

Blood pressure improvement and long-term outcomes after carotid stenting in patients with hypertension and atherosclerotic carotid stenosis

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Abstract

Background: This study aimed to investigate the relationship between long-term blood pressure improvement following carotid stenting (CAS) and clinical outcomes.

Methods: In this study, 178 patients with hypertensive and carotid stenosis who underwent CAS were retrospectively analyzed and divided into a blood pressure improvement group (BP-I, n=59) and a blood pressure nonimprovement group (BP-NI, n=77) on the basis of whether their blood pressure improved at 24 months; the other 42 nonhypertensive patients were considered blank controls (BCs). The primary outcome was the major adverse cardiovascular and cerebrovascular event (MACCE)-free survival rate at 24 months. Survival analysis was performed using the Kaplan–Meier method, and Cox proportional hazards models were employed to identify potential predictors of MACCEs.

Results: Blood pressure improvement was achieved by 43.4% of the patients. The MACCE-free survival rate was significantly greater in the BP-I group than in the BP-NI group ($P=0.048$) at 24 months, and no difference was detected between the BP-I and BC groups; the MACCE-free survival rate was higher in the BC group, but the difference was not significant. Perioperative hemodynamics were less stable in the BP-I group, as these patients required significantly more dopamine support ($p<0.01$) and experienced longer durations of hypotension ($p=0.01$) than the patients in the BP-NI group did. Multivariate Cox regression revealed that a preoperative systolic blood pressure ≥ 140 mmHg was the strongest independent risk factor for MACCEs ($HR=6.26$; $p<0.001$), while moderate-to-high echogenicity plaques ($HR=0.29$; $p=0.019$) and a stent diameter >8 mm ($HR=0.13$; $p=0.002$) were significant protective factors.

Conclusion: Long-term blood pressure improvement following CAS is a potential indicator of better long-term outcomes.

Clinical trial number not applicable

Introduction

Ischemic stroke is among the leading causes of death and long-term disability worldwide, with up to 20% of cases attributable to carotid atherosclerotic stenosis (1, 2), and the risk of cerebrovascular events increases with stenosis (3, 4). Carotid artery stenting (CAS) has evolved into a well-established minimally invasive endovascular treatment modality that is particularly well suited for patients with severe stenosis, especially high-risk patients with carotid endarterectomy (CEA) (5). The radial support force of the stent stabilizes vulnerable atherosclerotic plaques, thereby preventing future stroke events (6–8). However, in clinical practice, in addition to local therapeutic effects, many patients experience a significant and persistent decrease in systemic blood pressure following CAS (9, 10). This phenomenon differs from acute, transient hypotension that occurs immediately after CAS. Acute hypotension is generally considered a direct consequence of the mechanical traction and stimulation of the baroreceptors in the carotid sinus during CAS angioplasty, which typically disappears within hours to days (11). Conversely, the prolonged reduced blood pressure observed in this study can persist for

months or even years. This clinical outcome is most commonly attributed to the baroreceptor resetting principle. The hypothesis is that chronic stenosis reduces baroreceptor sensitivity. The restoration of normal perfusion through revascularization via CAS triggers a functional readaptation that reduces the systemic blood pressure set point, which provides sustained antihypertensive benefits(12). However, these hypotheses lack direct clinical evidence. This gap in knowledge prompted us to conduct the present study. This study aims to determine whether this sustained reduction in blood pressure translates to quantifiable clinical benefits, such as reducing the risk of major adverse cardiovascular and cerebrovascular events (MACCEs), and to explore the molecular mechanisms underlying this beneficial phenomenon.

Materials and methods

Study design and population

This study was a multicenter retrospective cohort study. Patients with severe carotid artery stenosis treated between January 2019 and December 2021 at five high-volume hospitals were included. Ultimately, a total of 178 patients were included in the analysis (Figure 1); the blood pressure of 59 patients improved during the follow-up period (BP-I group), the blood pressure of 77 patients did not improve (BP-NI group) at the 24-month follow-up, and 42 nonhypertensive patients were included in the blank control (BC) group. All patients underwent color ultrasound and cerebral computed or magnetic resonance tomography examinations; the cerebral vessels and coronary arteries were evaluated by preoperative CT angiography (CTA), and then CAS was performed in the interventional room according to the established protocol. All the patients signed a treatment-related informed consent form. The study protocol was approved by the Ethics Review Committee of the First Affiliated Hospital of Xi'an Jiaotong University (Ethics No. XJTU1AF-CRF-2022-037), and additional research-related informed consent was not required because of its retrospective design. All treatment measures and follow-up procedures were in accordance with relevant clinical guidelines and system regulations.

