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Article

Hypotensive Effect After Left Atrial Appendage Clipping in Patients With Atrial Fibrillation: The Role of B-Type Natriuretic Peptide

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Abstract

Background: Atrial fibrillation (AF) is associated with an increased risk of thromboembolism, primarily due to thrombus formation in the left atrial appendage (LAA). While anticoagulation represents the standard therapy, patients with a high risk of bleeding require alternative strategies such as LAA clipping (LAAC); hypotension has been observed in some patients post-LAAC. This study aimed to investigate the hypotensive effect after LAAC in patients with AF, and to explore the potential mediating role of B-type natriuretic peptide (BNP). **Methods**: A retrospective single-center analysis was conducted on 99 patients who underwent a standalone totally thoracoscopic LAAC. Plasma BNP levels, aldosterone, electrolyte levels, and blood pressure were measured preoperatively and at defined intervals postoperatively. Echocardiographic parameters and medication use were also analyzed. **Results**: A significant increase in BNP level was observed following LAAC (150.9 pg/mL [interquartile range (IQR) 77.8–260.7] to 316.2 pg/mL [IQR 197.5–466.8]; $p < 0.001$), accompanied by a significant decrease in aldosterone (5.5 ng/dL [IQR 3.0–8.7] to 2.5 ng/dL [IQR 1.4–3.4]; $p < 0.001$). Moreover, LAAC was associated with a marked reduction in systolic (127.3 ± 12.3 mmHg to 116.9 ± 12.4 mmHg; $p < 0.001$) and diastolic (80.7 ± 7.7 mmHg to 67.4 ± 9.3 mmHg; $p < 0.001$) blood pressure. Significant decreases in serum sodium and increases in serum potassium were also noted. A significant reduction in the use of angiotensin converting enzyme inhibitor (ACEI)/angiotensin receptor blocker (ARB) and calcium channel blocker (CCB) antihypertensive agents was observed postoperatively. No significant changes in cardiac structure or function were detected. The decrease in blood pressure persisted at 6-month follow-up. **Conclusions**: LAAC-induced hypotension may be partly mediated by BNP-driven suppression of aldosterone, thereby promoting natriuresis and reduced blood volume. These findings highlight the need for postoperative hemodynamic monitoring and antihypertensive medication adjustment in AF patients who undergo LAAC.

Keywords: atrial fibrillation; left atrial appendage clipping; blood pressure; B-type natriuretic peptide; aldosterone; surgery

1. Introduction

Atrial fibrillation (AF) is a common cardiac arrhythmia with an overall prevalence of 1–2%. The incidence of AF increases by 1% annually with advancing age, reaching 15% in individuals aged ≥70 years [1–3]. AF primarily predisposes to left atrial thrombus formation, with >90% of thrombi originating in the left atrial appendage (LAA) [4,5]. Dislodgement of these thrombi may lead to stroke or systemic embolism [6]. Current guidelines recommend anticoagulation therapy for patients with a high thromboembolic risk (CHA_2DS_2-VASc score ≥2) [7]. Commonly used anticoagulants include warfarin and direct oral anticoagulants (DOACs) [8]. However, AF patients with a high bleeding risk (HAS-BLED score >3) cannot tolerate long-term anticoagulation. In such cases, left atrial appendage clipping (LAAC) serves as an alternative mechanical strategy to reduce stroke risk [9], providing an option when anticoagulation is contraindicated (HAS-BLED indicates Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly, Drugs/alcohol concomitantly). The procedure for LAA closure involves epicardial blunt clipping of the LAA, thereby isolating it from the left atrium and reducing thrombotic risk [10,11]. The evidence to date indicates that AF patients may experience reduced blood pressure following LAAC [12], possibly because blunt compression from the LAA clip on the tissue leads to massive release of B-type natriuretic peptide (BNP) into the bloodstream. Nevertheless, the intrinsic mechanism for the decrease in blood pressure remains unclear. The aim of this study was therefore to investigate the impact of LAAC on blood pressure and to explore the potential underlying mechanisms.

2. Materials and Methods

2.1 Study Population

A retrospective analysis was conducted on patients who underwent totally thoracoscopic LAAC at Beijing Tiantan Hospital, Capital Medical University, between January 2020 and May 2025. A total of 99 patients were included in this study, comprising 71 males (71.7%) and 28 females (28.3%). Among them, 69 patients (69.7%) had a history of cerebral infarction, 12 (12.1%) had cerebral hemorrhage, and 75 (75.8%) had hypertension. Detailed demographic and clinical characteristics of the study cohort are presented in Table 1.

2.2 Inclusion and Exclusion Criteria

The study enrolled patients aged >18 years who underwent stand-alone totally thoracoscopic LAAC with documented AF on multi-lead electrocardiogram (ECG). AF was classified according to the duration and treatment strategy: paroxysmal AF (<7 days), persistent AF (≥7 days), long-standing persistent AF (>1 year with continued rhythm control attempts), and permanent AF (>1 year with no further rhythm control efforts). Exclusion criteria comprised: (1) history of ipsilateral thoracotomy, (2) prior open-heart surgery, (3) concomitant valvular heart disease, or (4) planned hybrid approaches (e.g., combined catheter ablation or surgical maze procedure).

