiring intravenous (IV)

medications, in-hospital transient ischemia attack (TIA), or reperfusion syndrome, in addition to operative, flow reversal and fluoroscopy times, and hospital length of stay. Stroke was defined as permanent neurologic symptoms that could include, full or partial visual loss, motor/sensory loss, speech abnormality, other new neurologic symptoms related to the right or left hemisphere, or symptom that are bilateral motor, sensory, or visual loss, diplopia, ataxia. TIA was defined as any focal neurologic deficit that resolved within twenty-four hours. Reperfusion syndrome was defined as any post-operative seizure associated with a headache or hemorrhage on brain imaging.

Statistical Analysis

Categorical baseline characteristics across the three groups were compared using Pearson χ^2 test or Fisher exact test; continuous variables were compared using Analysis of Variance (ANOVA). Multivariable logistic regression analysis was used to model the relationship between balloon/angioplasty timing and the outcome of interest. A total of twenty-six covariates were included in the original model. These include age, sex, race, ethnicity, symptomatic status, body mass index (BMI), hypertension, diabetes, coronary artery disease (CAD), congestive heart failure (CHF), prior coronary intervention, chronic obstructive pulmonary disease (COPD), chronic kidney disease (CKD), hemodialysis, smoking status, degree of stenosis, prior ipsilateral carotid intervention, elective procedure, use of general anesthesia and preoperative medications, including aspirin, P2Y12 receptor antagonists, anticoagulants, beta blockers, angiotensin converting enzyme (ACE) inhibitors, and statins. All appropriate theory-based categorical-categorical interactions were tested for and those that were found significant were presented. The variables included in the final models were age, race, symptomatic status, comorbidities (diabetes, CAD, CKD, COPD), preoperative smoking, prior ipsilateral CEA or CAS, prior contralateral carotid revascularization, anesthesia type, and preoperative use of beta-blockers and anticoagulants. A separate analysis was conducted stratifying stroke, death, and stroke/death by baseline symptoms among the three group. All calculations were completed using STATA 15.1 (StataCorp LP, College Station, Tex). A p-value <.05 was considered statistically significant.

RESULTS

A total of 5,161 patients (36.6% females) underwent TCAR between 2016 and 2019. Among all patients, 34.7% (n=1,791) underwent pre-SB, 25.0% (n=1,290) underwent post-SB, and 40.3% (n=2,080) underwent prepost-SB.

When compared to pre-SB patients, post-SB and prepost-SB patients were more likely to be older (age ≥ 75 years, pre-SB: 46.9% vs. post-SB:49.5% and prepost-SB: 51.5%, p=.04), and less likely to be symptomatic (pre-SB:34% vs. post-SB: 28.1% and prepost-SB:28.1%, p<.001). They also had more cardiac comorbidities including a history of CAD (49.2% vs. 53.3% and 53.7%, p=.01) or CHF (17.0% versus 18.8% and 20.1%, p=.04). They were more likely to undergo the procedure under general anesthesia (79.3% versus 86.1% and 82.0%, p<.001) and to receive preoperative β blockers (56.2% vs. 56.4% vs. 59.8%, p=.04) (Table I). The mean balloon diameter used for post-stenting was 5 +/− 0.9 mm (range, 2–30 mm).

Univariable analysis was used to determine differences in outcomes. Thirty-day follow-up was available for 26.7% of the cohort (n=1,379). No significant differences were observed among the three groups in terms of in-hospital stroke and death (pre-SB:1.4%, post-SB:1.2%, prepost-SB:1.4%, p=.92). Similarly, in-hospital MI, reperfusion syndrome and the 30-day outcomes of stroke, death and MI in patients with available 30-day data were not significantly different between the 3 groups (Table II). However, compared to patients in the pre-SB group, post-SB and prepost-SB patients had higher rates of in-hospital TIA (0.2% versus 0.9% and 1.0%, p<.01), and post-procedural hypotension (13.1% versus 16.6% and 16.8%, p<.01). They also had longer operative times (69.5 versus 72.3 and 74.28 minutes, p<.001), flow reversal times (9.7 versus 11.0 and 12.1minutes, p<.001) and fluoroscopy times (5.2 versus 5.7 and 6.5, p<.001) compared to patients receiving pre-SB (Table II)