The inclusion and exclusion criteria for patient selection were as follows.

Inclusion criteria:

1. Patients with symptomatic carotid stenosis and stenosis $\geq 70\%$ according to the European Carotid Surgery Trial (ECST) study
2. Patients with a life expectancy of 2 years or more and less than 80 years of age
3. Patients without any additional contraindications
4. The physician performed the procedure >100 times

Exclusion criteria:

1. Disabling stroke
2. Fatal stroke
3. Severe bilateral carotid stenosis ($\geq 70\%$) or contralateral carotid occlusion
4. Renal artery stenosis greater than 50%
5. Intracranial arterial stenosis greater than 70%
6. Symptomatic coronary artery disease
7. Uncontrolled hypertension, a systolic blood pressure greater than 180 mmHg despite the use of medication, or a history of hemorrhagic disease within 3 months
8. Patients with cardiac stroke, aspirin- and clopidogrel-resistant or active cancer and those who refused to participate

Endovascular procedure

All patients received dual-antiplatelet therapy (aspirin, 100 mg qd; clopidogrel 75 mg) and atorvastatin (20 mg) every day before the procedure for at least 3 days. This treatment regimen was implemented for 3 months, after which aspirin (100 mg) and atorvastatin (20 mg) were taken every day. Each procedure involved puncturing the femoral artery under local anesthesia with 1% lidocaine. After intravenous heparinization (70 U/kg), the long 7F sheath was guided to the common carotid artery. A cerebral protection device (Emboshield or Spider RX) was deployed at the distal internal carotid artery via a 0.014-inch wire system. Next, predilation was performed by using a 4.0- to 5.0-mm balloon catheter to initially widen the vessel (as a preventative measure, 0.5 mg of atropine was given before this step); then, a self-expanding stent (Acculink or Protégé RX) was placed across the lesion. If the residual stenosis was still greater than 30%, postdilation was performed using a larger 5- to 6-mm balloon catheter. Residual stenosis of less than 30% was defined as technical success. Finally, a closure device was applied to procedure site.

Surveillance

After the operation, all patients were moved to the intensive care unit (ICU) for close monitoring, and the medical team (including a doctor and a nurse) performed neurological checks every two hours. Low blood pressure (systolic pressure < 90 mmHg) was treated with intravenous fluids or specific drugs (such as dopamine). This treatment was used only if the patient was not bleeding and was not experiencing heart failure. If a patient reported symptoms or had any concerns during the study period, a separate medical team performed an examination. A follow-up appointment was scheduled for each patient at discharge.

Definitions

Long-term blood pressure improvement was defined as a composite endpoint designed to evaluate the overall blood pressure-related benefits of carotid stenting. This definition requires that patients meet one of the following criteria during long-term postoperative follow-up: 1. Discontinuation of medication: Their blood pressure remains below the target level of 140/90 mmHg without the use of antihypertensive drugs or meets stricter standards in patients adjusted for comorbidities. 2. The number or dosage of antihypertensive medications was significantly reduced compared with the preoperative baseline, while blood pressure control was maintained. On the basis of this definition, we divided the cohort into three groups: the BP-I group, in which the patients met the aforementioned criteria; the BP-NI group, in which the patients underwent CAS but did not meet the aforementioned improvement criteria; and the CK group, in which the patients underwent CAS treatment and did not exhibit hypertension pre- or postoperatively. Procedural hypotension and bradycardia were defined as any episode of decreased blood pressure or pulse that required intervention during the procedure and that resolved within 24 h. (13) Carotid restenosis was set at > 50% on the basis of ultrasound criteria. (14) Stroke and transient ischemic attack (TIA) were defined as previously reported; severe stroke included fatal and disabling events, mild stroke included nondisabling events, and myocardial infarction (MI) was defined as previously reported (15, 16). We systematically collected blood pressure data for each patient. We defined the baseline blood pressure as the average office blood pressure measured within 24 hours prior to surgery. The postoperative blood pressure was measured at multiple key time points: immediately after surgery; 6, 24, and 48 hours after surgery; at discharge; and at the 24-month postoperative follow-up. To accurately assess the net change in blood pressure, we recorded the types and doses of antihypertensive medications used by patients at each time point. The primary study end point was the occurrence of major adverse cardiovascular and cerebrovascular events (MACCE) within 2 years, which was defined as a composite of any of the following events: all-cause death, any stroke, and myocardial infarction. Secondary endpoints included each single event and target vessel revascularization (TLR) or hemodynamically significant restenosis. All endpoint events were evaluated by a certified independent neurologist with expertise in vascular disease.

