

# Aortic Aneurysm

## Female Sex is Associated with Adverse Cardiac Remodeling and Increased Antihypertensive Requirements After Thoracic Endovascular Aortic Repair

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**Background:** Thoracic endovascular aortic repair (TEVAR) has largely replaced open surgical repair (OSR) for many thoracic aortic pathologies due to lower perioperative morbidity and mortality. However, the increased aortic stiffness imparted by stent grafts may have long-term cardiovascular consequences, particularly among women. The purpose of this study is to evaluate sex-specific differences in cardiac remodeling and antihypertensive requirements following TEVAR compared with OSR.

**Methods:** A retrospective review was conducted of 671 adult patients who underwent TEVAR or OSR for thoracic aortic aneurysm or dissection between January 2015 and January 2022. Of these, 101 patients met inclusion criteria, with complete 3-year antihypertensive medication and  $\geq 1$ -year echocardiographic follow-up data. Primary outcomes included changes in left ventricular ejection fraction (LVEF), left ventricular hypertrophy (LVH), and antihypertensive medication requirements. Multivariable Poisson generalized estimating equation and logistic regression models were used to identify factors associated with medication escalation and cardiac remodeling.

**Results:** Among female patients, TEVAR was associated with significantly greater antihypertensive medication requirements at 12, 24, and 36 months postoperatively ( $P < 0.05$ ) compared with OSR. Worsening LVH occurred more frequently after TEVAR ( $P < 0.05$ ), whereas changes in LVEF, rates of stroke, myocardial infarction, and 1-year mortality were similar between groups. In multivariable analysis, TEVAR independently predicted higher antihypertensive requirements (incidence rate ratio (IRR) 1.48; 95% CI, 1.27–1.73;  $P < 0.001$ ), while OSR was associated with significantly lower odds of worsening LVH compared with TEVAR (OR 0.11; 95% CI, 0.01–0.63;  $P = 0.012$ ). No significant differences were observed between TEVAR and OSR among male patients.

**Conclusion:** Female patients undergoing TEVAR demonstrated greater adverse cardiac remodeling and higher antihypertensive requirements than those undergoing OSR. These findings suggest that sex-related differences in aortic stiffness and device oversizing may contribute to poorer long-term remodeling outcomes in women, underscoring the need for sex-specific endograft sizing and development of more compliant endografts.

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## INTRODUCTION

Thoracic endovascular aortic repair (TEVAR) has become the preferred intervention for many thoracic aortic pathologies over open surgical repair (OSR). TEVAR offers decreased length of hospital stay and reduced perioperative morbidity and mortality, including lower rates of early death, spinal cord ischemia, renal insufficiency, and reoperation for bleeding.<sup>1,2</sup> Despite these advantages, TEVAR is associated with increased aortic stiffness, attributed to the mechanical properties of the stent grafts which are orders of magnitude less compliant than native aortic tissue.<sup>3–6</sup> Increased aortic stiffness raises left ventricular afterload, which can contribute to cardiac remodeling and elevated systolic blood pressure (SBP). Elevated SBP, in turn, accelerates aortic stiffening. Our previous study has shown that patients undergoing TEVAR were likely to require more antihypertensive medications compared to patients with OSR, suggesting increased aortic stiffness following TEVAR.<sup>7</sup> The increased antihypertensive requirements observed after TEVAR likely reflect the reciprocal relationship between SBP and aortic stiffness.<sup>8</sup>

Given the increasing utilization of TEVAR over OSR, it is essential to evaluate both short- and long-term outcomes across diverse patient populations, including potential sex-based differences. Although thoracic aortic disease is more prevalent in men, women demonstrate disproportionately faster aneurysm growth, higher risks of rupture and dissection, and a greater incidence of acute aortic events at smaller diameters.<sup>9</sup> Current literature consistently shows that female patients experience inferior postoperative outcomes compared with male patients following both TEVAR and OSR, including higher long-term mortality<sup>10,11</sup> and higher rates of access-related complications after TEVAR.<sup>12</sup> The underlying reasons for these disparities are likely multifactorial. One critical issue is that women remain persistently underrepresented in endovascular device trials relative to disease prevalence, a disparity that has remained unchanged over time.<sup>13,14</sup> Inferior postoperative outcomes in women may also be explained by more adverse preoperative characteristics. Compared to male patients, women are more likely to be older, have higher baseline frailty, comorbidity association, and aortic stiffness at baseline at the time of TEVAR.<sup>10,15</sup> Additionally, female patients undergoing TEVAR often have smaller absolute aortic diameters but larger aortic size indices relative to males, leading to a proportionally greater aneurysm burden and higher likelihood of presenting

with symptomatic or ruptured aneurysms.<sup>16</sup> These anatomical constraints are reflected in the instructions-for-use for endovascular devices, which disproportionately exclude women compared to men.

