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Impact Of Prophylactic Postoperative Vasopressors On Outcomes Of Patients Undergoing Thoracic Endovascular Aortic Repair

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TITLE PAGE

Title: Impact Of Prophylactic Postoperative Vasopressors On Outcomes Of Patients Undergoing Thoracic Endovascular Aortic Repair

Short Title: Prophylactic Postoperative Vasopressors For TEVAR

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VQI Protocol Number: # 4348

Keywords: Thoracic endovascular aortic repair, spinal cord ischemia, prevention, hemodynamic augmentation, controlled hypertension, prophylactic vasopressors, mortality.

ARTICLE HIGHLIGHTS

Type of research: Retrospective review of prospectively collected Vascular Quality Initiative (VQI) data

Key findings: Prophylactic vasopressors used for the prevention of spinal cord ischemia in patients undergoing TEVAR are associated with increased odds of postoperative 30-day mortality (OR, 3.23, 95%CI: 2.58-4.04, p<0.001) and other adverse events.

Take home message: The role of prophylactic vasopressors during TEVAR should be re-evaluated, and the decision for its administration should be made on an individualized basis, considering comorbidities and risk factors.

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In this VQI-based study, prophylactic vasopressors used during TEVAR to prevent spinal cord ischemia are associated with increased odds of 30-day mortality (OR, 3.23, p<0.001) and other adverse events. The role of prophylactic vasopressors during TEVAR should be re-evaluated and administered on an individualized basis, considering comorbidities and risk factors.

ABSTRACT

Background:

Prophylactic postoperative vasopressors (PPV) are used to induce hemodynamic augmentation to prevent spinal cord ischemia (SCI) in patients undergoing thoracic endovascular aortic repair (TEVAR). However, the scientific evidence on its effectiveness and safety is limited. This study aims to investigate the safety and effectiveness of PPV in patients undergoing TEVAR in a multi-institutional real-world setting.

Methods:

All TEVAR patients in the Society for Vascular Surgery (SVS) Vascular Quality Initiative (VQI) database between March 1, 2012, and January 28, 2023, were identified. Univariable and multivariable analyses were performed to assess the association between the use of PPV and the rates of postoperative 30-day mortality, Major Adverse Cardiovascular Events (MACE, defined as new onset of postoperative myocardial infarction (MI), congestive heart failure (CHF), dysrhythmias, or stroke), SCI, and other adverse events for patients undergoing TEVAR.

Results:

Out of 25,549 reviewed patients, 11,342 underwent TEVAR, matching the study criteria, and were analyzed. 2,420 patients (21.3%) received PPV. Patients who received PPV had significantly higher rates of 30-day mortality (9.3% vs 2.1%, $p<0.001$), MACE (18.2% vs 7.4%, $p<0.001$), postoperative SCI (2.6% vs 1.3%, $p<0.001$), and other adverse events. After adjusting for confounders, the use of PPV was associated with significantly increased odds of 30-day mortality (OR, 3.23, 95%CI: 2.58-4.04, $p<0.001$), MACE (OR, 2.18, 95%CI: 1.89-2.51, $p<0.001$), postoperative SCI (OR, 1.46, 95%CI: 1.05-2.01, $p=0.022$) and other adverse events.

Conclusions:

PPV was associated with significantly higher odds of 30-day mortality, MACE, SCI, and other adverse outcomes. The role of PPV should be re-evaluated, and the decision for administration should be made on an individualized basis, considering patient comorbidities and risk factors. A prospective study is required to confirm these findings.

MANUSCRIPT

INTRODUCTION

Spinal cord ischemia (SCI) is one of the infrequent but devastating complications during thoracic endovascular aortic repair (TEVAR).[1–3] Several risk factors have been identified for SCI after TEVAR, such as left subclavian artery (LSA) coverage without antegrade flow preservation, parallel or previous abdominal aortic surgery, extensive thoracic aortic coverage, renal insufficiency, perioperative hypotension, and hypogastric artery coverage. [2,4,5] For such patients with high-risk features undergoing TEVAR, the Society for Vascular Surgery (SVS) practice guidelines recommend neuroprotective measures such as prophylactic controlled hypertension and cerebrospinal fluid drainage (CSFD) to optimize spinal cord perfusion.[6] Although prophylactic controlled hypertension with vasopressors is used in clinical practice, the scientific evidence on its effectiveness and safety is limited.

This study aims to investigate the safety and effectiveness of prophylactic postoperative vasopressors (PPV) during TEVAR in a real-world, multi-institutional setting.

METHODS

Database: This is a retrospective review of prospectively collected data for patients undergoing TEVAR in the SVS Vascular Quality Initiative (VQI) database. This database has robust documentation of demographic, clinical, procedural, and postoperative variables from over 1000 hospitals in the United States and Canada.[7] Centers participate in VQI voluntarily and provide the required variables for all consecutive cases of eligible procedures. Variables are extracted from medical records by trained personnel, and the database undergoes periodic source audits by the SVS Patient Safety Organization (PSO). Detailed information about the VQI is available at www.vqi.org. This study is exempt from Institutional Review Board (IRB) approval and patient

informed consent, as it has deidentified patient data. The study was approved by the SVS PSO Research Advisory Committee (RAC) and has protocol number #4348.