Statistical analysis

Statistical analysis of the clinical data was performed using SPSS 25.0 software. $p < 0.05$ was considered to indicate statistical significance. Categorical variables were analyzed by the chi-square test, Fisher's exact test, or ANOVA, as appropriate, while continuous variables were first tested for normality. Continuous variables that conformed to a normal distribution are expressed as the mean $\pm$ standard deviation (mean $\pm$ SD), and comparisons between two groups were performed with independent samples t tests; those that did not conform to a normal distribution are expressed as the median $\pm$ interquartile range (median $\pm$ IQR), and comparisons between groups were performed with the Mann–Whitney U test. Categorical variables are expressed as numbers of patients (percentages), and comparisons between groups were performed using the χ^2 test or Fisher's exact test, as appropriate. Survival curves were plotted using the Kaplan–Meier method to assess the 24-month MACCE-free survival rate, and log-rank tests were used to compare differences among groups. To identify risk factors for MACCEs, univariate Cox risk regression analyses were first performed for all baseline characteristics;

then, multivariate Cox models including potential or significant risk factors were constructed. Univariate factors with a P value of <0.1, risk factors, and potential factors that affected blood pressure in patients across the clinics were included in the multifactorial Cox model analyses. Risk factors and protective factors for MACCEs were determined and are expressed as odds ratios (ORs) and 95% confidence intervals (CIs).

Results

Patient demographics and clinical characteristics

No differences were observed among the three groups in terms of demographic or clinical characteristics ($p>0.05$), except that the proportion of patients requiring two or more antihypertensive medications was significantly greater in the BP-NI group than in the BP-I group (72.7% vs. 54.2%, $p = 0.025$), as this was part of their baseline antihypertensive therapy regimen. These findings suggest that the hypertensive state of the patients in the BP-NI group may have been more difficult to control preoperatively (Table 1).

In terms of the lesion and procedural characteristics, the proportion of patients with moderate-to-high echogenicity plaques was significantly greater in the BP-I group than in the BP-NI group (71.2% vs. 51.9%, $p < 0.01$), and these features were less common in the BC group than in the BP-I and BP-NI groups. Other characteristics, including the lesion side, stenosis degree, stent design, and dilatation strategy, were comparable among the three groups (Table 2, $p > 0.05$).

Periprocedural hemodynamic characteristics

The baseline hemodynamic parameters of the patients were similar across all three groups ($p > 0.05$). The overall incidence of bradycardia and postoperative hypotension did not significantly differ across groups ($p=0.54$). However, the management and duration of hemodynamic instability differed significantly across the groups. While the proportion of patients requiring dopamine did not significantly differ across the groups ($p = 0.23$), the total dose administered was significantly lower in the BP-NI group (0.75 ± 1.77 mg) than in the BP-I (1.39 ± 3.04 mg) and control groups (2.76 ± 3.49 mg) ($p < 0.01$). Conversely, prolonged hypotension (>24 h) was significantly less frequent in the BP-NI group (19.5%) than in the BP-I (28.8%) and control groups (45.2%) (Table 3, $p=0.01$).

Clinical outcomes and blood pressure changes at 24 months

At the 24-month follow-up, compared with the BP-NI group, the BP-I group maintained significantly lower systolic (median 130 vs. 140 mmHg, $p < 0.01$) and diastolic (median 80 vs. 90 mmHg, $p < 0.01$) blood pressures (Table 4). Similarly, the median diastolic blood pressure was 80 mmHg (interquartile range [IQR]: 70–90 mmHg) in the BP-I group, which was significantly lower than the median diastolic blood pressure of 90 mmHg (IQR: 80–90 mmHg) in the BP-NI group ($p < 0.01$) (Table 4). The individual components of MACCEs did not differ significantly among the groups. However, the cumulative 24-

month MACCE incidence rate was lower in the BP-I (6.8%) and control (7.1%) groups than in the BP-NI group (18.2%). The overall MACCE-free survival rate tended to differ across the three groups ($p = 0.08$), and Kaplan–Meier analysis revealed a significantly higher MACCE-free survival rate in the BP-I group than in the BP-NI group (log-rank $p = 0.048$) (Figure 2).