While postoperative sex-based outcome disparities following TEVAR are increasingly recognized, little is known about whether these differences extend to long-term hemodynamic changes. To address this gap, we investigated sex-specific differences in cardiac remodeling and aortic stiffness following TEVAR and OSR.

## METHODS

This was a retrospective, single-institution review of 671 adult patients who underwent TEVAR or OSR for thoracic aortic aneurysm, aortic dissection, or both between January 2015 and January 2022 at Loyola University Medical Center. Of these 671 patients, 101 met the inclusion criteria and were included in the final analysis. Inclusion criteria consisted of patients aged 18 years or older who underwent either OSR or TEVAR for thoracic aortic disease and had complete 3-year antihypertensive medication follow-up data as well as at least 1 year of echocardiogram follow-up. Exclusion criteria included concomitant cardiac surgery, severe aortic valve disease, and blunt thoracic aortic injury.

Parts of the work and patient data included in this manuscript have been previously published. Specifically, our prior study<sup>7</sup> included 566 adult patients who underwent OSR or TEVAR between January 2015 and December 2020 at Loyola University Medical Center of whom 100 met the inclusion criteria. In the current analysis, 29 patients from the original cohort were excluded because of incomplete echocardiographic follow-up. The current study extends the study period through January 2022 (671 patients total screened) and refined inclusion criteria to require complete echocardiographic follow-up. An additional 30 patients from the expanded study period were newly identified, resulting in a final cohort of 101 patients. All echocardiographic analyses, blood pressure outcomes, and antihypertensive utilization data for these 30 newly enrolled patients represent original findings not previously reported. Critically, the prior publication did not include any sex-stratified outcome comparisons. The current study's primary novel contribution is a dedicated analysis of sex-based differences in outcomes following OSR and TEVAR.

Collected demographic and clinical variables included age, sex, body surface area (BSA), smoking history, hypertension (HTN), hyperlipidemia, diabetes mellitus (DM), coronary artery disease (CAD), renal insufficiency, end-stage renal disease (ESRD), previous aortic repair, and previous stroke. Postoperative outcomes included changes in the number and dosage of antihypertensive medications, left ventricular ejection fraction (LVEF), left ventricular hypertrophy (LVH), stroke, myocardial infarction, and mortality. Aortic aneurysms were categorized by location as ascending, arch, descending, or combined. Aortic dissections were reported based on the DeBakey classification: Type I, Type II, Type IIIa, or Type IIIb. The Loyola University Chicago Institutional Review Board approved this study for retrospective review of deidentified clinical data and waived the requirement for informed consent.

The primary outcomes of interest were cardiac remodeling and changes in the number and dosage of antihypertensive medications. To assess cardiac function, transthoracic echocardiographic imaging results were collected, including only images interpreted by experienced cardiologists at our institution. Cardiac remodeling was assessed by comparing preoperative and postoperative ( $\geq 1$  year) LVEF and LVH measurements. Worsening LVEF was defined as any numerical reduction in ejection fraction compared with the preoperative echocardiogram. LVH was determined based on the interpretation documented in the echocardiogram report. Worsening LVH was defined as progression from none or mild to moderate or severe compared with the preoperative study. The presence of LVH and reduced LVEF are well-established markers of adverse cardiac remodeling and are associated with increased cardiovascular morbidity and mortality.<sup>17,18</sup>

Medication information was collected preoperatively and at 12 months, 24 months, and 36 months postoperatively. Escalation in the number or dosage of antihypertensive medications was used as a surrogate marker of elevated blood pressure and increased aortic stiffness given the well-established bidirectional relationship between arterial stiffness and blood pressure.<sup>19,20</sup> The REASON study demonstrated that patients with higher aortic stiffness, as measured by pulse wave velocity, exhibit a blunted systolic blood pressure response to antihypertensive therapy and are more likely to require higher doses or combination therapy to achieve blood pressure control.<sup>21</sup> Therefore, longitudinal changes in antihypertensive therapy were used to reflect postoperative alterations in vascular compliance.