2.3 AF Management Strategy Rationale

All enrolled patients were deemed unsuitable for rhythm control therapy based on three primary clinical considerations: (1) the predominance of long-standing, persistent, and permanent AF; (2) documented failure of prior anti-arrhythmic drug therapy; and (3) patients with permanent AF (>12 months duration) who opted against rhythm control strategies. Consistent with current guideline recommendations [7], surgical clip placement was performed exclusively as a stand-alone procedure for stroke prevention in this high-risk population. To specifically isolate the effects of LAAC, hybrid approaches involving concomitant ablation procedures were systematically excluded, as per the study protocol design.

2.4 Surgical Method

The patient was placed in the right lateral decubitus position with the left upper limb suspended. General anesthesia was induced with double-lumen endotracheal intubation, and invasive monitoring was established via radial arterial line and central venous catheterization. Three thoracic ports were created: first, a 5-mm trocar at the 4th intercostal space (ICS), anterior axillary line (operative port); second, a 5-mm trocar at the 5th ICS, posterior axillary line (operative port); and finally, a 10-mm trocar at the 6th ICS, anterior axillary line (thoracoscopic port). An additional 2-cm incision was made at the 7th ICS, posterior axillary line for LAA clip delivery. Upon entering the thoracic cavity, the lung was retracted posteriorly using a peanut dissector to expose the left pericardium and phrenic nerve. The pericardium was incised 1 cm posterior to the phrenic nerve using electrocautery, extending from the inferior border of the left pulmonary artery to the base of the LAA. The pericardium was then suspended to fully expose the LAA. The root diameter of the LAA was measured, and an appropriately sized C-Clip® device (Med-zenith, Inc., Beijing, China) was selected. The clip was introduced through the 2-cm incision, looped over the LAA apex, and positioned at the LAA base. It was deployed gradually under continuous hemodynamic and electrocardiographic monitoring. Final release was performed only after meeting the following criteria under transesophageal echocardiographic (TEE) guidance: (1) absence of significant ECG changes, (2) complete cessation of LAA flow on color Doppler imaging, and (3) confirmation of clip placement at the LAA base with a residual stump length of <1 cm. Upon confirmation of stable hemodynamics and meeting all the release criteria, the clip was fully released. Finally, the pericardium was closed, a chest tube was placed through the thoracoscopic port, and the incisions were sutured. The success of LAAC was confirmed by pre-discharge left atrial CTA in all cases.

2.5 Blood Pressure Measurements

The patient's blood pressure was measured each morning while they were quiet in the ward. Three measurements were taken to record the mean blood pressure value. Bilateral upper limb blood pressure was measured, and the blood pressure of the higher limb was also recorded. Arterial pressure was systematically assessed at the following predefined peri- and post-operative intervals: preoperatively, on the day of surgery, and on the first, second, and third postoperative days, concluding with a final measurement prior to discharge. As part of the long-term follow-up, blood pressure was again recorded during the outpatient clinic visit at 6 months post-discharge.

2.6 Transthoracic Cardiac Ultrasound Measurements

Transthoracic echocardiography was performed using a GE Vivid E95 ultrasound system (GE Healthcare, Chicago, IL, USA) to obtain standardized measurements of left atrial anteroposterior diameter (LAAPD), ejection fraction (EF), and left ventricular end-diastolic diameter (LVEDD) at baseline (preoperative) and 1-week postoperative time points. All examinations were conducted with patients in the resting supine position following standard protocols. The following measurements were obtained from the parasternal long-axis view: left atrial diameter was measured at end-systole as the maximal vertical distance between the anterior and posterior walls of the left atrium, perpendicular to the long axis of the ventricle. Left ventricular internal diameter was measured at end-diastole, defined as the frame immediately preceding mitral valve closure.

For each parameter, measurements were performed across three consecutive cardiac cycles that demonstrated optimal visualization of cardiac structures. The mean value of these measurements was calculated and used for subsequent analysis to minimize beat-to-beat variability. All measurements were performed offline by an experienced sonographer using dedicated analysis software (EchoPAC, GE Healthcare, Chicago, IL, USA).

2.7 Blood Sample Processing

Fasting venous blood samples were collected preoperatively, on the operative day, postoperative days 1–3, and at 1 week after surgery. After centrifugation, plasma samples were sent to the central laboratory for analysis. Plasma BNP and aldosterone levels were measured by chemiluminescence immunoassay using an automated biochemical analyzer, while serum sodium and potassium concentrations were determined by spectrophotometry.

2.8 Pharmacological Data Collection

The use of medications was captured through the extraction of electronic health records and direct patient interviews. Medication profiles documenting antihypertensives, anticoagulants, and antiplatelet agents were systematically recorded at preoperative baseline, as well as 1-week and 6-month post-LAAC.