Risk factors for MACCEs

Eleven significant potential risk factors for restenosis were identified via univariate Cox regression analysis and subsequently used to construct a multivariable Cox model. A preoperative systolic blood pressure greater than 140 mmHg was the strongest independent risk factor for MACCEs, with a significantly greater hazard ratio (HR = 6.26; 95% CI: 2.26–17.35; $p < 0.001$) compared with the other factors. Conversely, a moderate-to-high plaque echogenicity (HR = 0.29; 95% CI: 0.10–0.82; $p = 0.019$) and a common carotid artery diameter greater than 8 mm (HR = 0.13; 95% CI: 0.03–0.48; $p = 0.002$) were identified as independent protective factors (Table S1).

Discussion

This study reveals two key findings with significant clinical implications. First, we confirmed that the sustained improvement in blood pressure observed in some patients following carotid artery stenting (CAS) is directly associated with a significant reduction in the incidence of major adverse cardiovascular and cerebrovascular events (MACCEs) over a two-year period. This finding provides the first robust evidence that the long-term benefits of CAS may extend beyond localized stroke prevention, at least partially through a systemic reduction in blood pressure. While previous studies have reported short-term reductions in blood pressure after CAS, few works have explored the long-term prognostic significance of this phenomenon(12, 17, 18). Our Kaplan–Meier analysis ($p = 0.048$) clearly revealed the prognostic trajectories of patients with sustained and non-sustained improvements in blood pressure. Thus, clinicians should consider post-CAS blood pressure monitoring and management as critical determinants of long-term outcomes rather than merely methods to evaluate postoperative complications.

Short- and long-term reductions in blood pressure were observed after CAS was performed. Acute transient hypotension is common perioperatively and is well documented. It is widely attributed to the direct mechanical stimulation of carotid sinus pressure receptors and typically resolves rapidly(19). In contrast, the sustained blood pressure improvement observed in our cohort represents a deeper, long-term physiological adaptation. This benefit is particularly pronounced in patients with preexisting hypertension, which is consistent with observations in other interventional therapy contexts, such as renal denervation, where a stronger therapeutic response is predicted by a higher baseline blood pressure(20, 21). Conversely, for individuals with normal blood pressure levels, this hypotensive effect may be minimal or entirely absent, suggesting targeted therapeutic benefits for specific populations. However, while the hemodynamic instability observed in the BP-I group may indicate a robust adaptive response, excessive or persistent perioperative hypotension is not benign. Such events are recognized

as risk factors for adverse outcomes, including cerebral hypoperfusion and myocardial ischemia, and these concerns are substantiated by extensive studies in the domains of critical care and anesthesiology(22, 23).

Consistent with the extensive epidemiological data, the multivariate Cox regression analysis revealed that a preoperative systolic blood pressure ≥ 140 mmHg was the most significant independent risk factor for MACCEs(24). Furthermore, we identified two independent protective factors: moderate-to-high echogenicity plaques and a common carotid artery stent diameter > 8 mm. The former may indicate a more stable lesion phenotype and lower inflammation at baseline, whereas the latter suggests that anatomical and mechanical factors play crucial roles in long-term postoperative vascular health. Furthermore, our analysis of perioperative hemodynamic factors revealed that both the BP-I group and the blank control group required higher doses of dopamine support and experienced longer periods of hypotension during the perioperative period. One possible explanation is that the vascular or neuromodulatory systems of these patients may have exhibited heightened responses to the mechanical intervention involved with CAS. The smoother perioperative hemodynamics of the BP-NI group may reflect the systemic insensitivity of these patients to mechanical stimulation, resulting in a failure to produce long-term beneficial regulatory effects(25).

From a pathophysiological perspective, the mechanism underlying this sustained reduction in blood pressure following CAS warrants further investigation. We postulate that this phenomenon is similar to the principles underlying baroreflex activation therapy (BAT). Specifically, the deployment of a self-expanding nitinol stent in the carotid bulb exerts a continuous outward radial force, which results in persistent mechanical stretching of the vascular wall and embedded baroreceptors. This chronic stimulation increases afferent nerve firing to the nucleus tractus solitarii in the brainstem, thereby downregulating central sympathetic outflow and increasing parasympathetic (vagal) tone (26–28). Over time, this shifts the baroreflex operating point to a lower blood pressure threshold—a phenomenon known as “baroreflex resetting”. This mechanistic framework explains not only the long-term hypotensive effect but also why the use of stents with larger diameters (> 8 mm) was identified as an independent protective factor against MACCEs in our analysis. An adequate stent size ensures an optimal radial force and sustained baroreceptor engagement. However, undersized stents may fail to sufficiently stretch the wall, and oversized stents might induce excessive endothelial injury and intimal hyperplasia(29, 30).