After adjusting for potential confounders (age, race, symptomatic status, comorbidities such as diabetes, CAD, CKD, COPD, smoking history, prior CEA or CAS, anesthesia, and preoperative beta-blockers and anticoagulants), no significant association was noted between post-SB or prepost-SB and in-hospital stroke/death after TCAR [OR(95% CI): post-SB, 0.88 (0.44–1.73), p=71; prepost-SB, 0.98 (0.57–1.70), p=.96] or post-procedural hypotension [OR(95% CI): post-SB, 1.26 (0.84–1.91), p=27; prepost-SB, 1.28 (0.99, 1.67), p=06] (Table III). However, patients in the post-SB and prepost-SB group had 4.2- and 4.8-times higher odds of in-hospital TIA compared with patients receiving pre-SB only [OR (95% CI): pre-SB, 4.2 (1.5–11.8), p=.01; prepost-SB, 4.8 (1.5–14.8), p=.01, respectively].

A subset analysis stratifying patient by symptomatic status is shown in Supplementary Table I (Crude outcomes) and Table IV (Adjusted outcomes). No significant interaction was found between post-stent ballooning and symptomatic status in predicting the primary and secondary outcomes (Table IV). In addition, the type of presenting symptoms (amaurosis fugax, TIA or stroke) didn’t affect the relationship between post-stent ballooning and in-hospital outcomes.

DISCUSSION

The present study shows no significant association between post-SB (alone or with pre-SB) and the risk of stroke and stroke/death after TCAR. The rates of in-hospital stroke in patients undergoing TCAR with pre-SB only were comparable to those receiving post-SB as well as those undergoing both pre- ands post-SB (1.1% vs. 1% vs. 1.3%, P=.77). Moreover, while symptomatic patients had slightly higher rates of stroke compared to their asymptomatic counterparts, the difference in stroke rates between post-SB and pre-SB in this subset of patients was not statistically significant. These findings might be attributable to the neuroprotective effects of the dynamisc flow reversal which carries embolic debris away from the cerebral circulation, in addition to direct access of the common carotid artery which avoids the need to pass wires and catheters through the aortic arch [7–13].

On the other hand, the increased risk of TIA observed in patients receiving post-SB might be attributable to smaller debris (microparticles) that could escape the flow reversal or that were released through the stent struts after discontinuation of flow reversal. In prospective study of patients undergoing CAS with the ENROUTE System, 10 out of 56 patients (17.9%) who

underwent diffusion-weighted magnetic resonance imaging (DW-MRI) were found to have new wh1ite matter lesions post-operatively [12] On the other hand, a systematic review of carotid revascularization studies by Schnaudigel et al involving 1363 CAS and 754 CEA procedures showed evidence of new brain lesions on DW-MRI in 37% and 10% (P < .01), respectively[14]. This suggests that flow reversal, although very effective in limiting distal embolization compared to other carotid revascularization procedures, might not be perfect in capturing all emboli [12] The majority of in-hospital TIAs (56.8%) occurred 6-hours postoperatively, whereas 37.8% occurred less than 6 hours after the procedures and 5.4% occurred intraoperatively. No significant difference was observed between the 3 study groups in terms of the timing of TIAs (P=.73). Although the crude rate of TIA observed with post-SB is relatively small (1%) and might not be immediately clinically significant, there is increasing evidence that silent embolic lesions might increase the risk of long-term stroke, dementia, and death and have even been suggested as a marker for peri-procedure complications[15–19].