Inclusion and Exclusion Criteria: All patients who underwent TEVAR between March 1, 2012, and January 28, 2023, were reviewed. Patients with open conversion, proximal landing zone <2, distal landing zone >5, and spinal cord ischemia at presentation due to aortic dissection were excluded from the study.

Definition of the exposure variable: The exposure variable is defined based on an existing VQI variable, according to which PPV is the postoperative use of vasopressors in patients who do not have any clinical signs or symptoms of SCI at the time of the initiation of the vasopressors.

Baseline Variables: The baseline demographic, clinical, and intraoperative characteristics were compared between the two groups of the main exposure variable (Table I).

Coronary artery disease (CAD) was defined as a history of angina or myocardial infarction (MI). The New York Heart Association functional classification was used for the definition of congestive heart failure (CHF).[8] Cerebrovascular disease (CVD) is defined as a history of transient ischemic attacks or stroke. Smoking status was grouped into any history of smoking vs none. Bypass surgery is defined as any non-coronary, non-intracranial arterial bypass for arterial occlusive disease. Urgency was categorized into three levels: emergent (surgery within 4 hours of presentation), urgent (surgery within 4-24 hours of presentation), and elective (planned/scheduled procedure after 24 hours). The length of aortic coverage is defined as the number of aortic zones covered with stent-grafts during the TEVAR.

Outcomes: Outcomes were compared between patients who received PPV and those who did not. The primary outcome is postoperative 30-day mortality. Secondary outcomes are postoperative Major Adverse Cardiovascular Events (MACE, defined as new onset of

postoperative MI, dysrhythmia, stroke, or CHF), SCI, hemodialysis (HD), intestinal ischemia, overall complications, length of intensive care unit (ICU) stay, as well as the total length of stay (LOS). Postoperative SCI was defined as any new postoperative spinal cord ischemia, transient or permanent, manifested as leg weakness or paralysis. Postoperative HD was defined as the postoperative new onset of renal failure, requiring temporary or permanent HD, not present at admission. Bowel ischemia was defined as evidence of ischemia documented with colonoscopy, bloody stools with death before colonoscopy/laparotomy, or other documented clinical diagnosis.

Subgroup analysis was performed for the elective and non-elective (urgent or emergent) subgroups as a potential source of confounding.

To address the possible confounding of the results by postoperative conditions that may be complicated by hemodynamic compromise and hypotension having additional indications for postoperative vasopressor support (e.g., postoperative bleeding, MI, CHF, sepsis), a sensitivity analysis was conducted, excluding patients who developed postoperative MI, CHF, as well as postoperative respiratory complications (postoperative pneumonia or reintubation) or intestinal ischemia as potential major sources for postoperative sepsis. To address the confounding effect of perioperative bleeding, multivariable models were adjusted for perioperative (intraoperative and postoperative) use of total packed red blood cell (PRBC) units.

Statistical Analysis: Continuous variables are presented as average ± standard deviation (SD), whereas discrete variables are presented as numbers of cases, with percentages (%). Categorical baseline characteristics were compared between PPV groups using contingency table analysis with the Pearson $\chi 2$ and Fisher's exact tests. Continuous variables were compared between PPV groups using the Student's t-test for two independent samples. Univariable and multivariable regression (MR) analyses were conducted to study the association of PPV with the

endpoints. The selection of covariates for the multivariable models was performed using a stepwise backward regression, as well as considering the clinical significance of covariates. Clinically important variables that were not significantly associated with the endpoints in univariable analysis were forced into the multivariable models if the $p<0.25$, considering parsimony of the variable selection. Variation between centers was accounted for by clustering them into lower, middle, and upper tertiles based on the average annual volume of procedures performed by each center. Multilevel-mixed regression models were used to address the variations across the clusters. A sensitivity analysis was performed using the propensity score-adjusted regression (PSR) method. The propensity score was calculated based on the 48 baseline variables presented in Table I. The Hosmer-Lemeshow test was used to assess the goodness of fit of the regression model, and receiver-operating characteristic (ROC) curves with the area under the curve (AUC) were used to evaluate the discrimination capacity of the models. Unadjusted odds ratios (OR) and adjusted odds ratios (aOR) with 95% confidence intervals (CI) were reported for binary discrete outcomes. All statistical analysis was performed using Stata/BE, version 19.5, software (StataCorp LP, College Station, TX). A two-sided p-value <0.05 was considered statistically significant for the final report.

RESULTS

Out of the 25,549 reviewed patients in the VQI TEVAR/complex-EVAR module, 11,342 matched the study criteria and were included in the final analysis, of which 2,420 (21.3%) received PPV. The average age of patients was 63.3 ± 16.6 years, 6,982 (61.6%) were male, 7,324 (64.6%) were white, 2,660 (23.5%) were black, and 1,358 (12.0%) were other races. The distribution of baseline characteristics between patients who received PPV and those who did not is presented in Table I.