## STATISTICAL ANALYSIS

Demographic and clinical continuous variables were summarized via medians and ranges while differences between procedure groups for these variables were assessed by the Wilcoxon rank-sum test. Categorical variables were summarized via frequencies and percentages, and differences between procedure groups for these variables were assessed by Fisher's exact test. Multivariable Poisson generalized estimating equation models assessing differences between surgery types in the number of medications over time, adjusting for demographic and clinical factors of interest. Multivariable logistic models for worsening ejection fraction and worsening left ventricular hypertrophy were also fit to examine which factors significantly affect these outcomes. Analyses were conducted in R 4.3.1.

## RESULTS

Table I summarizes the demographics and clinical characteristics of female ( $n = 50$ ) and male ( $n = 51$ ) patients who underwent TEVAR or OSR. The median age was 66 years in the female cohort and 59 years in the male cohort. Male patients had a significantly higher BSA ( $P < 0.05$ ), whereas a greater proportion of current smokers was observed in the female cohort ( $P < 0.05$ ). No significant sex-based differences were identified in age, BMI, or comorbidities, including HTN, DM, CAD, ESRD, renal insufficiency, stroke, or hyperlipidemia (HLD). Similarly, there were no significant sex differences in prior aortic repair, underlying pathology (aneurysm, dissection, or both), or thoracic aortic disease distribution, including ascending, arch, thoracic, or combined involvement.

No significant differences were observed in postoperative outcomes between the female and male cohort, including the number or dosage escalation of antihypertensive medications at 12, 24, and 36 months after surgery (Table II). Similarly, measures of cardiac remodeling, including LVEF and LVH at follow-up, did not differ between cohorts. Rates of stroke, myocardial infarction, and mortality at 1 year were also comparable between female and male patients.

Table I also presents the demographics and clinical characteristics of female patients treated with TEVAR ( $n = 20$ ) or OSR ( $n = 30$ ). No significant differences were observed in age, BMI, BSA, smoking status, or comorbidities, including HTN, DM, CAD, ESRD, renal insufficiency, or HLD between the two groups. A higher prevalence of prior stroke was noted among female patients treated with