In TFCAS, the additional manipulation performed during ballooning, whether pre-SB or post-SB, has been associated with increased stroke risk and hemodynamic instability after [20–21]. A study from the VQI showed an independent association between post-SB and 2-fold increase in the odds of hemodynamic instability (OR: 2.13, 95%CI: 1.51–3.01, p<0.001) and 2.4-folds increase in the odds of stroke (OR: 2.37, 95%CI: 1.01–5.62, p<0.05) after TFCAS [5]. Similarly, in a prospective multicenter study by Lauricella et al, patients receiving post-SB during TFCAS had higher rates of macroscopic debris, hemodynamic instability, and microembolic signals on transcranial doppler compared with those receiving maximum pre-SB[22]. The higher stroke rate is likely the result of the balloon pushing the struts of the stent through the plaque and dislodging emboli which current distal embolic protection devices (EPD) cannot effectively capture, in addition to post-procedure embolization through the struts occurring after removal of the EPD. Moreover, sustained hemodynamic instability secondary to the pressure exerted over the carotid bulb during balloon inflation could compromise cerebral perfusion and increase the incidence of immediate periprocedural stroke[22–23]. Hemodynamic instability has been also linked to an increase in periprocedural MI, death, and length of stay.[23]

Moreover, as shown in our study, post-SB during TCAR is not associated with significant hemodynamic instability on multivariable adjustment. One hypothesis is that patients undergoing TCAR have several comorbidities such hypertension, CAD, diabetes mellitus and advanced age which are frequently associated with a dysfunctional carotid baroreflex[24]. Gupta et al found that patients with diabetes were protected from persistent hemodynamic instability, likely because of an impaired ability to develop reflex bradycardia during balloon inflation and stent deployment [25]. This is in line with other studies that demonstrated that the cardiovagal limb of the baroreflex is impaired in patients with diabetes whereas sympathetic output may be normal to slightly attenuated[26–27].

Other factors associated with reduced hemodynamic instability after stenting include a history of prior CEA which causes a reduction in the number of baroreceptors and arterial compliance due to scar formation and habitual smoking[28–29]. Almost 50% of patients in our study were ≥ 75 years of age, 91% had hypertension, 50% had CAD and 38% had diabetes.

In addition, a history of prior CEA was found in 16% of our patients whereas 22.5% were current smokers and 52% were ex-smokers (Table I). Another possible hypothesis is the maintenance of high systolic blood pressure (in the range of 140–160 mmHg) during TCAR to ensure brain perfusion. With the increased education of the high likelihood of hypotension after stenting (secondary to the radial force of the stent on the carotid baroreceptors), several providers have changed the perioperative management of patients undergoing TCAR. Besides intraoperative blood pressure management, many surgeons administer glycopyrrolate instead of or along with atropine due to its longer duration of action and less associated hemodynamic fluctuations[30–32]. Moreover, from our clinical experience, many maintain their patients on a phenylephrine drip and a portable monitor to avoid hemodynamic instability. Since no detailed information on the perioperative management of patients are available in the VQI dataset, we cannot identify the exact reason for the lack of difference in postprocedural hypotension between patients receiving post-SB and those receiving pre-SB only. However, from our clinical experience, we know that almost all surgeons follow a strict protocol with and perform TCAR time-out ensuring higher mean arterial pressure and pretreating with glycopyrrolate prior to crossing the lesion and performing PTA. [33–34]

While supporters of post-SB indicate that it decreases the incidence of in-stent restenosis by re-establishing the normal luminal diameter[35], others believe that the main goal of CAS should be plaque stabilization and treating the underlying pathophysiology rather than only resolving the stenosis and re-establishing maximal blood flow.[5,36] Currently, there is limited evidence to support improved long-term patency with post-SB[35,37–40]. A large meta-analysis on the outcomes of post-SB during TFCAS found no difference in angiographic, clinical outcomes and long-term patency rates between selective and consistent PTA use [35]. Other studies argued that, although re-stenosis rates might be higher in CAS without PTA, these patients are rarely symptomatic and re-angioplasty of recurrent lesions with neointimal formation is usually straightforward[37–40,41–42].