Outcomes

In univariable analysis, patients who received PPV had a significantly higher probability of overall 30-day mortality (9.3% vs 2.1%, p<0.001), MACE (18.2% vs 7.4%, p<0.001), postoperative SCI (2.6% vs 1.3%, p<0.001), and other adverse outcomes (Table II).

After adjusting for potential confounders, the use of PPV was associated with significantly higher odds of overall 30-day mortality (aOR: 3.23, 95%CI: 2.58-4.04, p<0.001), MACE (aOR: 2.18, 95%CI: 1.89-2.51, p<0.001), postoperative SCI (OR: 1.46, 95%CI: 1.05-2.01, p=0.022), and other adverse outcomes (Table III).

Subgroup analysis

In the elective subgroup, patients who received PPV had a significantly higher rates of overall 30-day mortality (3.6% vs 0.8%, p<0.001), MACE (13.1% vs 5.6%, p<0.001), and SCI (1.8% vs 0.9 %, p=0.007). After adjustment for confounders, PPV was associated with higher odds of overall 30-day mortality (aOR: 3.68, 95%CI: 2.32-5.84, p<0.001), MACE (aOR: 2.05, 95%CI: 1.66-2.53, p<0.001), and a tendency towards a statistically significantly higher odds of SCI (aOR: 1.55, 95%CI: 0.92-2.61, p=0.096) (Table IV).

In the non-elective subgroup, patients who received PPV had a significantly higher rate of overall 30-day mortality (15.7% vs 4.1%, p<0.001), MACE (23.8% vs 10.1%, p<0.001), and SCI (3.4% vs 1.9%, p=0.007). After adjustment for confounders, PPV was associated with higher odds of overall 30-day mortality (aOR: 3.12, 95%CI: 2.41-4.04, p<0.001) and MACE (aOR: 2.28, 95%CI: 1.89-2.75, p<0.001), but not SCI (aOR: 1.40, 95%CI: 0.92-2.11, p=0.115) (Table V).

Sensitivity analysis

PSR yielded similar results to the main group analysis for all outcomes, except for postoperative CHF, which approached statistical significance (aOR: 1.45, 95% CI: 0.96-2.18, p = 0.075) (Table III).

After excluding those patients who developed postoperative MI (142 patients, 1.2%), CHF (112 patients, 1.0%), respiratory complications (824 patients, 7.3%), or intestinal ischemia (110 patients, 1.0%), 10,302 patients were available for the sensitivity analysis. The results of the univariable and multivariable analyses are similar to the main group analysis (Supplemental Table I).

DISCUSSION

In this real-world, multi-institutional retrospective cohort study, the use of PPV for patients undergoing TEVAR is associated with increased odds of overall 30-day mortality, MACE, and other adverse outcomes (Table III).

Spinal cord ischemia is one of the most devastating complications of thoracic and thoracoabdominal aortic repair, both open and endovascular.[1–3] Increasing the spinal cord perfusion pressure (SCPP) through either increasing the systemic mean arterial pressure (MAP) or decreasing the spinal pressure (SP) is a key strategy for both prevention and treatment of SCI during thoracic or thoracoabdominal aortic repair (SCPP=MAP-SP).[9] Intraoperative adjuncts, such as distal aortic perfusion and cerebrospinal fluid drainage (CSFD), have been used in open aortic repair to mainly lower the incidence of immediate spinal cord ischemia, without significantly impacting the risk of SCI in the delayed setting.[10–15] Hypotensive episodes, along with CSFD complications, were identified as major risk factors for delayed spinal cord injury.[15,16] With the introduction of TEVAR, the pattern of SCI changed, with the majority of cases occurring in the delayed setting.[2,17] Although TEVAR has dramatically lowered the incidence of hypotensive

episodes in the perioperative setting compared to open repair, preceding episodes of hypotension are still risk factors for the development of SCI after TEVAR, along with other risk factors for SCI, justifying the need to increase SCPP for SCI prevention.[18]

The SVS clinical practice guidelines of thoracic endovascular aortic repair for descending thoracic aortic aneurysms recommend the preventive use of both controlled hypertension and CSFD for TEVAR patients, who have a high risk for SCI.[6] However, in real-world practice, there is a variation of SCI prevention protocols among centers based on institutional experience.[2,19–22] In the survey completed by the eight principal investigators of the U.S. Aortic Research Consortium (ARC), capturing the current practices and management strategies related to the prevention and treatment of SCI during endovascular aortic repair, hemodynamic augmentation was the most frequently recommended preventive measure (7 out of 8 investigators, 87.5%) in high-risk patients, with a MAP goal above 90 mm Hg (5 out of 7 investigators, 71%), followed by prophylactic spinal drains (6 out of 8 investigators, 75%).[23] The concern that spinal drains may cause as many neurological complications as they potentially prevent has advocated for limiting the use of prophylactic CSFD only to high-risk patients and to the therapeutic setting for those who have already developed clinical signs of SCI.[15,24-27] This emphasizes the important role of controlled hypertension for SCI prevention during endovascular aortic repair, although the safety and efficacy of prophylactic vasopressors for the prevention of SCI have not been widely studied.[28,29]

The current study attempts to fill this gap in the literature, showing that PPV for the prevention of SCI is associated with increased odds of overall 30-day mortality and other adverse outcomes for patients undergoing TEVAR (Table III).