**Table I.** Baseline demographics and clinical characteristics stratified by sex and procedure type

|                           | Females ( <i>N</i> = 50) |                      |                                            | Males ( <i>N</i> = 51) |                      |                                            |                                      |
|---------------------------|--------------------------|----------------------|--------------------------------------------|------------------------|----------------------|--------------------------------------------|--------------------------------------|
| Characteristic            | TEVAR ( <i>N</i> = 20)   | OSR ( <i>N</i> = 30) | <i>P</i> value <sup>b</sup><br>(procedure) | TEVAR ( <i>N</i> = 19) | OSR ( <i>N</i> = 32) | <i>P</i> value <sup>b</sup><br>(procedure) | <i>P</i> value <sup>b</sup><br>(sex) |
| Demographics              |                          |                      |                                            |                        |                      |                                            |                                      |
| Age, median (range)       | 65 (31–84)               | 66 (28–82)           | 0.812                                      | 62 (38–81)             | 59 (27–84)           | 0.599                                      | 0.098                                |
| BMI, median (range)       | 31.2 (15.9–52.1)         | 27.9 (18.6–51.3)     | 0.330                                      | 28.6 (23.4–41.5)       | 29.1 (26.0–56.0)     | 0.606                                      | 0.153                                |
| BSA, median (range)       | 1.89 (1.23–2.62)         | 1.76 (0.00–2.21)     | 0.576                                      | 2.12 (1.87–2.62)       | 2.20 (1.70–3.27)     | 0.441                                      | <0.001 <sup>a</sup>                  |
| Comorbidities             |                          |                      |                                            |                        |                      |                                            |                                      |
| HTN                       | 18 (90.0%)               | 23 (76.7%)           | 0.285                                      | 17 (89.5%)             | 26 (81.3%)           | 0.694                                      | 0.796                                |
| DM                        | 3 (15.0%)                | 1 (3.3%)             | 0.289                                      | 2 (10.5%)              | 3 (9.4%)             | >0.999                                     | >0.999                               |
| CAD                       | 6 (30.0%)                | 13 (43.3%)           | 0.387                                      | 7 (36.8%)              | 6 (18.8%)            | 0.192                                      | 0.204                                |
| ESRD                      | 1 (5.0%)                 | 0 (0.0%)             | 0.400                                      | 4 (21.1%)              | 0 (0.0%)             | 0.016 <sup>a</sup>                         | 0.362                                |
| Renal insufficiency       | 2 (10.0%)                | 2 (6.7%)             | >0.999                                     | 2 (10.5%)              | 2 (6.3%)             | 0.623                                      | >0.999                               |
| Smoker                    | 4 (20.0%)                | 12 (40.0%)           | 0.216                                      | 3 (15.8%)              | 1 (3.1%)             | 0.140                                      | 0.003 <sup>a</sup>                   |
| Previous aortic repair    | 5 (25.0%)                | 3 (10.0%)            | 0.240                                      | 6 (31.6%)              | 1 (3.1%)             | 0.008 <sup>a</sup>                         | 0.786                                |
| Stroke                    | 8 (40.0%)                | 1 (3.3%)             | 0.002 <sup>a</sup>                         | 2 (10.5%)              | 3 (9.4%)             | >0.999                                     | 0.263                                |
| HLD                       | 8 (40.0%)                | 13 (43.3%)           | >0.999                                     | 7 (36.8%)              | 14 (43.8%)           | 0.771                                      | >0.999                               |
| Aortic anatomy            |                          |                      |                                            |                        |                      |                                            |                                      |
| Aneurysm                  | 11 (55.0%)               | 24 (80.0%)           | 0.079                                      | 6 (31.6%)              | 21 (65.6%)           | 0.003 <sup>a</sup>                         | 0.097                                |
| Dissection                | 8 (40.0%)                | 6 (20.0%)            |                                            | 8 (42.1%)              | 11 (34.4%)           |                                            |                                      |
| Both                      | 1 (5.0%)                 | 0 (0.0%)             |                                            | 5 (26.3%)              | 0 (0.0%)             |                                            |                                      |
| Ascending                 | 0 (0.0%)                 | 23 (95.8%)           | <0.001 <sup>a</sup>                        | 0 (0.0%)               | 19 (90.5%)           | <0.001 <sup>a</sup>                        | 0.169                                |
| Arch                      | 0 (0.0%)                 | 0 (0.0%)             |                                            | 3 (27.3%)              | 0 (0.0%)             |                                            |                                      |
| Descending                | 11 (91.7%)               | 0 (0.0%)             |                                            | 6 (54.6%)              | 0 (0.0%)             |                                            |                                      |
| Combination               | 1 (8.3%)                 | 1 (4.2%)             |                                            | 2 (18.2%)              | 2 (9.5%)             |                                            |                                      |
| Dissection type (DeBakey) |                          |                      |                                            |                        |                      |                                            |                                      |
| Type I                    | 0 (0.0%)                 | 5 (83.3%)            | <0.001 <sup>a</sup>                        | 2 (15.4%)              | 8 (80.0%)            | 0.005 <sup>a</sup>                         | 0.443                                |
| Type II                   | 0 (0.0%)                 | 1 (16.7%)            |                                            | 0 (0.0%)               | 0 (0.0%)             |                                            |                                      |
| Type IIIa                 | 6 (66.7%)                | 0 (0.0%)             |                                            | 9 (69.2%)              | 2 (20.0%)            |                                            |                                      |
| Type IIIb                 | 3 (33.3%)                | 0 (0.0%)             |                                            | 2 (15.4%)              | 0 (0.0%)             |                                            |                                      |

BMI, body mass index; BSA, body surface area; CAD, coronary artery disease; DM, diabetes mellitus; ESRD, end-stage renal disease; HLD, hyperlipidemia; HTN, hypertension; OSR, open surgical repair; TEVAR, thoracic endovascular aortic repair.

<sup>a</sup>*P* < 0.05 considered statistically significant.

<sup>b</sup>Wilcoxon rank sum test; Fisher's exact test.