2.9 Statistical Analysis

All statistical analyses were performed using SPSS 22.0 (IBM Inc., Chicago, IL, USA). Normally distributed continuous variables were presented as the mean ± standard deviation ($\bar{x}$ ± s), and within-group comparisons were analyzed by paired samples *t*-test. For variables measured serially over time (e.g., blood pressure at preoperative, intraoperative, postoperative days 1–3, discharge, and 6-month time points), a Repeated-Measures Analysis of Variance (Repeated-Measures ANOVA) was used to assess the overall effect of time. Non-normally distributed continuous variables were presented as the median (interquartile range) (M [P25, P75]), and within-group comparisons were analyzed by the Wilcoxon signed-rank test. Categorical variables were presented as the number of cases (percentage) [*n* (%)], and between-group comparisons were analyzed by the chi-square test. Binary logistic regression was employed to identify factors associated with reduced blood pressure (dependent variable: yes/no). Candidate predictors were entered into the model using the forward likelihood ratio method. Results are reported as the odds ratio (OR) with 95% confidence interval (CI). Model fit was assessed using the Hosmer–Lemeshow test, and a two-tailed *p*-value < 0.05 was considered statistically significant for all analyses.

3. Results

3.1 Baseline Characteristics

This study enrolled 99 patients comprising 71 males (71.7%) and 28 females (28.3%), with a mean age of 66.1 ± 7.4 years. The median CHA_2DS_2-VASc and HAS-BLED scores of the cohort were 5 (IQR 4–6) and 4 (IQR 2–5), respectively. A total of 69.7% of patients had a history of cerebral infarction, while 12.1% had experienced cerebral hemorrhage. Hypertension was present in 75.8% of patients. Persistent AF was the most common AF type (56.6%), followed by permanent AF (32.3%) and paroxysmal AF (11.1%). Complete baseline characteristics are presented in Table 1.

Table 1. Basic clinical data of patients with AF.

Variable	Value
Age (years)	66.1 ± 7.4
Gender (male)	71 (71.7%)
Height (cm)	170 (162, 175)
Weight (kg)	72.1 ± 11.8
CHA_2DS_2-VASc score	5 (4, 6)
HAS-BLED score	4 (2, 5)
Cerebral infarction	69 (69.7%)
Cerebral hemorrhage	12 (12.1%)
Type of atrial fibrillation	
Paroxysmal	11 (11.1%)
Persistent	56 (56.6%)
Permanent	32 (32.3%)
Coronary artery disease	30 (30.3%)
Myocardial infarction	4 (4.0%)
Hypertension	75 (75.8%)
Peripheral vascular disease	82 (82.8%)
Type 2 diabetes mellitus	31 (31.3%)

Note: Data is presented as the mean ± SD, n (%), or median (IQR). HAS-BLED indicates Hypertension, Abnormal renal/liver function, Stroke, Bleeding history or predisposition, Labile INR, Elderly, Drugs/alcohol concomitantly.

3.2 BNP, Aldosterone, and Blood Pressure Before and After LAAC

Comparative analysis revealed significant postoperative changes across multiple physiological parameters. BNP levels showed a marked elevation from preoperative levels of 150.9 pg/mL (IQR 77.8–260.7) to 316.2 pg/mL (IQR 197.5–466.8) ($p < 0.001$), while aldosterone concentrations showed a significant reduction from 5.5 ng/dL (IQR 3.0–8.7) to 2.5 ng/dL (IQR 1.4–3.4) ($p < 0.001$). Concurrently, clinically relevant decreases were observed in both systolic (127.3 ± 12.3 mmHg to 116.9 ± 12.4 mmHg; $p < 0.001$) and diastolic blood pressure (80.7 ± 7.7 mmHg to 67.4 ± 9.3 mmHg; $p < 0.001$). Electrolyte analysis revealed a decrease in serum sodium (142.0 ± 2.5 mmol/L to 137.6 ± 2.8 mmol/L; $p < 0.001$), accompanied by a modest but statistically significant increase in serum potassium (3.9 ± 0.4 mmol/L to 4.1 ± 0.3 mmol/L; $p < 0.001$). Complete comparative data are presented in Table 2.

3.3 Cardiac Structural Changes Following LAAC

Pre-discharge echocardiographic evaluation revealed no significant changes in cardiac structure or function compared to preoperative measurements. The left ventricular end-diastolic diameter (LVEDD) remained stable (46.3 ± 4.5 mm preoperatively vs 46.2 ± 4.5 mm at discharge, $p = 0.629$). Similarly, left atrial anteroposterior diameter (LAAPD) showed no significant change (40.7 ± 6.5 mm vs 40.0 ± 5.7 mm, $p = 0.066$). Left ventricular ejection fraction (EF) was well preserved and showed no significant change (61.1 ± 5.7% vs 60.8 ± 5.4%, $p = 0.893$). Complete echocardiographic data are presented in Table 3.