Although the patients who received a PPV may be overall "sicker" than those who did not receive a PPV (Table I), the multivariable models were adjusted for all potential confounders, including ones that are expected to correlate with clinical severity, such as ASA class, presentation, urgency, EBL, total (intraoperative and postoperative) PRBC units, the volume of crystalloids, etc. The results were confirmed in the elective and non-elective subgroups (Tables IV, V), as well as further corroborated in the PSR models (Table III) and the sensitivity analysis (Supplemental Table I). The effect size of the association of PPV with the outcome variables was at least moderate.[30] These findings suggest that PPV for the prevention of SCI may be independently associated with the adverse outcomes for patients undergoing TEVAR.

These findings align with some previous reports, which demonstrated that hemodynamic augmentation with vasopressors may cause dose-dependent adverse clinical outcomes, such as increased mortality, end-organ ischemic events, such as MI, stroke, worsening of the spinal cord ischemia, and renal failure.[26,31,32] Inoue et al. found that the use of Dopamine and age over 65 are both strong independent predictors of mortality in patients receiving hemodynamic augmentation for spinal cord injury, without any benefit for improvement of the spinal cord status.[33] In the same study, 74% of patients who received a pressor developed some pressor-associated complication, such as ventricular tachycardias, ST-segment elevation, troponin elevation, and the incidence of these complications reached 84% in patients above 60 years of age.[33] Auchet et al. demonstrated that the dose of vasopressors is a strong independent predictor for patient mortality.[34] Sandhu et al. found that 90% of delayed paraplegia after the repair of thoracic and thoracoabdominal aortic aneurysm occurs in patients with systolic blood pressure (SBP) lower than 130 mm Hg, however, controlled hypertension with an SBP higher than 150 mm Hg is associated with significantly higher rates of pressor-induced complications such as end-organ ischemia, intracranial bleeding,

and organ failure.[26] This study not only demonstrates the role of preceding hypotensive episodes in the development of SCI and that pressor-induced hypertension is associated with adverse patient outcomes, but it also suggests that there may be a safe blood pressure window (SBP, 130 - 150 mm Hg) where the risk of SCI and vasopressor-associated adverse effects are both relatively low. High blood pressure was associated with increased rates of in-hospital cardiovascular morbidities and mortality in surgical and non-surgical patients in other studies, too.[35–42] Several studies demonstrated a U-shaped association between blood pressure and in-hospital cardiovascular morbidities and mortality, suggesting that there may be an optimal blood pressure range with lower rates of adverse cardiovascular outcomes.[43–45] The association of PPV with increased odds of overall MACE and all of its components (Table III) highlights the significant role of cardiovascular mechanisms in the adverse sequelae of vasopressors, aligning with previous reports.[26,31,32] Specifically, vasopressors increase systemic vascular resistance, cardiac afterload, and, in the case of vasopressors with cardiotropic effects, also increase cardiac contractility and heart rate, all of which result in increased oxygen demand of cardiac muscle cells, cardiac ischemia, and compromised perfusion of other organs.[46,47]

Earlier studies demonstrated that patient age and comorbidities may modify the association of PPV with adverse cardiovascular events, and patients over 65 receiving hemodynamic augmentation experienced significantly higher rates of cardiovascular morbidity, highlighting the role of limited physiologic reserve for tolerating excessive vasoconstriction.[32,33]

An interesting finding of the current study is the association of PPV with an increased rate of postoperative SCI (2.6% vs 1.3%, $p<0.001$) instead of preventing this complication (Table II). Although patients who receive a PPV may have a higher baseline risk of developing SCI based on the indications for PPV, after adjusting for confounders, including variables that define the high

baseline risk for SCI development, PPV is still associated with an increased risk of SCI in both MR and PSR models (Table III), as well as in the sensitivity analysis (Supplemental Table I). However, this association was not statistically significant in the non-elective subgroup, whereas PPV was associated with a tendency towards higher odds of SCI in the elective subgroup (Tables IV, V). This suggests that, in some circumstances, prophylactic vasopressors may have an independent association with an increased risk of postoperative SCI. These findings align with previous reports, demonstrating that hemodynamic augmentation may be associated with an increased risk of the development of SCI.[26,31–33] There was also a dose-dependent association between arterial blood pressure and the incidence of SCI in experimental animal studies, demonstrating that avoiding hypotension with mild-to-moderate hemodynamic augmentation can prevent SCI,[48,49,] but excessively high blood pressures are associated with an increased risk of the development of spinal cord injury.[50] Many of the institutional MAP goal protocols for TEVAR have a clear lower arterial blood pressure cut-off (MAP > 90 mm Hg) but do not have a clear upper cut-off, which may result in excessive hemodynamic augmentations.[23]

This suggests that the role of prophylactic vasopressors may need to be reevaluated. Patients may significantly benefit from the prevention of excessive hemodynamic augmentation with lower and narrower MAP goals, using lower doses of vasopressors with relatively safe profiles, closer arterial blood pressure monitoring, which may protect the spinal cord as well as cause less postoperative cardiovascular morbidities and mortality, especially for patients who already have compromised cardiovascular reserve. Further prospective studies are required to test this hypothesis, but the results of the current study demonstrate that in the real-world multi-institutional setting, the use of PPV for the prevention of SCI in patients undergoing TEVAR is

associated with increased rates of postoperative adverse outcomes, including 30-day mortality, MACE, and SCI.