**Table II.** Antihypertensive medication requirements and postoperative outcomes by sex and procedure type

| Characteristic                                 | Females ( <i>N</i> = 50)  |                      |                                            | Males ( <i>N</i> = 51)    |                         |                                            |                                      |
|------------------------------------------------|---------------------------|----------------------|--------------------------------------------|---------------------------|-------------------------|--------------------------------------------|--------------------------------------|
|                                                | TEVAR<br>( <i>N</i> = 20) | OSR ( <i>N</i> = 30) | <i>P</i> value <sup>b</sup><br>(procedure) | TEVAR<br>( <i>N</i> = 19) | OSR<br>( <i>N</i> = 32) | <i>P</i> value <sup>b</sup><br>(procedure) | <i>P</i> value <sup>b</sup><br>(sex) |
| Antihypertensive medication requirements       |                           |                      |                                            |                           |                         |                                            |                                      |
| Number escalation                              | 7 (35.0%)                 | 8 (26.7%)            | 0.547                                      | 7 (36.8%)                 | 13 (40.6%)              | >0.999                                     | 0.404                                |
| Dosage escalation                              | 5 (25.0%)                 | 4 (13.8%)            | 0.456                                      | 3 (15.8%)                 | 5 (15.6%)               | >0.999                                     | 0.794                                |
| Medications at<br>0 month, median<br>(range)   | 2 (0–5)                   | 2 (0–4)              | 0.097                                      | 2 (0–7)                   | 2 (0–4)                 | 0.146                                      | 0.975                                |
| Medications at<br>12 months, median<br>(range) | 2.5 (1–4)                 | 1 (0–4)              | <0.001 <sup>a</sup>                        | 2 (0–5)                   | 2 (0–5)                 | 0.450                                      | 0.639                                |
| Medications at<br>24 months, median<br>(range) | 3 (1–5)                   | 1.5 (0–4)            | 0.003 <sup>a</sup>                         | 2 (0–5)                   | 2 (0–5)                 | 0.367                                      | 0.958                                |
| Medications at<br>36 months, median<br>(range) | 2.5 (1–5)                 | 1.5 (0–4)            | 0.003 <sup>a</sup>                         | 2 (0–5)                   | 2 (0–5)                 | 0.546                                      | 0.875                                |
| Cardiac remodeling                             |                           |                      |                                            |                           |                         |                                            |                                      |
| EF% at baseline,<br>median (range)             | 60 (40–72.5)              | 60 (40–75)           | 0.912                                      | 60 (45–65)                | 60 (40–80)              | 0.803                                      | 0.786                                |
| EF% at follow-up,<br>median (range)            | 58.8 (45–70)              | 60 (35–67.5)         | 0.856                                      | 60 (30–65)                | 60 (25–72)              | 0.602                                      | 0.628                                |
| Worsening EF                                   | 3 (15.0%)                 | 3 (10.0%)            | 0.672                                      | 1 (5.3%)                  | 4 (12.5%)               | 0.639                                      | 0.760                                |
| LVH at baseline                                | 5 (25.0%)                 | 10 (33.3%)           | 0.754                                      | 9 (47.4%)                 | 10 (31.3%)              | 0.369                                      | 0.529                                |
| Worsening LVH                                  | 7 (35.0%)                 | 1 (3.3%)             | 0.005 <sup>a</sup>                         | 4 (21.1%)                 | 8 (25.0%)               | >0.999                                     | 0.455                                |
| Postoperative outcomes at 12 months            |                           |                      |                                            |                           |                         |                                            |                                      |
| Stroke                                         | 2 (10.0%)                 | 4 (13.3%)            | >0.999                                     | 2 (10.5%)                 | 2 (6.3%)                | 0.623                                      | 0.525                                |
| Myocardial infarction                          | 0 (0.0%)                  | 1 (3.3%)             | >0.999                                     | 0 (0.0%)                  | 2 (6.3%)                | 0.523                                      | >0.999                               |
| Mortality                                      | 1 (5.0%)                  | 1 (3.3%)             | >0.999                                     | 1 (5.3%)                  | 1 (3.1%)                | >0.999                                     |                                      |

EF%, ejection fraction; LVH, left ventricular hypertrophy; MI, myocardial infarction; OSR, open surgical repair; TEVAR, thoracic endovascular aortic repair.

<sup>a</sup>*P* < 0.05 considered statistically significant.

<sup>b</sup>Wilcoxon rank sum test; Fisher's exact test.

TEVAR compared with OSR (*P* < 0.05). There were no significant differences in the frequency of prior aortic repair and underlying aortic pathology (aneurysm, dissection, or both). Ascending aortic aneurysms and type A dissections were treated with OSR, while thoracic aortic aneurysms and type B dissections were treated with TEVAR (*P* < 0.001).

Postoperative outcomes in female patients undergoing TEVAR or OSR are summarized in Table II. No baseline difference in the number of antihypertensive medications was observed at 0 months between the TEVAR and OSR female patients. Antihypertensive medication use was significantly higher in the TEVAR cohort at 12, 24, and 36 months postoperatively (*P* < 0.05) (Fig. 1). However, there were no significant differences between groups in the number of agents added or in dosage

escalation over time, indicating that while TEVAR patients required more medications overall, the longitudinal pattern of escalation was comparable between cohorts. A greater proportion of TEVAR patients developed worsening LVH at follow-up (*P* < 0.05). Baseline LVEF and LVH did not differ between groups, and no significant changes in LVEF were observed at follow-up. One-year rates of stroke, myocardial infarction, and mortality were comparable between TEVAR and OSR cohorts.