Table 2. Postoperative hemodynamic and biochemical changes following LAAC.

Variable	Pre-operative	Post-operative	t/Z	p
BNP (pg/mL)	150.9 (77.8, 260.7)	316.2 (197.5, 466.8)	–5.937	<0.001
Aldosterone (ng/dL)	5.5 (3.0, 8.7)	2.5 (1.4, 3.4)	5.155	<0.001
Systolic BP (mmHg)	127.3 ± 12.3	116.9 ± 12.4	5.949	<0.001
Diastolic BP (mmHg)	80.7 ± 7.7	67.4 ± 9.3	12.497	<0.001
Serum sodium (mmol/L)	142.0 ± 2.5	137.6 ± 2.8	12.992	<0.001
Serum potassium (mmol/L)	3.9 ± 0.4	4.1 ± 0.3	–4.207	<0.001

Note: Data, mean ± SD or median (IQR); BP, blood pressure; BNP, B-type natriuretic peptide; LAAC, LAA clipping.

Table 3. Cardiac function before and after LAAC.

Transthoracic echocardiography parameters	Pre-operative	Discharge	t	p
LVEDD (mm)	46.3 ± 4.5	46.2 ± 4.5	0.484	0.629
LAAPD (mm)	40.7 ± 6.5	40.0 ± 5.7	1.864	0.066
EF (%)	61.1 ± 5.7	60.8 ± 5.4	0.135	0.893

Note: Data, mean ± SD; LVEDD, left ventricular end-diastolic diameter; LAAPD, left atrial anteroposterior diameter; EF, ejection fraction.

3.4 Comparison of Pre- and Post-LAAC Medication

Pharmacological analysis demonstrated significant postoperative reductions in the utilization of ACEI/ARB (33.3% to 13.1%, p = 0.001) and CCB (29.2% to 16.1%, p = 0.027) after 7 days (Table 4). The use of anticoagulants (e.g., warfarin and DOACs), antiplatelets (e.g., aspirin and aspirin+clopidogrel), and amiodarone regimens remained largely unchanged (all $p > 0.05$). The use of beta-blockers showed no significant change (64.6% to 63.6%, p = 0.882). A total of 97 patients were followed up for 6 months. The use of ACEI/ARB at 6 months was significantly less than pre-operatively (15.5% vs 33.3%, p = 0.005), whereas CCB use was not significantly different (20.6% vs 29.2%, p = 0.161). The use of other medications showed no significant changes compared to preoperative levels. These findings suggest that targeted de-escalation of antihypertensive agents may partially account for the observed hemodynamic alterations.

3.5 Binary Logistic Regression Analysis

Binary logistic regression analysis was performed to identify independent factors associated with changes in blood pressure. The Hosmer-Lemeshow test revealed the model had a good fit to the data (χ^2 = 7.391, df = 8, p = 0.495). Elevated plasma BNP was a significant independent predictor of reduced blood pressure (adjusted OR = 0.124, 95% CI: 0.035–0.443, p = 0.001), with an inverse association. Elevated aldosterone was also a significant predictor of altered blood pressure (adjusted OR = 5.254, 95% CI: 1.333–20.711, p = 0.018). In contrast, sex, age, adjustment of antihypertensive medication, and history of hypertension were not statistically significant predictors of blood pressure changes.

3.6 Blood Pressure Trends and Follow-up

The dynamic changes in blood pressure following the LAAC procedure are summarized in Fig. 1. A clear postoperative reduction in both systolic and diastolic blood pressure was observed. The trend analysis for systolic blood pressure (SBP) revealed a nadir on postoperative day 3 (POD3), while the peak decline in diastolic blood pressure (DBP) occurred earlier on postoperative day 1 (POD1). Statistical analysis using repeated measures ANOVA further confirmed these trends, as illustrated by the plots of the estimated marginal means. Critically, the follow-up data at 6 months demonstrated a persistent and statistically significant reduction in both SBP and DBP compared to preoperative baseline levels ($p < 0.001$).

4. Discussion

The principal thromboembolic complications of AF stem from left atrial thrombus formation and subsequent systemic embolization, manifesting as cerebral infarction, peripheral arterial embolism, or visceral organ ischemia [13]. Current pharmacological prophylaxis relies on anticoagulant therapy, including warfarin and DOACs such as rivaroxaban and edoxaban [14,15]. However, patients with an elevated risk of bleeding (HAS-BLED score >3) face significant hemorrhagic complications with anticoagulation, necessitating alternative mechanical approaches for thromboembolic prevention [16].

Hemodynamic studies have shown that approximately 90% of left atrial thrombi originate in the LAA due to blood stasis during AF. This pathophysiological mechanism supports LAA closure as a targeted intervention, where physical isolation of the appendage from the circulatory system achieves thromboprophylaxis [17]. Totally thoracoscopic LAAC represents a minimally invasive surgical technique

Table 4. Pre-and post-LAAC medication profiles.