Limitations

This is a retrospective study, and it should be expected to have limitations such as confounding due to non-random allocation of patients and due to indication, as well as residual and unmeasured confounding. VQI is a multi-institutional database and uses trained personnel for data entry and extensive data auditing, but human errors, missing data, and coding errors are still unavoidable. The VQI does not document the used vasopressors, their doses, or the duration of their use. It also does not capture the specific indication for vasopressor use. Instead, it categorizes vasopressor administration as prophylactic or therapeutic based on the presence or absence of SCI at the time of initiation. However, within the context of SCI prevention during TEVAR, vasopressors initiated even for hemodynamic instability in the absence of SCI are still considered prophylactic for spinal cord protection, which is the very focus of the current study. Patients who received PPV may theoretically have concomitant indications for vasopressor initiation (postoperative bleeding, MI, CHF, sepsis). However, the sensitivity analysis demonstrated that postoperative complications associated with hemodynamic instability and serving as an additional indication for vasopressors were only in a small percentage of patients, and after excluding these patients, the results were unchanged. This suggests that patients who started receiving postoperative vasopressors in the absence of SCI at the time of vasopressor initiation, defined as prophylactic, were not likely to have another existing indication, other than hemodynamic augmentation for TEVAR. The VQI does not document postoperative sepsis as an outcome variable, and postoperative respiratory complications (pneumonia or reintubation) and intestinal ischemia were used as major sources of postoperative sepsis in the sensitivity analysis. Another

source of sepsis may be postoperative surgical site infections (SSIs), but VQI started documenting access-site infections (superficial, deep, organ/space SSI) in 2020, and only two cases with access-site infections were identified in the current study (1 case with superficial and 1 case with deep SSI) from 2020 to 2023. Therefore, extrapolating the number of postoperative SSI cases on the whole study sample (between March 1, 2012, and January 28, 2023), sepsis caused by SSI is not expected to be a significant source of confounding in the current study.

CONCLUSIONS

In the real-world multi-institutional setting, the use of prophylactic vasopressors for the prevention of spinal cord ischemia in patients undergoing TEVAR is associated with increased rates of overall 30-day mortality, MACE, SCI, and other adverse effects. The role of prophylactic vasopressors during TEVAR should be reevaluated, and the decision for their administration should be made on an individualized basis, considering patient age and comorbidities. Stricter monitoring of perioperative blood pressure and narrower MAP goals to avoid both hypotensive episodes and excessive vasoconstriction may be appropriate, especially for patients with a decreased cardiovascular reserve, although the exact MAP goals are less clear. Future prospective trials are required to confirm these findings.

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Table I. Baseline patient characteristics per PPV status

		PPV	No PPV	
		N = 2,420	N = 8,922	P-value
Demographic characteristics				
Age, years		64.1 ± 16.4	63.1 ± 16.6	0.007
Gender, male		1,448 (59.8%)	5,534 (62.0%)	0.049
Ethnicity, Hispanic or Latino		147 (6.1%)	612 (6.9%)	0.170
Race	White	1,576 (65.1%)	5,748 (64.4%)	0.476
	Black	546 (22.6%)	2,114 (23.7%)	
	Other	298 (12.3%)	1,060 (11.9%)	
Height, cm		171.4 ± 11.3	171.7 ± 11.3	0.152
Weight, kg		84.5 ± 22.4	84.1 ± 23.8	0.456
Medical history				
CAD		338 (14.0%)	1,277 (14.3%)	0.666
CHF		304 (12.6%)	940 (10.5%)	0.005
CVD		267 (11.0%)	875 (9.8%)	0.076
HTN		1,961 (81.0%)	7,217 (80.9%)	0.874
DM		351 (14.5%)	1,319 (14.8%)	0.731
HD		55 (2.3%)	210 (2.4%)	0.815
Smoking		1,547 (63.9%)	5,901 (66.1%)	0.042
COPD		549 (22.7%)	2,027 (22.7%)	0.972
EF < 50 %		240 (9.9%)	745 (8.4%)	0.015
Decreased preop functional status		830 (34.3%)	2,593 (29.1%)	<0.001
Transferred from another hospital		967 (40.0%)	2,878 (32.3%)	<0.001
Preoperative hemoglobin, g/dl		11.8 ± 2.2	12.0 ± 2.1	<0.001
Preoperative creatinine, mg/dl		1.2 ± 0.8	1.1 ± 0.7	<0.001
Abnormal cardiac stress test		61 (2.5%)	213 (2.4%)	0.705
Surgical history				
History of bypass surgery		124 (5.1%)	454 (5.1%)	0.944
CABG		169 (7.0%)	571 (6.4%)	0.303
PCI		247 (10.2%)	835 (9.4%)	0.208
CEA or CAS		37 (1.5%)	167 (1.9%)	0.206
History of PVI		92 (3.8%)	302 (3.4%)	0.321
History of aortic surgery, open or endo		667 (27.6%)	1,904 (21.3%)	<0.001
Medications				
Aspirin		1,131 (46.7%)	4,097 (45.9%)	0.475
P2Y12 inhibitors		133 (5.5%)	610 (6.8%)	0.018
Statins		1,178 (48.7%)	4,392 (49.2%)	0.632
Beta blockers		1,646 (68.0%)	5,911 (66.2%)	0.103
ACE inhibitors		867 (35.8%)	3,408 (38.2%)	0.033
Anticoagulants		296 (12.2%)	1,070 (12.0%)	0.749
Aortic disease/intervention related characteristics				
Aortic pathology	Aneurysm	736 (30.4%)	3,093 (34.7%)	<0.001