With respect to plaque morphology, our finding that moderate-to-high echogenicity is a protective factor aligns with established histological correlates of carotid atherosclerosis(31, 32). Hypoechoic plaques are predominantly lipid rich with thin fibrous caps, rendering them highly vulnerable to rupture and distal microembolization during wire navigation and stent deployment. In contrast, moderate-to-high echogenic plaques are typically fibrotic or calcified, indicating a more stable inflammatory milieu. The presence of stable plaques likely mitigates the risk of perioperative cerebral microinfarcts, which, even when clinically silent, can contribute to long-term cognitive decline and increased vulnerability to MACCEs (33–35).

Limitations

This study has several limitations. First, as this was a retrospective study, selection bias and confounding factors cannot be completely ruled out. Second, the relatively small sample size may have affected the statistical power of the analyses, particularly when low-incidence events were assessed. Third, the blood pressure measurements relied primarily on static clinical recordings rather than 24-hour ambulatory blood pressure monitoring (ABPM). ABPM provides a more granular assessment of blood pressure variability and nocturnal changes and eliminates the “white-coat” effect, which could refine our understanding of post-CAS hemodynamic changes.

Conclusions

Our results reveal that carotid artery stenting has additional benefits that extend significantly beyond the localized restoration of cerebral perfusion. The sustained improvement in blood pressure following CAS is a critical surrogate marker for long-term cardiovascular and cerebrovascular health, indicating a significantly reduced risk of MACCEs.

Declarations

Acknowledgments

The language of this article was edited by American Journal Experts (AJE).

Author contributions

Study design: Haodi Zhang, Hufei Chang and Lin Yang; data collection and data analysis: Haodi Zhang, Hufei Chang, Feng Jiang, Yi Jia, Jiabin Wang, Ze Chen. Yuhao Lin, Zhaosheng Jia, Jiale Liang, Yichu Guo, Longlong Cong, Jianfeng Han and Lin Yang; data interpretation: all authors; writing: Haodi Zhang and Lin Yang; revision and review of the article: all authors.

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Conflict of interest

The authors declare that there are no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Availability of Data and Materials

The data that support the findings of this study are available from the hospital but restrictions apply to the availability of these data, which were used under license for the current study, and so are not publicly

available. Data are however available from the authors upon reasonable request and with permission of the hospital.

Ethical

This study was approved by the ethics committee of the First Affiliated Hospital of Xi'an Jiaotong University (XJTU1AF-CRF-2022-037) in accordance with the ethical standards provided in the 1964 Declaration of Helsinki and its later amendments, and all patients provided written informed consent.

All participating centers

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Tables

Table 1. Baseline clinical characteristics of the study population.

	BP-NI (n = 77)	BP-I (n = 59)	BC (n = 42)	p value
Age (year)	68 ± 8	68 ± 7	66 ± 9	0.18
BMI	24.17 (22.31, 25.39)	23.45 (21.97, 25.46)	23.58 (21.97, 24.96)	0.48
Age≥70	32 (41.6%)	27 (45.8%)	14 (33.3%)	0.45
Male	65 (84.4%)	52 (88.1%)	39 (92.9%)	0.41
BMI≥26	14 (18.2%)	9 (15.3%)	5 (11.9%)	0.67
HBP	73 (94.8%)	59 (100.0%)	N/A	0.13
HBP degree	56 (72.7%)	32 (54.2%)	N/A	0.03
Dual-medicine for HBP control	5 (6.5%)	26 (44.1%)	N/A	<0.01
PAD	4 (5.2%)	6 (10.2%)	2 (4.8%)	0.55
CAD	27 (37.0%)	26 (47.3%)	13 (34.2%)	0.36
Diabetes	27 (35.1%)	18 (30.5%)	14 (33.3%)	0.86
Hyperlipidemia	21 (27.3%)	14 (23.7%)	8 (19.0%)	0.60
Homocysteine	13 (16.9%)	8 (13.6%)	4 (9.5%)	0.54
Smoking	59 (76.6%)	39 (66.1%)	35 (83.3%)	0.13
Drinking	11 (14.3%)	9 (15.3%)	8 (19.0%)	0.79

BP-NI group: blood pressure nonimprovement group; BPI group: blood pressure improvement group; BC: blank control; M: male; BMI: body mass index; HBP: high blood pressure; PAD: peripheral arterial disease; CAD, coronary artery disease. P values were compared among the three groups.

Table 2. Angiographic and procedural characteristics.