Medication Class	Pre-op (n = 99)	Post-op day 7 (n = 99) / 6 months (n = 97)	χ^2	*p*
Anticoagulants				
Warfarin	4 (4.0%)	0 (0%)	4.082	0.121
		0 (0%)	4.001	0.120
DOACs	50 (50.5%)	49 (49.5%)	0.020	0.887
		49 (50.5%)	0.000	0.999
Antiplatelets				
Aspirin	49 (49.4%)	44 (44.4%)	0.507	0.476
		43 (44.3%)	0.525	0.478
Aspirin+Clopidogrel	6 (6.1%)	1 (1.0%)	3.702	0.118
		1 (1.0%)	3.599	0.116
Antihypertensives				
ACEI/ARB	33 (33.3%)	13 (13.1%)	11.327	0.001
		15 (15.5%)	8.460	0.005
Beta-blockers	64 (64.6%)	63 (63.6%)	0.022	0.882
		61 (62.8%)	0.066	0.880
CCB	29 (29.2%)	16 (16.1%)	4.860	0.027
		20 (20.6%)	1.966	0.161
Others				
Amiodarone	18 (18.1%)	19 (19.1%)	0.033	0.855
		17 (17.5%)	0.014	0.905

Note: Data presented as n (%); DOACs, new oral anticoagulants; ACEI, Angiotensin Converting Enzyme Inhibitor; ARB, Angiotensin Receptor Blocker; CCB, Calcium Channel Blocker.

that results in complete anatomical exclusion of the LAA, while avoiding the morbidity associated with open cardiac procedures [18].

Our findings align with previous research by Turagam *et al.* [12], who compared 247 patients undergoing LAAC with 124 patients receiving transcatheter left atrial appendage occlusion (LAAO). These authors found a significant postoperative decrease in blood pressure in the LAAC group compared to the LAAO group, which persisted for more than a year [12]. Similarly, Maybrook *et al.* [19] observed a postoperative decrease in blood pressure in a cohort of 76 patients undergoing percutaneous LAA ligation. Both authors postulated that BNP may be a key regulatory mediator in the reduction of blood pressure, although neither investigated the specific underlying mechanisms. The LAA possesses endocrine function, with its primary secretory product being BNP. In humans, the majority of BNP is derived from atrial and ventricular tissues. When atrial pressure increases, the appendage releases BNP into the bloodstream to regulate water and sodium metabolism. Elevated BNP levels inhibit aldosterone production, leading to reduced levels of systemic aldosterone [20]. Decreased aldosterone levels subsequently promote sodium retention, water reabsorption, and potassium excretion, leading to enhanced sodium excretion, increased urine output, and reduced blood volume, potentially contributing to lower blood pressure [21]. In addition, the vasodilatory effects of BNP can further reduce blood pressure [22]. The proposed mechanism for LAAC-induced blood pressure reduction is shown in Fig. 2.

The current study found that BNP levels increased significantly after LAA clip placement (150.9 [IQR 77.8, 260.7] vs. 316.2 [IQR 197.5, 466.8] pg/mL, $p < 0.001$), possibly due to the blunt clipping effect of the device causing mechanical compression of the appendage tissue and inducing the release of stored BNP into the circulation. BNP may also be released through degranulation following ischemic necrosis of the LAA. Over time, the clipped LAA undergoes progressive atrophy and fibrosis, and the BNP-releasing effect gradually subsides [23]. In contrast, postoperative BNP elevation was not observed in patients undergoing LAA surgical amputation [24], since complete resection of the appendage using a surgical stapler prevents the release of BNP contained within the LAA tissue into the systemic circulation. Unlike transcatheter LAA occlusion, no elevation in BNP level was observed following implantation of the LAA occlusion device [25]. This is probably because the occluder blocks the appendage orifice without compressing the appendage tissue itself. Our study observed BNP elevation rather than LAA occlusion. This may be attributed to the significant clamping force applied by the LAA clip at the appendage base, causing a different impact on appendage exclusion than that achieved by occlusion devices.

BNP has been shown to decrease the expression of aldosterone synthase mRNA, thus reducing the production of