Table III presents results from the multivariable Poisson GEE model assessing the escalation of antihypertensive medication in female patients after surgery. Increasing age (incidence rate ratio (IRR) 1.007, 95% CI 1.001–1.013, *P* = 0.017) and higher BMI (IRR 1.021, 95% CI 1.011–1.031, *P* < 0.001) were independently associated with greater likelihood of

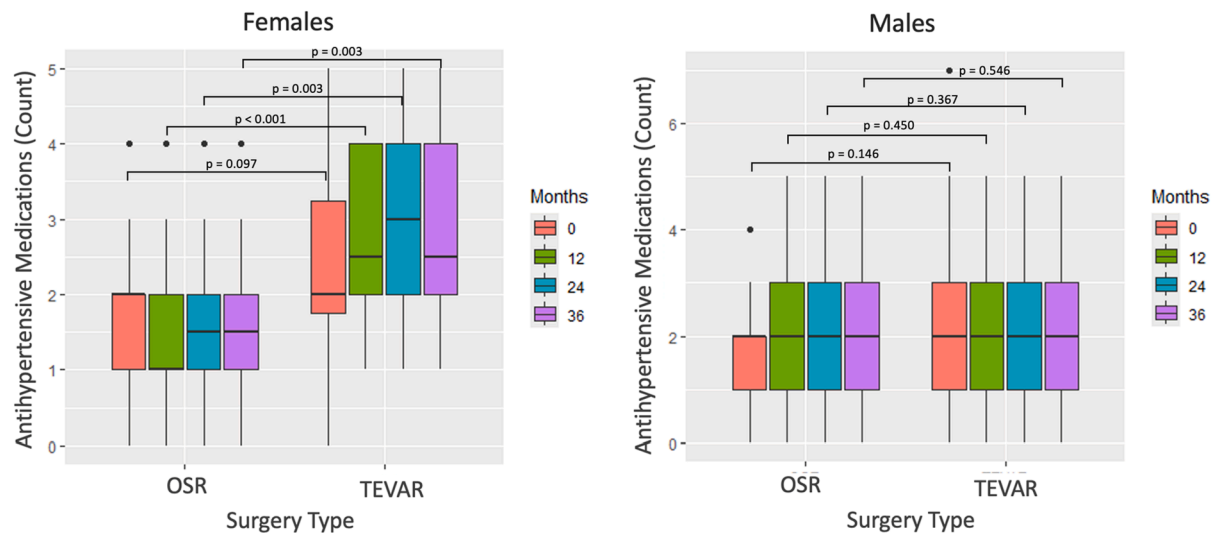


**Fig. 1.** Antihypertensive escalation in female patients with OSR or TEVAR compared with male patients.

**Table III.** Multivariable model for antihypertensive medication escalation in female patients

| Characteristic          | IRR   | 95% CI       | P value             |
|-------------------------|-------|--------------|---------------------|
| Age (y)                 | 1.007 | 1.001, 1.013 | 0.017 <sup>a</sup>  |
| BMI                     | 1.021 | 1.011, 1.031 | <0.001 <sup>a</sup> |
| Months                  | 1.002 | 0.997, 1.008 | 0.38                |
| Surgery type: OSR (ref) | —     | —            | —                   |
| Surgery type: TEVAR     | 1.479 | 1.266, 1.729 | <0.001 <sup>a</sup> |
| Stroke                  | 1.100 | 0.894, 1.354 | 0.37                |

BMI, body mass index; IRR, incidence rate ratio; CI, confidence interval; ref, reference.

<sup>a</sup> $P < 0.05$  considered statistically significant.

**Table IV.** Multivariable model for worsening LVH in female patients

| Characteristic            | OR    | 95% CI       | P-value            |
|---------------------------|-------|--------------|--------------------|
| Age (y)                   | 1.003 | 0.944, 1.075 | 0.92               |
| BMI                       | 0.967 | 0.852, 1.089 | 0.57               |
| Surgery type: TEVAR (ref) | —     | —            | —                  |
| Surgery type: OSR         | 0.108 | 0.010, 0.628 | 0.012 <sup>a</sup> |
| Stroke                    | 1.473 | 0.221, 10.11 | 0.68               |

BMI, body mass index; OR, odds ratio; CI, confidence interval; LVH, left ventricular hypertrophy; ref, reference.