Given the lack of evidence supporting long term patency and the added risk of TIA, it might be better to consider PTA in select cases where the operator feels is necessary based on the degree of residual stenosis, lesion anatomy and degree of calcification [3,5,40]. Till now, there is no consensus among interventionalists about what constitutes a safe threshold to prompt postdilation as well as the impact of residual restenosis on postoperative outcomes [38–40,41,44] While a target of less than 30% residual stenosis has been recommended, many are comfortable with 50% or more residual stenosis [37,43,45]. In a study by Ogata et al, the authors state that pre-dilatation alone without post-dilatation can achieve a sufficient luminal opening to prevent restenosis, and that omission of post-dilatation and the use of a closed cell stent might be beneficial to minimize such restenosis incidents. However, in cases with >30% residual stenosis immediately after CAS, the authors suggest that post-dilatation might be considered for symptomatic patients [46]. On the other hand, a study of 412 patients undergoing TFCAS found a restenosis rate of 7.7% on long-term follow-up in patients with residual stenosis ≥ 20% with no effect on long-term clinical outcomes[37]. Interestingly, the authors demonstrated an inverse relationship between the degree of residual stenosis and the periprocedural outcome, suggesting that higher residual stenosis might contribute to decreased plaque disruption with distal embolization and hemodynamic instability[37].

Due to limited follow-up data in our study, we focused on in-hospital outcomes. However, adverse events related to stent deployment and stent angioplasty are more likely to appear in the immediate postoperative period. Moreover, we were unable to assess the impact of post-SB during TCAR on in-stent restenosis and whether its use could lead to better patency outcomes and recurrent stroke prevention. Longer studies are therefore needed to assess potential long-term outcomes of restenosis, reinterventions and recurrent stroke as well as the potential impact of the observed TIA's on neurocognitive function. No information is available on the indication for post-SB and whether it was related to residual stenosis observed on completion imaging or to the operator's preference and experience. In addition, there is a potential for residual bias due to unmeasured confounders that could affect the outcomes, such as the degree of balloon inflation (in atm pressure) and the size of balloons used for pre-stent SB. Aggressive angioplasty is more likely to result in adverse outcomes. Another limitation of this study is due to its retrospective nature which predisposes to coding errors, residual bias due to unmeasured confounders as well as selection bias. Finally, neurological outcomes were determined clinically with or without imaging confirmation or a formal neurological evaluation which might inflict some ascertainment bias. However, this would equally affect all 3 study groups, and should not change our findings. Despite these limitations, our study is the first to address the safety of post-stent ballooning during TCAR.

CONCLUSIONS

Post-SB was used in 65.3% of TCAR patients in our study. In patients with symptomatic and asymptomatic carotid artery stenosis, post-SB during TCAR seems to be safe without an increased risk of stroke/death or hemodynamic instability. These results highlight the neuroprotective benefits of flow reversal employed during TCAR. However, the slightly increased risk of in-hospital TIA observed with post-SB (1%) mandates caution and additional study.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Article Highlights

Type of Research:

Retrospective review of prospectively collected Vascular Quality Initiative data.

Key Findings:

Out of 5,161 TCAR procedures performed between September 2016 and May 2019, 34.7% utilized pre-stent ballooning only (pre-SB), 25% used post-stent ballooning only (post-SB) and 40.3% had both (prepost-SB). There was no association between the use of poststent ballooning and peri-operative stroke, death and stroke/death. However, patients undergoing post-SB and prepost-SB had slightly higher rates of in-hospital transient ischemic attacks (TIA) (post-SB:0.9%, prepost-SB: 1% vs. pre-SB: 0.2%; $p<.01$) and post-procedural hypotension (16.6% and 16.8% vs. 13.1% respectively; $p<0.001$). On multivariable analysis, post-SB and prepost-SB were associated with four times the odds of in-hospital TIA [OR (95%CI): pre-SB: 4.24 (1.51, 11.8); prepost-SB: 4.76 (1.53, 14.79), $p=0.01$] compared to pre-SB alone.