	Dissection	937 (38.7%)	3,081 (34.5%)	
	Other*	747 (30.9%)	2,748 (30.8%)	
Maximal aortic diameter, mm		50.2 ± 16.4	48.9 ± 16.7	0.010
Presentation	Ruptured	263 (10.9%)	558 (6.2%)	<0.001
	Symptomatic, not ruptured	1,277 (52.8%)	4,313 (48.3%)	
	Asymptomatic	880 (36.4 %)	4,051 (45.4%)	
Urgency	Emergent	580 (24.0%)	1402 (15.7%)	<0.001
	Urgent	576 (23.8%)	2,093 (23.5%)	
	Elective	1,264 (52.2%)	5,427 (60.8%)	
Anesthesia, general		2,350 (97.1%)	8,669 (97.2%)	0.881
ASA Class >3		1,701 (70.3%)	5,257 (58.9%)	<0.001
Intraoperative characteristics				
Contrast, ml		114.0 ± 76.2	100.5 ± 70.4	<0.001
Crystalloids, ml		1,740.1 ± 1,228.1	1,619.0 ± 1,044.7	<0.001
EBL, ml		272.3 ± 568.1	185.1 ± 509.3	<0.001
Fluoroscopy time, min		24.7 ± 27.4	22.5 ± 26.7	<0.001
Total PRBC, units		3.8 ± 8.3	1.5 ± 7.0	<0.001
Total procedure time, min		138.6 ± 98.6	123.2 ± 85.8	<0.001
Application of IVUS or TTE		1,524 (63.0%)	4,654 (52.2%)	<0.001
Number of aortic stent-grafts		1.7 ± 0.8	1.6 ± 0.7	<0.001
Proximal landing zone, zone 2		966 (39.9%)	3,120 (35.0%)	<0.001
Distal landing zone, zone 5		1,676 (69.3%)	5,531 (62.0%)	<0.001
Length of aortic coverage, zones		1.9 ± 0.8	1.8 ± .08	<0.001
Perioperative prophylactic spinal drain		939 (38.8%)	2,773 (31.1%)	<0.001
Center operative volume, tertiles	Lower	698 (28.8%)	3,139 (35.2%)	<0.001
	Middle	912 (37.7%)	2,988 (33.5%)	
	Upper	810 (33.5%)	2,795 (31.3%)	

Discrete variables presented as number of cases (%); continuous variables presented as mean ± standard deviation; PPV, prophylactic postoperative vasopressors; CAD, coronary artery disease; CHF, congestive heart failure; CVD, cerebrovascular disease; HTN, hypertension; DM, diabetes mellitus; HD, hemodialysis; COPD, chronic obstructive pulmonary disease; EF, ejection fraction; CABG, coronary artery bypass graft; PCI, percutaneous coronary intervention; CEA, carotid endarterectomy; CAS, carotid artery stenting; PVI, peripheral vascular intervention; ACE, angiotensin-converting enzyme; ASA, American Society of Anesthesiologists; EBL, estimated blood loss; PRBC, packed red blood cells; IVUS, intravascular ultrasound; TTE, transthoracic echocardiography; *, other aortic pathologies include penetrating aortic ulcer (PAU), intramural hematoma (IMH), traumatic aortic injury, and aortic thrombus.

Table II. Univariable association of PPV with the outcome variables

		PPV	No PPV	
		N = 2,420	N = 8,922	P-value
30-day mortality		226 (9.3%)	184 (2.1%)	<0.001
MACE		441 (18.2%)	657 (7.4%)	<0.001
	MI	61 (2.5%)	81 (0.9%)	<0.001
	CHF	38 (1.6%)	74 (0.8%)	<0.001
	Dysrhythmias	293 (12.1%)	387 (4.3%)	<0.001
	Stroke	134 (5.5%)	194 (2.2%)	<0.001
SCI		62 (2.6%)	118 (1.3%)	<0.001
HD		193 (8.0%)	308 (3.4%)	<0.001
Intestinal ischemia		75 (3.1%)	35 (0.4%)	<0.001
Overall complications		831 (34.3%)	1,337 (15.0%)	<0.001
Length of ICU stay		6.9 ± 10.0	3.6 ± 5.1	<0.001
Total LOS		15.7 ± 41.4	10.4 ± 35.7	<0.001

Discrete variables presented as number of cases (%); continuous variables presented as mean ± standard deviation; PPV, prophylactic postoperative vasopressors; MACE, Major Adverse Cardiovascular Events; MI, myocardial infarction; CHF, congestive heart failure; SCI, spinal cord ischemia; HD, hemodialysis; ICU, intensive care unit; LOS, length of stay.