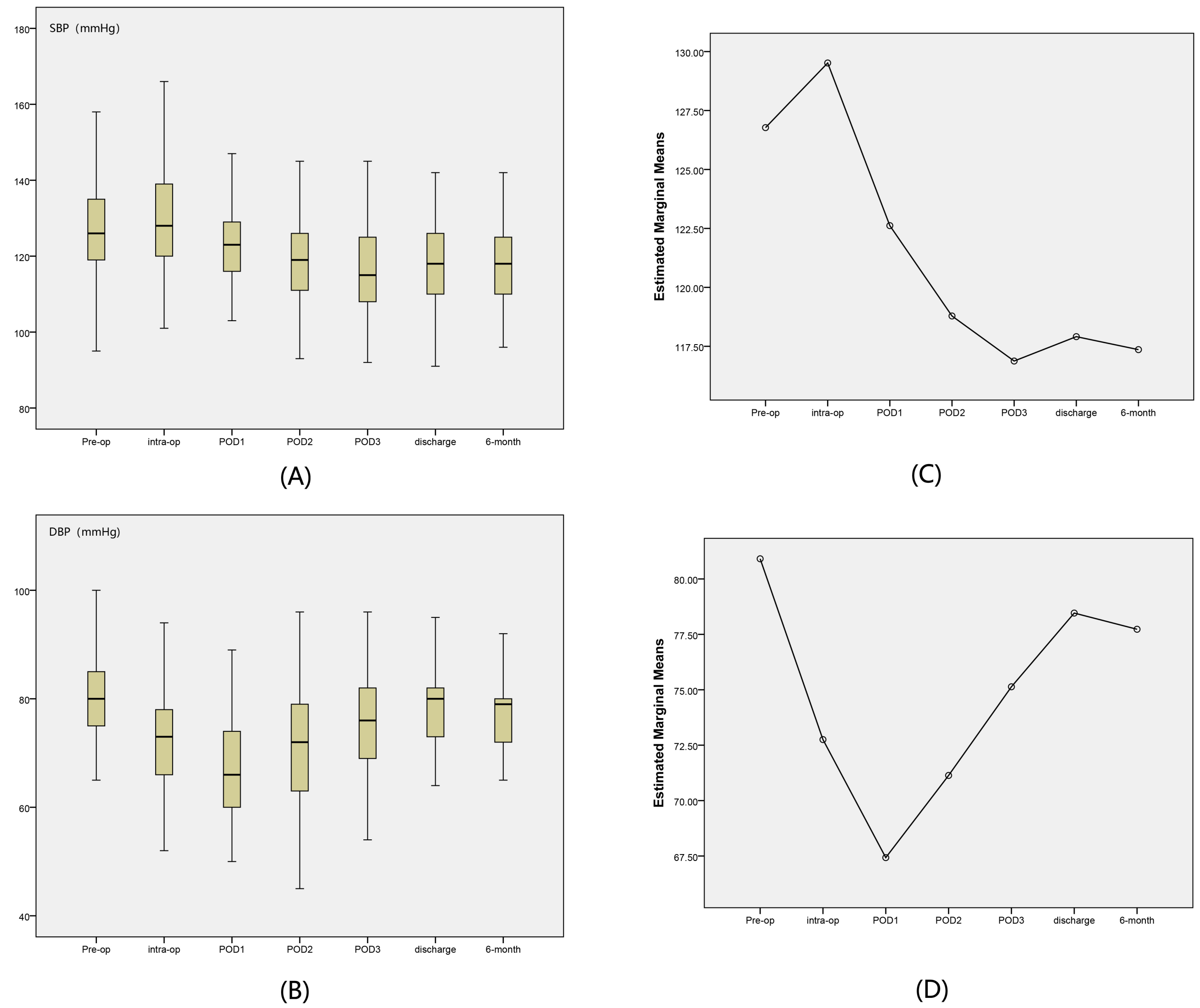

Fig. 1. Blood pressure dynamics before and after LAAC. (A) Trend for SBP, showing peak SBP reduction at POD3. (B) Trend for DBP, showing peak decline in DBP at POD1. (C) Estimated marginal means for SBP from repeated measures ANOVA. (D) Estimated marginal means for DBP from repeated measures ANOVA. Follow-up to 6 months showed that both SBP and DBP were significantly lower than preoperative levels ($p < 0.001$). SBP, systolic blood pressure; DBP, diastolic blood pressure; POD1, postoperative day 1.

aldosterone [26]. In the present study, the increase in BNP led to a reduction in aldosterone (5.5 [IQR 3.0, 8.7] vs. 2.5 [IQR 1.4, 3.4] ng/dL, $p < 0.001$), thereby promoting natriuresis and diuresis and likely contributing to the observed decrease in serum sodium concentration (142.0 $\pm$ 2.5 vs. 137.6 $\pm$ 2.8 mmol/L, $p < 0.001$). Since aldosterone also has antidiuretic effects, its decrease promoted diuresis, thereby reducing the effective blood volume and possibly explaining the observed decline in postoperative blood pressure (SBP: 127.3 $\pm$ 12.3 vs. 116.9 $\pm$ 12.4 mmHg; DBP: 80.7 $\pm$ 7.7 vs. 67.4 $\pm$ 9.3 mmHg; $p < 0.001$).