Take Home Message:

The authors suggest judicious use of post-stent ballooning with TCAR to reduce the risk of neurological complications and hemodynamic depression.

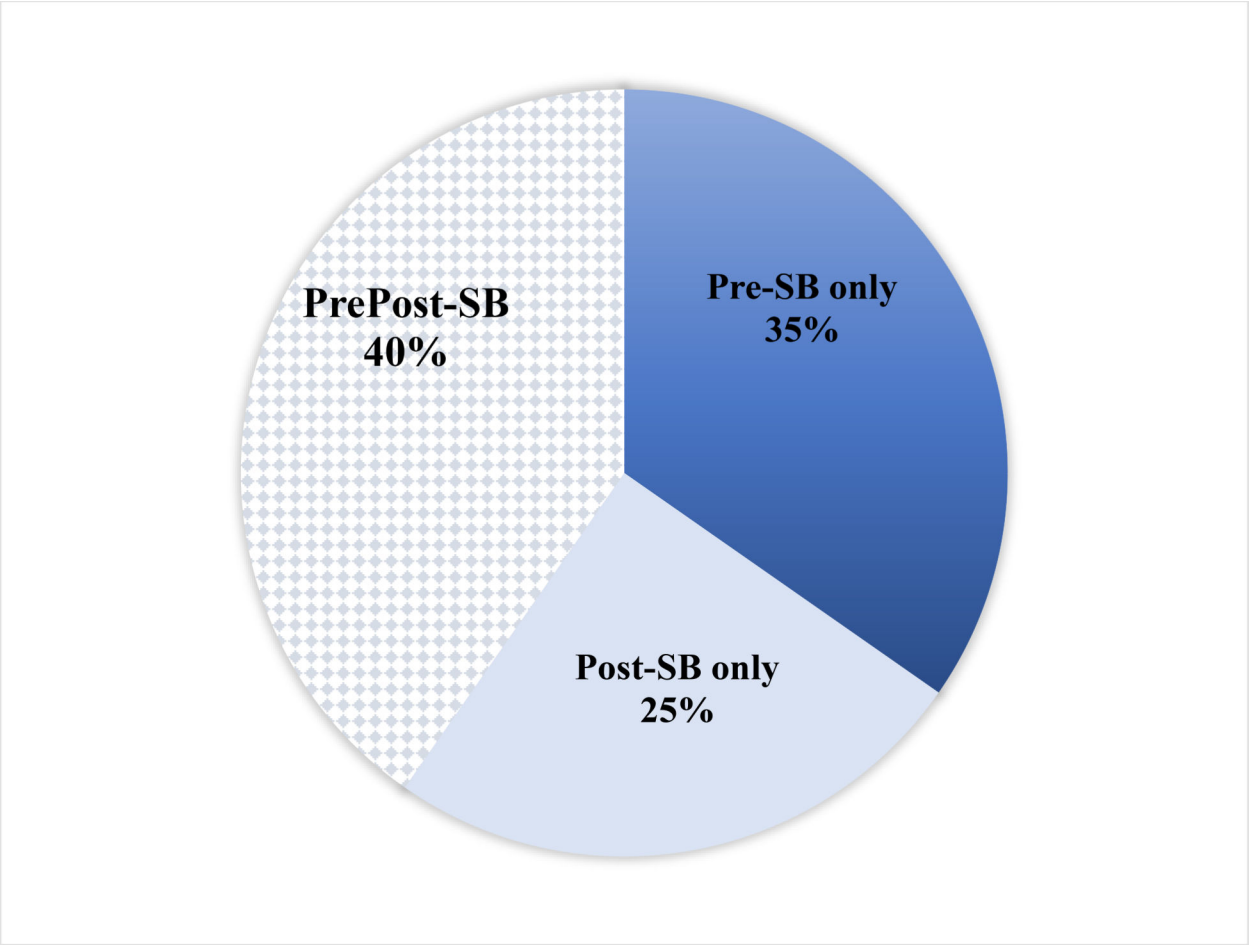

Figure 1.