Table III. Multivariable association of PPV with the outcome variables in MR and PSR models

		MR		PSR	
		aOR [95%CI]	**p-value**	**aOR [95%CI]**	**p-value**
30-day mortality		3.23 [2.58, 4.04]	<0.001	3.05 [2.48, 3.76]	<0.001
MACE		2.18 [1.89, 2.51]	<0.001	2.14 [1.87, 2.45]	<0.001
	MI	2.32 [1.64, 3.30]	<0.001	2.11 [1.49, 2.99]	<0.001
	CHF	1.59 [1.06, 2.41]	0.025	1.45 [0.96, 2.18]	0.075
	Dysrhythmias	2.39 [2.02, 2.83]	<0.001	2.32 [1.96, 2.73]	<0.001
	Stroke	2.16 [1.71, 2.74]	<0.001	2.03 [1.61, 2.57]	<0.001
SCI		1.46 [1.05, 2.01]	0.022	1.42 [1.03, 1.95]	0.032
HD		1.94 [1.58, 2.38]	<0.001	1.98 [1.63, 2.40]	<0.001
Intestinal ischemia		5.60 [3.66, 8.57]	<0.001	5.06 [3.34, 7.67]	<0.001
Overall complications		2.26 [2.02, 2.52]	<0.001	2.27 [2.04, 2.53]	<0.001
Length of ICU stay*		2.30 [2.03, 2.56]	<0.001	2.34 [2.05, 2.63]	<0.001
Total LOS*		3.83 [2.15, 5.55]	<0.001	3.92 [2.20, 5.63]	<0.001

PPV, prophylactic postoperative vasopressors; MR, multivariable regression; PSR, propensity score-adjusted regression; aOR, adjusted odds ratio; CI, confidence interval; *, for the length of ICU stay and total LOS, adjusted slope coefficients (aB1) of multivariable linear regression models are reported; MACE, Major Adverse Cardiovascular Events; MI, myocardial infarction; CHF, congestive heart failure; SCI, spinal cord ischemia; HD, hemodialysis; ICU, intensive care unit; LOS, length of stay.

MR models are adjusted for baseline demographic characteristics (age, gender, ethnicity, race, height, weight), medical history (CAD, DM, HD, smoking status, decreased preoperative functional status, transfer from another hospital), surgical history (aortic surgery, bypass surgery), medication history (Aspirin, Statin, ACE inhibitors, Betta blockers), aortic pathology, intraoperative characteristics (fluoroscopy time, total procedure time, contrast, EBL, crystalloids, total PRBC), presentation, urgency, aortic diameter, preoperative creatinine, preoperative hemoglobin, ASA class, perioperative prophylactic spinal drain, number of used aortic stent-grafts, length of aortic coverage, proximal landing zone (zone 2), distal landing zone (zone 5), and clustered by hospital volume tertiles.

Table IV. Univariable and multivariable association of PPV with the outcome variables in the elective subgroup

		Univariable			Multivariable	
		PPV (N=1,264)	**No PPV (N=5,427)**	**p-value**	**aOR [95%CI]**	**p-value**
30-day mortality		45 (3.6%)	41 (0.8%)	<0.001	3.68 [2.32, 5.84]	<0.001
MACE		166 (13.1%)	304 (5.6%)	<0.001	2.05 [1.66, 2.53]	<0.001
	MI	26 (2.1%)	30 (0.6%)	<0.001	3.40 [1.95, 5.92]	<0.001
	CHF	14 (1.1%)	35 (0.6%)	0.082	1.39 [0.73, 2.67]	0.317
	Dysrhythmias	105 (8.3%)	182 (3.4%)	<0.001	2.09 [1.61, 2.71]	<0.001
	Stroke	54 (4.3%)	88 (1.6%)	<0.001	2.40 [1.67, 3.45]	<0.001
SCI		23 (1.8%)	51 (0.9%)	0.007	1.55 [0.92, 2.61]	0.096
HD		55 (4.4%)	163 (3.0%)	0.015	1.30 [0.94, 1.81]	0.118
Intestinal ischemia		12 (1.0%)	12 (0.2%)	<0.001	3.07 [1.29, 7.30]	0.011
Overall complications		304 (24.0%)	571 (10.5%)	<0.001	2.16 [1.83, 2.55]	<0.001
Length of ICU stay*		4.3 ± 5.8	2.4 ± 3.5	<0.001	1.22 [0.99, 1.45]	<0.001
Total LOS*		12.5 ± 49.8	7.5 ± 18.3	<0.001	4.18 [2.50, 5.86]	<0.001

PPV, prophylactic postoperative vasopressors; aOR, adjusted odds ratio; CI, confidence interval; MACE, Major Adverse Cardiovascular Events; MI, myocardial infarction; CHF, congestive heart failure; SCI, spinal cord ischemia; HD, hemodialysis; ICU, intensive care unit; LOS, length of stay. *, for the length of ICU stay and total LOS, adjusted slope coefficients (aB1) of multivariable linear regression models are reported.