	BP-NI (n=77)	BP-I (n = 59)	BC (n = 42)	p value
Stenosis > 70%	72 (93.5%)	53 (89.8%)	38 (90.5%)	0.73
Location (R)	39 (50.6%)	27 (45.8%)	22 (52.4%)	0.77
Plaque echogenicity ≥ moderate	40(51.9%)	42(71.2%)	22(52.4%)	<0.01
Tapered stent	42 (54.5%)	33 (55.9%)	19 (45.2%)	0.52
Post-dilatation	36 (46.8%)	18 (30.5%)	18 (42.9%)	0.15
Pre-dilation	76 (98.7%)	59 (100.0%)	41 (97.6%)	0.71
Pre-dilation diameter (mm)	4.56 ± 0.76	4.66 ± 0.95	4.29 ± 0.74	0.07
CCA diameter (mm)	7.48 ± 0.99	7.69 ± 0.82	7.45 ± 0.81	0.28
CCA diameter > 8 mm	30 (39.0%)	23 (39.0%)	11 (26.2%)	0.32
ICA diameter (mm)	5.09 ± 0.76	5.26 ± 0.67	5.10 ± 0.70	0.33
ICA diameter > 5 mm	41 (53.2%)	36 (61.0%)	25 (59.5%)	0.62
Dilation No. ≥ 2 times	19 (24.7%)	22 (37.3%)	16 (38.1%)	0.18

BP-NI group: blood pressure nonimprovement group; BPI group: blood pressure improvement group; BC: blank control; R: right; CCA: common carotid artery; ICA: internal carotid artery; No.: number. P values were compared among the three groups.

Table 3. Perioperative hemodynamic Parameters.

	BP-NI (n=77)	BP-I (n=59)	BC (n = 42)	p value
Pre-op heart rate (bpm)	72 ± 9	72 ± 8	71 ± 9	0.83
Pre-op SBP (mmHg)	144 ± 18	140 ± 16	N/A	0.11
Pre-op DBP (mmHg)	77 ± 10	78 ± 9	N/A	0.78
Post-op heart rate (bpm)	56 ± 8	58 ± 8	57 ± 10	0.36
Duration of bradycardia	4.5 ± 5.2	4.9 ± 5.5	3.3 ± 2.7	0.27
Post-op SBP (mmHg)	101 ± 16	98 ± 15	96 ± 13	0.28
Post-op DBP (mmHg)	60 ± 12	57 ± 10	57 ± 8	0.31
Vasoactive drugs use	24 (40.7%)	20 (52.6%)	20 (48.8%)	0.48
Dopamine USE	24 (31.2%)	18 (30.5%)	19 (45.2%)	0.23
Total dopamine dose (mg)	0.75 ± 1.77	1.39 ± 3.04	2.76 ± 3.49	<0.01
HR reduction (bpm)	16 ± 9	14 ± 9	14 ± 9	0.47
Bradycardia	52 (67.5%)	38 (64.4%)	29 (69.0%)	0.87
Hypotension	20 (26.0%)	20 (33.9%)	14 (33.3%)	0.54
Reduction in SBP (mmHg)	44 ± 20	41 ± 18	37 ± 17	0.15
SBP drop > 50 mmHg,	28 (36.4%)	18 (30.5%)	10 (23.8%)	0.36
Reduction in DBP (mmHg)	17 ± 13	20 ± 12	19 ± 8	0.35
DBP drop > 20 mmHg	31 (40.3%)	35 (59.3%)	23 (54.8%)	0.06
Hypotension duration > 24 h	15 (19.5%)	17 (28.8%)	19 (45.2%)	0.01