Distribution of Balloon Angioplasty Use during TCAR

Pre-SB: pre-stent ballooning only; post-SB: post-stent ballooning only; prepost-SB: both pre-stent and post-stent ballooning

Table 1.

Baseline Characteristics

	Pre-SB *N=1,791 (34.7)*	Post-SB *N=1,290 (25.0)*	PrePost-SB *N=2,080 (40.3)*	P-value
Age in years, median (IQR)	74 (67–80)	74 (67–80)	75(68–88)	.04
Age ≥ 75 years	840 (46.9%)	638 (49.5%)	1,061 (51.5%)	.04
Female Gender	647(36.1)	480 (37.2)	763 (36.7)	.83
Non-White Race	199 (11.1)	125 (9.7)	177 (8.5)	.02
Hispanic or Latino	60 (3.4)	67 (5.2)	63 (3.0)	<.01
Symptomatic Status	609 (34.0)	363 (28.1)	584 (28.1)	<.001
Amaurosis fugax	67 (3.7)	42 (3.3)	66 (3.2)	.59
Transient Ischemic Attack	299 (16.7)	198 (15.4)	295 (14.2)	.10
Strohe	339 (18.9)	179 (13.9)	311 (15.0)	<.001
BMI in Kg/m^2, mean ± SD	28.3 ± 5.7	27.9 ± 5.5	28.8 ± 7.5	.001
Hypertension	1,613 (90.0)	1,164 (90.3)	1,913 (92.0)	.07
Diabetes	703 (39.2)	1459 (35.6)	807 (38.8)	.09
Coronary Artery Disease	881 (49.2)	688 (53.3)	1,117 (53.7)	.01
Congestive heart failure	304 (17.0)	243 (18.8)	419 (20.1)	.04
Prior CABG/PCI	702 (39.2)	555 (43.0)	849 (40.8)	.10
Chronic obstructive pulmonary disease	499 (27.9)	354 (27.4)	580 (27.9)	.95
Chronic kidney disease	661 (37.5)	502 (40.1)	804 (39.4)	.31
Hemodialysis	24 (1.3)	28 (2.1)	37 (1.7)	.41
Current Smoker	415 (23.2)	300 (23.3)	447 (21.5)	.22
Prior Ipsilateral CEA	316 (17.7)	224 (17.4)	286 (13.8)	.001
Prior Ipsilateral CAS	15 (0.8)	21 (1.6)	32 (1.5)	.09
Elective procedures	1,590(88.8)	1,173 (90.9)	1,881 (90.4)	.10
General Anesthesia	1,419 (79.3)	1,110 (86.1)	1,704 (82.0)	<.001
Preoperative Medications				
Aspirin	1,591 (88.8)	1,142 (88.5)	1,876 (90.2)	.23
P2Y12-Receptor Antagonists	1,558 (87.0)	1,095 (85.0)	1,820 (87.5)	.09
Anticoagulants	259 (14.5)	159 (12.3)	317 (15.2)	.06
Beta Blockers	1,006 (56.2)	728 (56.4)	1,244 (59.8)	.04
ACE Inhibitors	953 (53.2)	676 (52.4)	1,148 (55.2)	.24
Statin	1,589 (88.7)	1,122 (87.0)	1,863 (89.6)	.08
Lesion-Specific Characteristics				
Degree of Stenosis ≥ 80%	947 (53.7)	662 (53.5)	1,123 (56.1)	.22
Lesion length in mm, median (IQR)	22 (15–30)	21 (15–30)	25 (19–30)	<0.001
Lesion Location				0.48
Bifurcation	398 (22.3)	298 (23.2)	431 (20.7)	
ICA	1,315 (73.6)	930 (72.3)	1,539 (74.1)	
CCA	72 (4.0)	56 (4.4)	106 (5.1)	
ECA	2 (0.1)	2 (0.2)	2 (0.1)	