The significant postoperative reductions in the use of ACEI/ARB (33.3% to 13.1%, $p = 0.001$) and CCB (29.2% to 16.1%, $p = 0.027$) likely reflect the proactive management of LAAC-induced hemodynamic alterations by clinicians. However, our binary logistic regression analysis indicated that adjustments in these antihypertensive medications were not independent predictors of reduced blood pressure. Given the association of the LAAC procedure with BNP-mediated volume depletion and vasodilation, continued administration of antihypertensive agents could risk potentiating severe hypotension. Conversely, the continued use of beta-blockers (64.6% to 63.6%, $p = 0.882$) prioritizes ventricular rate control without exacerbating a reduction in vascular tone. These coordinated adjustments in medication represent a meticulous clinical response to LAAC-induced hypotension effects. At the 6-month follow-up, the use of ACEI/ARB remained lower than the preoperative level, while the use of CCB approached preoperative rates. Collectively, this demonstrates a reduced intensity of antihypertensive medication compared with the preoperative period.

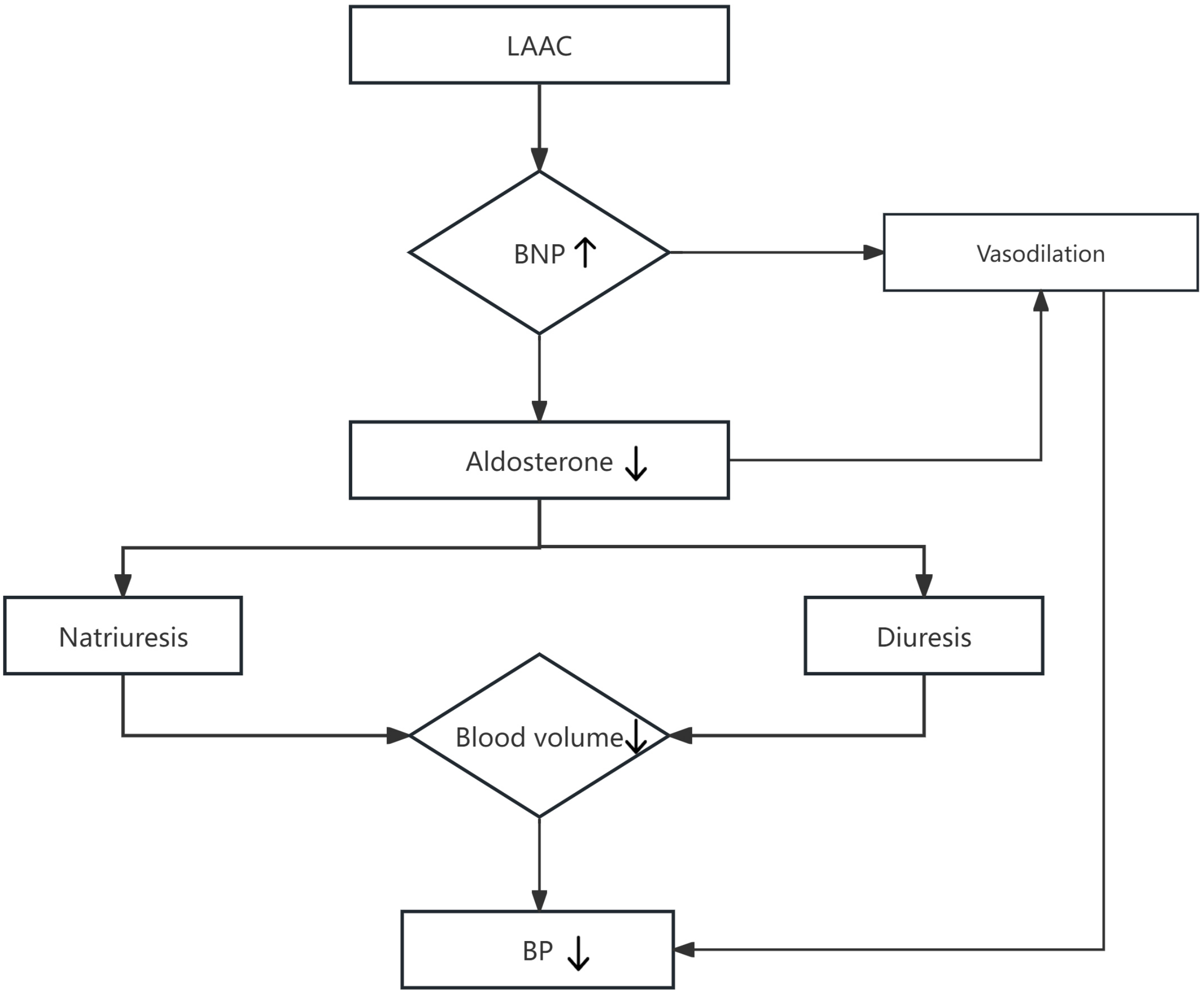

Fig. 2. Potential mechanistic pathway for LAAC-induced reduction of blood pressure. Down arrows: After LAAC, LAA releases BNP into the bloodstream. Elevated BNP promotes vasodilation and inhibits aldosterone production. The decrease in aldosterone levels subsequently promotes natriuresis and diuresis, leading to reduced blood volume and ultimately contributing to lower blood pressure.