<sup>a</sup> $P < 0.05$  considered statistically significant.

escalation. Compared with OSR, TEVAR was strongly associated with increased antihypertensive medication requirements (IRR 1.479, 95% CI 1.266–1.729,  $P < 0.001$ ). Stroke history was not

significantly associated with escalation (IRR 1.100, 95% CI 0.894–1.354,  $P = 0.37$ ), and time since surgery (months) did not demonstrate a significant effect (IRR 1.002, 95% CI 0.997–1.008,  $P = 0.38$ ).

Table IV presents the results of the multivariable logistic model assessing the likelihood of developing worsening LVH in female patients after surgery. Neither age (OR 1.003, 95% CI 0.944–1.075,  $P = 0.92$ ) nor BMI (OR 0.967, 95% CI 0.852–1.089,  $P = 0.57$ ) were associated with worsening LVH. Compared with TEVAR, OSR was associated with a significantly lower likelihood of developing worsening LVH (OR 0.108, 95% CI 0.010–0.628,  $P = 0.012$ ). Stroke history was not significantly associated with worsening LVH (OR 1.473, 95% CI 0.221–10.11,  $P = 0.68$ ).

Table I presents the demographics and clinical characteristics of male patients treated with TEVAR ( $n = 19$ ) or OSR ( $n = 32$ ). No significant differences were observed in age, BMI, BSA, smoking status, or comorbidities, including HTN, DM, CAD, renal insufficiency, HLD, or stroke between the two groups. A higher prevalence of ESRD and prior aortic repair were noted in the TEVAR cohort ( $P < 0.05$ ). Ascending aortic aneurysms and type A dissections were managed with OSR, while thoracic and arch aortic aneurysms and type B dissections were treated with TEVAR ( $P < 0.05$ ).

Postoperative outcomes among male patients who underwent TEVAR or OSR are summarized in Table II. Antihypertensive medication use at 12, 24, and 36 months did not differ significantly between cohorts. No differences were observed in

the number of agents required or in dosage escalation. Baseline LVEF and LVH were similar between groups, with no significant interval change identified on follow-up. One-year rates of stroke, myocardial infarction, and mortality were likewise comparable between TEVAR and OSR cohorts.

## DISCUSSION

In this study, we observed that female patients undergoing TEVAR experienced greater adverse cardiac remodeling despite escalation in antihypertensive therapy. This suggests that postoperative increases in medication use may be insufficient to counteract the hemodynamic effects of heightened aortic stiffness in women. Older women are known to exhibit greater baseline aortic stiffness than men, largely due to the loss of estrogen-mediated vascular protection after menopause.<sup>15</sup> Increased stiffness has been closely linked to left ventricular hypertrophy, diastolic dysfunction, and treatment-resistant hypertension, all of which may predispose women to adverse remodeling after endograft implantation. Furthermore, our dataset captured baseline comorbidities and select postoperative cardiac parameters but did not systematically track the longitudinal development of new or worsening comorbidities that may independently influence blood pressure over time. In particular, aortic insufficiency, which may develop as a consequence of increased aortic stiffness following repair, was not assessed, yet could meaningfully contribute to changes in blood pressure trajectory. Future studies should incorporate longitudinal comorbidity surveillance, including serial evaluation for aortic insufficiency, to more fully characterize the hemodynamic burden driving adverse cardiac remodeling in this population.

Our primary objective was mechanistic rather than procedural comparison, specifically to explore how different types of aortic intervention may differentially affect aortic compliance and cardiac remodeling. The observed association between TEVAR in women and higher rates of adverse cardiac remodeling should therefore be interpreted as hypothesis-generating and not as evidence of superiority or inferiority of one procedure over another.

The increased risk of cardiac remodeling and antihypertensive escalation observed in female patients may also reflect the impact of endograft oversizing. Because women have smaller native aortic diameters, they are more likely to experience greater relative oversizing. Kuo et al. demonstrated that

excessive stent oversizing in patients with narrow aortas was associated with increased left ventricular wall thickening and hypertrophy.<sup>22</sup> Furthermore, sex-based vascular studies have shown that women possess thinner aortic walls and higher activity of matrix metalloproteinases, which may exacerbate the effects of endograft-induced stiffness and promote maladaptive remodeling.<sup>9</sup> These findings underscore the importance of individualized stent sizing and postoperative blood pressure management, particularly in women with smaller aortic diameters.

In contrast, male patients generally have larger aortic diameters and consequently experience less stent oversizing and postprocedural stiffening. This may contribute to their reduced need for antihypertensive escalation and lesser degrees of cardiac remodeling. Du et al. reported that TEVAR facilitated reversal of adverse left ventricular remodeling among patients who achieved favorable aortic remodeling following repair of acute type B dissection.<sup>23</sup> However, those results were derived predominantly from male cohorts, limiting their generalizability to female patients.