	Pre-SB *N=1,791 (34.7)*	Post-SB *N=1,290 (25.0)*	PrePost-SB *N=2,080 (40.3)*	P-value
ICA Distal Tortuosity				<0.001
None/Mild	1,312 (73.3)	905 (70.2)	1,376 (66.2)	
Moderate	172 (9.6)	150 (11.6)	224 (10.8)	
Severe	48 (2.7)	38 (2.9)	61 (2.9)	
Missing	259 (14.5)	197 (15.3)	419 (20.1)	
Lesion Calcifications				<0.001
None	240 (13.4)	145 (11.2)	205 (9.9)	
<=25% circumference	205 (11.5)	245 (19.0)	259 (12.4)	
26–50% circumference	172 (9.6)	174 (13.5)	245 (11.8)	
51–99% circumference	411 (22.9)	239 (18.5)	420 (20.2)	
100% circumferential	15 (0.8)	18 (1.4)	28 (1.3)	
Protruding into lumen	9 (0.5)	10 (0.8)	14 (0.7)	
Missing	739 (41.3)	459 (35.6)	909 (43.7)	

Abbreviations: *pre-SB: pre-stent ballooning only; post-SB: post-stent ballooning only; prepost-SB: both pre-stent and post-stent ballooning; IQR: interquartile range; BMI: body mass index; SD :standard deviation; CABG/PCI: Coronary artery bypass grafting/percutaneous coronary intervention; CEA: carotid endarterectomy; CAS: carotid artery stenting.*

Table 2.

In-Hospital and 30-day Outcomes

	Pre-SB *N=1,791*	Post-SB *N=1,290*	PrePost-SB *N=2,080*	P-value
In-Hospital Outcomes				
Death	11 (0.6)	4 (0.3)	6 (0.3)	.28
Ipsilateral Stroke	16 (0.9)	10 (0.8)	22 (1.1)	.72
Stroke	19 (1.1)	13 (1.0)	26 (1.3)	.77
TIA	4 (0.2)	12 (0.9)	21 (1.0)	<.01
Myocardial Infarction	8 (0.5)	9 (0.7)	10 (0.48)	.60
Stroke/Death	25 (1.4)	16 (1.2)	29 (1.4)	.92
Stroke/Death/MI	29 (1.6)	24 (1.9)	38 (1.8)	.85
Reperfusion Syndrome	8 (0.5)	5 (0.4)	8 (0.4)	.95
Completion Angiography				
Yes, without occlusion	1,160 (64.88)	867 (67.63)	1,389 (67)	.38
Yes, with occlusion	7 (0.39)	2 (0.16)	2 (0.10)	.38
Post-procedural hypotension	235 (13.1)	213 (16.6)	348 (16.8)	<.01
Post-procedural hypertension	274 (15.3)	174 (13.5)	258 (12.4)	.03
Operative time, mean ± SD	69.5 ± 27.1	72.3 ± 29.7	74.28 ± 29.7	<.001
Flow Reversal time, mean ± SD	9.7 ± 6.7	11.0 ± 8.6	12.1 ± 7.6	<.001
Fluoroscopy time, mean ± SD	5.2 ± 4.3	5.7 ± 8.7	6.5 ± 7.8	<.001
Length of stay in days, median (IQR)	1 (1–2)	1 (1–2)	1 (1–2)	.79
Thirty-day Mortality	16 (0.9)	9 (0.7)	11 (0.5)	.40
Thirty-Day Follow-up *	*N=358, 20%*	*N=553, 42.9%*	*N=468, 22.5%*	
Stroke	2 (0.6)	6 (1.1)	9 (2.0)	.19
Stroke/Death	4 (1.1)	7 (1.3)	9 (1.9)	.57
Stroke/D eath/MI	5 (1.4)	12 (2.2)	11 (2.4)	.60

Abbreviations: *pre-SB: pre-stent ballooning only; post-SB: post-stent ballooning only; prepost-SB: both pre-stent and post-stent ballooning; SD: standard deviation; TIA: transient ischemic attack; MI:myocardial infarction.*

* Event rates are calculated as the percentage of patients with the outcome out of the patients with available 30-day follow-up.