Furthermore, LAAC did not significantly alter cardiac structure [27], as evidenced by unchanged left ventricular end-diastolic diameter, left atrial anteroposterior diameter, and left ventricular ejection fraction compared to preoperative measurements. These findings suggest that LAAC does not impair cardiac systolic function or left atrial architecture, indicating the observed decrease in blood pressure is unlikely to be related to cardiac functional changes.

The hemodynamic alterations following LAAC were characterized by a distinct pattern of blood pressure changes. The most pronounced reduction in SBP was observed on postoperative day 3. Although a partial recovery was noted by the time of discharge, SBP levels remained lower than preoperative baseline values. In contrast, the peak decline in DBP occurred earlier, on postoperative day 1, with DBP returning to near-baseline levels upon discharge. Severe hypotension, defined as SBP <90 mmHg requiring vasoactive medication support (e.g., dopamine, norepinephrine), occurred in 5 (5.1%) patients. Fortunately, the majority of these episodes were transient and not associated with significant adverse clinical outcomes. The impact of LAAC on blood pressure demonstrated a degree of persistence. At the 6-month follow-up, both SBP and DBP remained lower than their respective preoperative levels. This sustained effect was further reflected in the reduced intensity of antihypertensive medication regimens. Specifically, the use of ACEI/ARB was lower compared to the preoperative period, and adjustments in antihypertensive therapy were consistent with the observed decline in blood pressure. It is important to note that while our results and those of others [12] suggest the effects are potentially sustained, the lack of long-term follow-up of our cohort precludes definitive conclusions about the durability of this hypotensive response. Whether this effect is transient or long-lasting is a critical question that should be addressed by future studies with longer follow-up.

The male predominance of our cohort aligns with the epidemiology of AF in older patients undergoing invasive interventions, but limits the generalizability of the results to women [28]. Moreover, our cohort had a high proportion of patients with a history of stroke, which is characteristic of the elevated thromboembolic and bleeding risk profile typical of candidates for LAAC.

We acknowledge that the hemodynamic changes observed in this study are likely to be multifactorial. The significant postoperative reduction in ACEI/ARB and CCB use is undoubtedly due to the decline in blood pressure. However, the rapid and substantial rise in BNP and fall in aldosterone occurred in the immediate postoperative period, temporally preceding or coinciding with adjustments in medication. This sequence suggests that hormonal changes may be the primary instigator, with drug de-escalation representing a prudent clinical response to the observed hemodynamic shifts. Furthermore, other perioperative factors, including anesthesia, fluid balance, and the physiological stress response to surgery, may have contributed to the initial changes. While our study design cannot definitively isolate the effect of LAAC from all potential confounders, the strong and consistent hormonal and electrolyte pattern strongly supports a specific role for LAA compression.

5. Limitations

Our study has several limitations that should be considered. Its retrospective, single-center design and modest sample size may limit the generalizability of the findings and introduce potential for selection bias. The lack of long-term follow-up data prevents assessment of whether the hypotensive effect was sustained. Although we have proposed a BNP-aldosterone mediated mechanism, the absence of data on other RAAS components (e.g., renin, angiotensin II) means the hormonal cascade cannot be fully characterized. Finally, the potential influence of unmeasured confounders cannot be entirely ruled out, despite the compelling hormonal data. Taken together, current evidence suggests that LAAC may influence blood pressure through BNP-mediated RAAS suppression, however its precise mechanism requires validation through multicenter controlled studies.

6. Conclusions

Our findings indicate that LAAC is associated with a significant postoperative decline in blood pressure. The observed increase in BNP level and concurrent decrease in aldosterone suggest a potential mechanism involving BNP-mediated natriuresis and vasodilation. While BNP elevation may contribute to this hypotensive effect, other perioperative factors could also play a role. Consequently, proactive management is often required, including de-escalation of antihypertensive medication and vigilance for severe hypotensive events in the postoperative period. Nevertheless, the increase in BNP level remains a potential contributing factor to the postoperative decrease in blood pressure after LAAC.

Abbreviations

AF, atrial fibrillation; LAA, left atrial appendage; LAAC, left atrial appendage clipping; BNP, B-type natriuretic peptide; LAAPD, left atrial anteroposterior diameter; LVEDD, left ventricular end-diastolic diameter; EF, ejection fraction; SBP, systolic blood pressure; DBP, diastolic blood pressure.

Availability of Data and Materials

The datasets used and analyzed during the current study are available from the corresponding author upon reasonable request.

Author Contributions

QY designed the research study and wrote the manuscript. FL contributed to the study design, data analysis, and manuscript writing and revision. HM was involved in data acquisition, data analysis, and manuscript writing and revision. DX contributed to data analysis and manuscript writing, and revision. All authors contributed to the critical revision of the manuscript for important intellectual content. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Review Board of Beijing Tiantan Hospital, Capital Medical University (No. KY2022-013-02). Written informed consent was obtained from all participating patients prior to data collection and analysis.

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Conflict of Interest

The authors declare no conflict of interest.

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