The multivariable models are adjusted for confounding effects of demographic, clinical, intraoperative characteristics, medications, aortic pathology, presentation, perioperative prophylactic spinal drain, number of used aortic stent-grafts, length of aortic coverage, proximal landing zone (zone 2), distal landing zone (zone 5), and clustered by hospital volume tertiles.

Table V. Univariable and multivariable association of PPV with the outcome variables in the non-elective subgroup

		Univariable			Multivariable	
		PPV (N=1,156)	**No PPV (N=3,495)**	**p-value**	**aOR [95%CI]**	**p-value**
30-day mortality		181 (15.7%)	143 (4.1%)	<0.001	3.12 [2.41, 4.04]	<0.001
MACE		275 (23.8%)	353 (10.1%)	<0.001	2.28 [1.89, 2.75]	<0.001
	MI	35 (3.0%)	51 (1.5%)	0.001	1.69 [1.07, 2.68]	0.024
	CHF	111 (9.6%)	278 (8.0%)	0.079	1.57 [0.92, 2.70]	0.100
	Dysrhythmias	188 (16.3%)	205 (5.9%)	<0.001	2.52 [2.01, 3.14]	<0.001
	Stroke	80 (6.9%)	106 (3.0%)	<0.001	2.00 [1.47, 2.73]	<0.001
SCI		39 (3.4%)	67 (1.9%)	0.004	1.40 [0.92, 2.12]	0.115
HD		138 (11.9%)	145 (4.2%)	<0.001	2.66 [2.02, 3.39]	<0.001
Intestinal ischemia		63 (5.4%)	23 (0.7%)	<0.001	7.05 [4.25, 11.71]	<0.001
Overall complications		527 (45.6%)	766 (21.9%)	<0.001	7.05 [4.25, 11.71]	<0.001
Length of ICU stay*		9.7 ± 12.5	5.4 ± 6.4	<0.001	3.34 [2.80, 3.88]	<0.001
Total LOS*		19.2 ± 29.4	15.0 ± 52.0	0.004	3.09 [-0.13, 6.31]	0.060

PPV, prophylactic postoperative vasopressors; aOR, adjusted odds ratio; CI, confidence interval; MACE, Major Adverse Cardiovascular Events; MI, myocardial infarction; CHF, congestive heart failure; SCI, spinal cord ischemia; HD, hemodialysis; ICU, intensive care unit; LOS, length of stay. *, for the length of ICU stay and total LOS, adjusted slope coefficients (aB1) of multivariable linear regression models are reported.

The multivariable models are adjusted for confounding effects of demographic, clinical, intraoperative characteristics, medications, aortic pathology, presentation, perioperative prophylactic spinal drain, number of used aortic stent-grafts, length of aortic coverage, proximal landing zone (zone 2), distal landing zone (zone 5), and clustered by hospital volume tertiles.

Supplemental Table I. Univariable and multivariable association of PPV with the outcome variables in the sensitivity analysis

	Univariable			Multivariable	
	PPV (N=1,950)	**No PPV (N=8,352)**	**p-value**	**aOR [95%CI]**	**p-value**
30-day mortality	104 (5.3%)	123 (1.5%)	<0.001	2.45 [1.80, 3.32]	<0.001
Dysrhythmias	152 (7.8%)	280 (3.4%)	<0.001	2.02 [1.64, 2.50]	<0.001
Stroke	69 (3.5%)	139 (1.7%)	<0.001	1.82 [1.34, 2.47]	<0.001
SCI	38 (2.0%)	96 (1.2%)	0.005	1.50 [1.02, 2.21]	0.041
HD	87 (4.5%)	242 (2.9%)	<0.001	1.34 [1.02, 1.76]	0.037
Overall complications	361 (18.5%)	769 (9.2%)	<0.001	1.87 [1.62, 2.16]	<0.001
Length of ICU stay*	4.8 ± 6.5	3.1 ± 3.9	<0.001	1.26 [1.05, 1.46]	<0.001
Total LOS*	12.9 ± 40.3	9.3 ± 33.6	<0.001	2.85 [1.10, 4.59]	0.001

PPV, prophylactic postoperative vasopressors; aOR, adjusted odds ratio; CI, confidence interval; SCI, spinal cord ischemia; HD, hemodialysis; ICU, intensive care unit; LOS, length of stay. *, for the length of ICU stay and total LOS, adjusted slope coefficients (aB1) of multivariable linear regression models are reported.

The multivariable models are adjusted for confounding effects of demographic, clinical, intraoperative characteristics, medications, aortic pathology, presentation, urgency, perioperative prophylactic spinal drain, number of used aortic stent-grafts, length of aortic coverage, proximal landing zone (zone 2), distal landing zone (zone 5), and clustered by hospital volume tertiles.