Table 3.

Adjusted In-hospital Outcomes

	Pre-SB	Post-SB		Pre-Post SB	
		OR (95 % CI)	*P-value*	*OR (95 % CI)*	*P-value*
In-Hospital Outcomes					
Mortality	*Ref*	0.33 (0.09, 1.14)	.08	0.42 (0.15, 1.13)	.09
Stroke	*Ref*	1.03 (0.48, 2.17)	.95	1.19 (0.62, 2.27)	.60
TIA	*Ref*	4.24 (1.51, 11.8)	.01	4.76 (1.53, 14.79)	.01
MI	*Ref*	1.48 (0.59, 3.69)	.40	0.89 (0.37, 2.12)	.79
Stroke/TIA	*Ref*	1.61 (0.89, 2.91)	.12	1.74 (0.97, 3.10)	.06
Stroke/Death	*Ref*	0.88 (0.44, 1.73)	.71	0.98 (0.57, 1.70)	.96
Stroke/Death/MI	*Ref*	1.12 (0.66, 1.90)	.68	1.05 (0.62, 1.78)	.85
Post-procedural Hypotension	*Ref*	1.26 (0.84, 1.91)	.27	1.28 (0.99, 1.67)	.06
Post-procedural Hypertension	*Ref*	0.87 (0.65, 1.16)	.35	0.82 (0.63, 1.07)	.15
Thirty-day Outcomes					
Mortality	*Ref*	0.63 (0.26–1.51)	.30	0.53 (0.26–1.08)	.08
Stroke	*Ref*	1.86 (0.43–8.1)	.41	2.90 (0.67–12.5)	.15
Stroke/Death	*Ref*	1.38 (0.38–5.04)	.62	1.55 (0.46–5.24)	.48
Stroke/Death/MI	*Ref*	1.70 (0.46–6.34)	.43	1.62 (0.48–5.51)	.44

Abbreviations: TIA, transient ischemic attack; MI, myocardial infarction; OR, odds ratio; CI, Confidence interval

Table 4.

Adjusted In-hospital Outcomes in Symptomatic and Asymptomatic Patients

		Asymptomatic Patients		Symptomatic Patients		Interaction (Difference-in-Difference)
	Pre-SB	Post-SB and Pre-Post SB*		Post-SB and Pre-Post SB		
		OR (95 % CI)	*P-value*	*OR (95 % CI)*	*P-value*	*P-value*
In-Hospital Outcomes						
Mortality	*Ref*	0.31 (0.09–1.05)	.06	0.62 (0.17–2.34)	.48	.44
Stroke	*Ref*	0.95 (0.44–2.08)	.91	1.29 (0.58–2.86)	.54	.58
TIA	*Ref*	4.2 (0.95–18.5)	.06	4.8 (1.15–20.0)	.03	.90
MI	*Ref*	0.94 (0.37–2.39)	.90	1.74 (0.36–8.36)	.49	.52
Stroke/TIA	*Ref*	1.49 (0.77–2.86)	.23	1.88 (0.91–3.88)	.09	.61
Stroke/Death	*Ref*	0.75 (0.39–1.41)	.37	1.24 (0.58–2.69)	.58	.29
Stroke/Death/MI	*Ref*	0.88 (0.50–1.56)	.67	1.38 (0.68–2.82)	.37	.32
Post-procedural Hypotension	*Ref*	1.25 (0.92–1.70)	.16	1.34 (0.91–1.98)	.14	.71
Post-procedural Hypertension	*Ref*	0.86 (0.67–1.11)	.26	0.80 (0.58–1.11)	.18	.66

*Post-SB and Pre-Post SB are considered as one group.

Abbreviations: TIA, transient ischemic attack; MI, myocardial infarction; OR, odds ratio; CI, Confidence interval