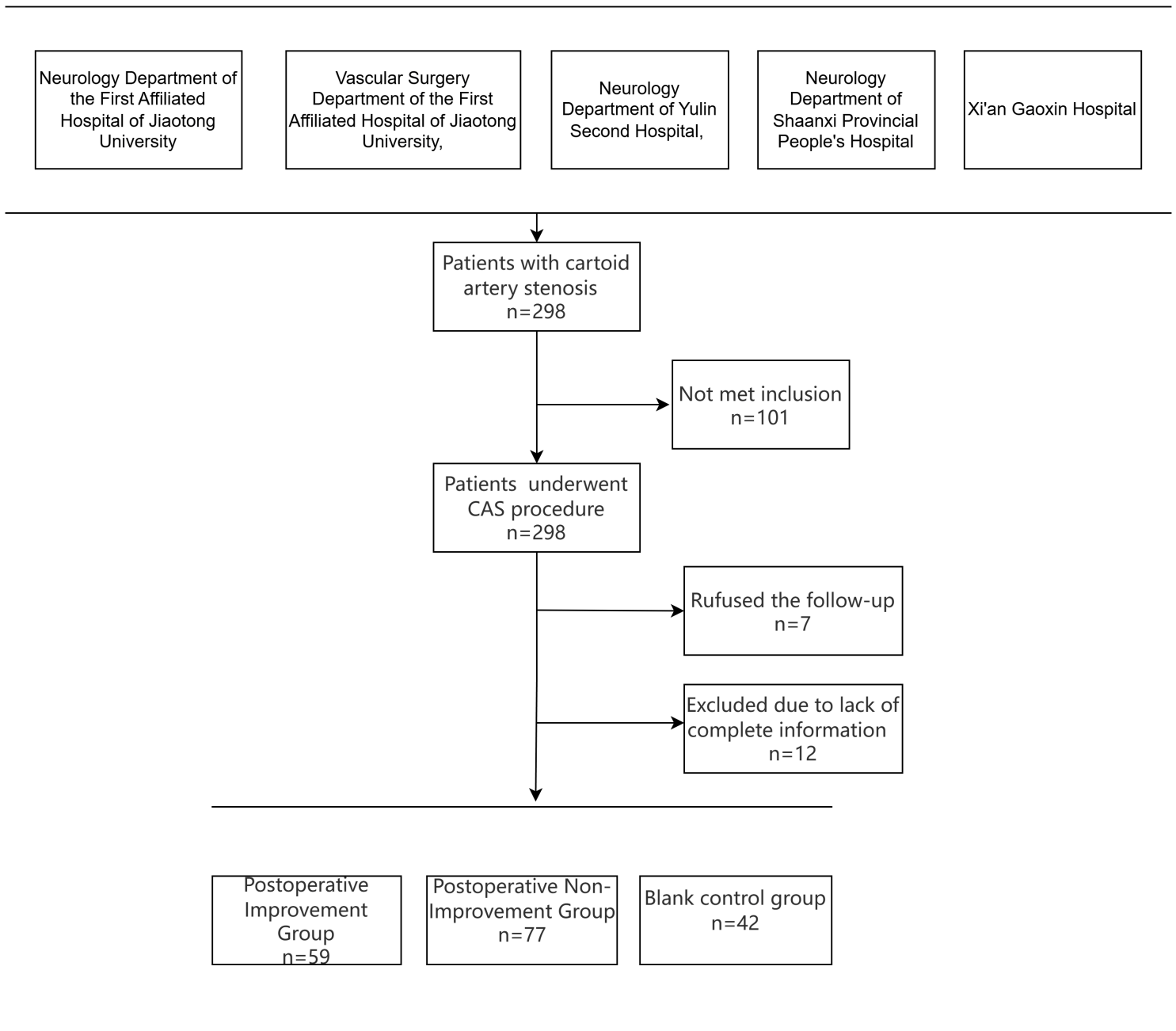
BP-NI group: blood pressure nonimprovement group; BPI group: blood pressure improvement group; BC: blank control; bpm: beats per minute; SBP: systolic blood pressure; DBP: diastolic blood pressure; mg: milligram; h: hour. P values were compared among the three groups.

Table 4. Follow-up outcomes at 24 months.

	BP-NI (n=77)	BP-I (n=59)	BC (n = 42)	p value
24-month SBP (mmHg)	140 (130, 140)	130 (130, 135)	N/A	<0.01
24-month DBP (mmHg)	90 (80, 90)	80 (70, 90)	N/A	<0.01
24-month SBP > 140 mmHg	48 (62.3%)	13 (22.0%)	N/A	<0.01
24-month DBP > 90 mmHg	52(67.5%)	17(28.8%)	N/A	<0.01
Post-operative stroke	4 (5.2%)	1 (1.7%)	0 (0.0%)	0.30
Restenosis	4 (5.2%)	1 (1.7%)	1 (2.4%)	0.57
Myocardial infarction	5 (6.5%)	1 (1.7%)	1 (2.4%)	0.38
Death	4 (5.2%)	1 (1.7%)	1 (2.4%)	0.57
Withdrawal of one drug	0 (0.0%)	16 (27.1%)	0 (0.0%)	<0.01
Withdrawal of $\geq$ two drugs	0 (0.0%)	1 (1.7%)	0 (0.0%)	0.57
MACCE-free survival	14 (18.2%)	4 (6.8%)	3 (7.1%)	0.08

BP-NI group: blood pressure nonimprovement group; BPI group: blood pressure improvement group; BC: blank control; SBP: systolic blood pressure; DBP: diastolic blood pressure; MACCE: major adverse cardiac and cerebrovascular event.

Figures



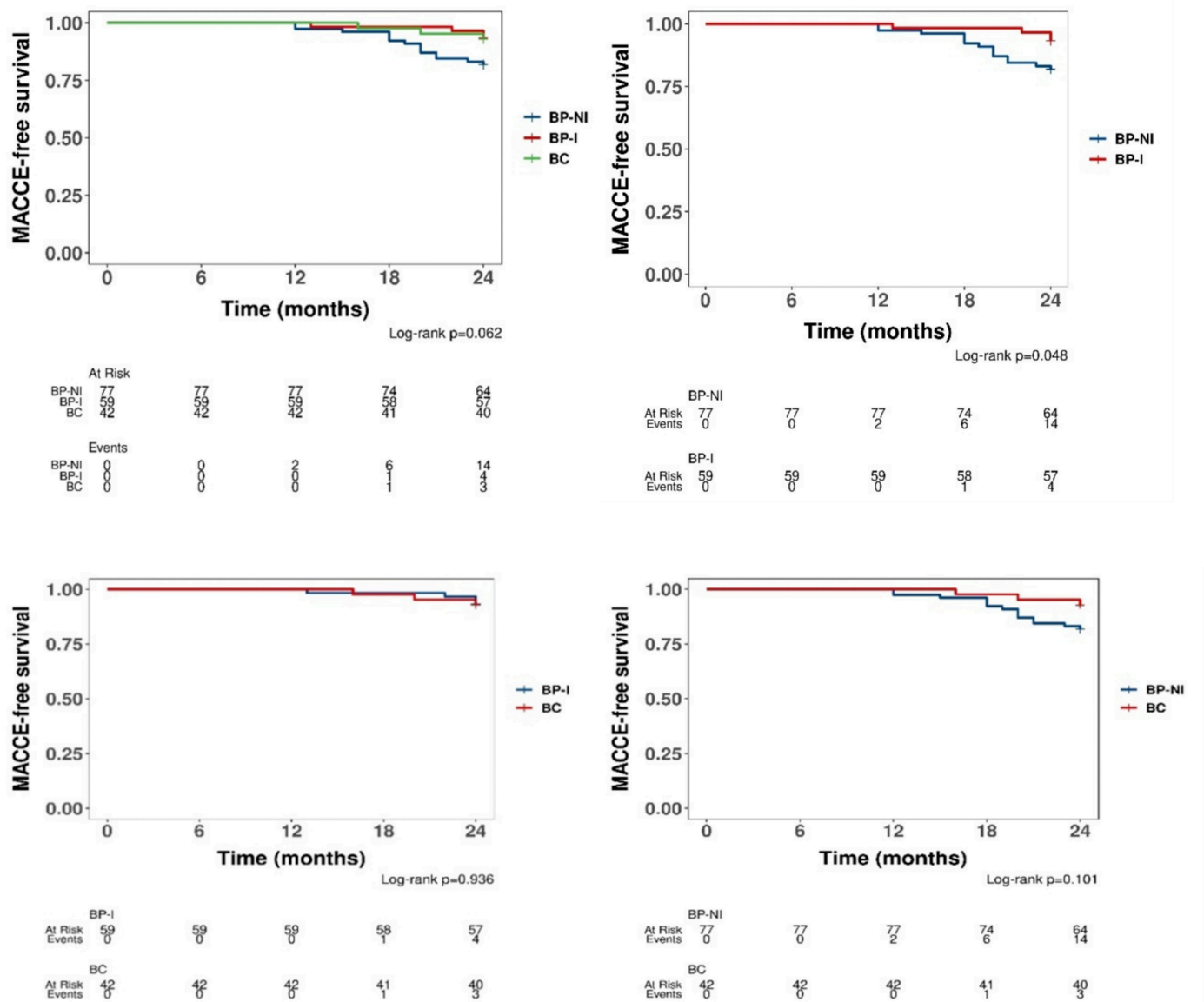


Figure 2

The MACCE--free survival rate at 24 months across the three groups according to Kaplan–Meier analysis. A, Overall comparison of the MACCE-free survival rate in the BP-NI (blue line), BP-I (red line), and BC (green line) groups. B, Comparison between the BP-I group and the BP-NI group. C, Comparison between the BP-I group and the BC group. D, Comparison between the BP-NI group and the BC group. MACCE, major adverse cardiac and cerebrovascular events; BP-NI group, blood pressure nonimprovement group; BP-I group, blood pressure improvement group; BC group, blank control group.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- [SupplementalTable1.docx](#)