Finally, a systematic review and meta-analysis of sex-based outcomes after TEVAR demonstrated significantly lower 2-year survival among women compared with men, despite similar short-term mortality.<sup>11</sup> The divergence in long-term outcomes may reflect the cumulative effects of increased aortic stiffness and adverse cardiac remodeling in women. These findings highlight the need for sex-specific considerations in endograft sizing, device design, and postoperative management, as well as for improved representation of female patients in endovascular device trials and registries.

## LIMITATIONS

Endograft length and oversizing data were not collected. Although experimental models show a length-dependent increase in aortic stiffness after stent-graft deployment, clinical studies have reported mixed results, with stiffness progression driven more by traditional cardiovascular risk factors than by graft length itself.<sup>3,5,24,25</sup> Postoperative echocardiograms were obtained during routine follow-up within 1–3 years rather than at prespecified study intervals, limiting assessment of the temporal course of cardiac remodeling.

Aortic pathology differed between cohorts: most TEVAR cases involved the descending thoracic aorta, whereas most OSR cases involved the ascending

aorta or arch. We acknowledge this represents an imperfect anatomical comparison. Ascending aortic repair was selected as an institution-matched open surgery cohort of sufficient size and comparable physiologic burden. An anatomically matched comparison with open thoracoabdominal aortic repair was not feasible due to low case volume, differing indications, and selection criteria. Time from initial dissection or aneurysm identification to repair was not systematically recorded and may be an important determinant of perioperative hemodynamics and longer-term remodeling outcomes; this should be prioritized in future prospective studies.

Furthermore, increased postoperative antihypertensive use may partly reflect closer surveillance and more aggressive medication titration after major aortic repair rather than true hemodynamic deterioration alone, a distinction our retrospective design cannot fully resolve.

## CONCLUSION

In this study, female patients undergoing TEVAR demonstrated greater adverse cardiac remodeling and higher antihypertensive requirements compared with those who underwent OSR. Given that female sex is independently associated with increased aortic stiffness and maladaptive cardiac remodeling after TEVAR, these findings highlight the need for sex-specific considerations in endograft sizing, device compliance, and postoperative management. Future studies should aim to enhance inclusion of female patients in endovascular trials and optimize device design to mitigate stiffness-related remodeling in women.

## CREDIT AUTHORSHIP CONTRIBUTION STATEMENT

**Karen Yuan:** Writing — review & editing, Writing — original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Cerie Ock:** Writing — review & editing, Writing — original draft, Data curation. **Irene Helenowski:** Writing — original draft, Software, Methodology, Formal analysis. **Jeffrey Schwartz:** Project administration, Conceptualization. **Michael C. Soult:** Writing — review & editing, Supervision, Resources, Project administration, Data curation. **Carlos F. Bechara:** Writing — review & editing, Supervision, Resources, Project administration, Methodology, Investigation, Data curation, Conceptualization.

## REFERENCES

1. Liu J, Gou D, Xu K, et al. Comparison of short-and long-term outcomes between endovascular and open repair for descending thoracic aortic aneurysm: a systematic review and meta-analysis. *Int J Surg* 2025;111:2662–74.
2. Cheng D, Martin J, Shennib H, et al. Endovascular aortic repair versus open surgical repair for descending thoracic aortic disease a systematic review and meta-analysis of comparative studies. *J Am Coll Cardiol* 2010;55:986–1001.
3. Yamashita Y, Oishi Y, Matsuyama S, et al. Determinants of changes in arterial stiffness after thoracic endovascular aortic repair. *Ann Vasc Surg* 2021;70:474–80.
4. Bissacco D, Conti M, Domanin M, et al. Modifications in aortic stiffness after endovascular or open aortic repair: a systematic review and meta-analysis. *Eur J Vasc Endovasc Surg* 2022;63:567–77.
5. de Beaufort HWL, Conti M, Kamman AV, et al. Stent-graft deployment increases aortic stiffness in an Ex vivo porcine model. *Ann Vasc Surg* 2017;43:302–8.
6. van Bakel TMJ, Arthurs CJ, Nauta FJH, et al. Cardiac remodeling following thoracic endovascular aortic repair for descending aortic aneurysms. *Eur J Cardiothorac Surg* 2019;55:1061–70.
7. Yuan K, Potluri VK, Gorantla A, et al. Cardiac remodeling and antihypertensive medication changes after thoracic endovascular aortic repair vs open surgical repair. *J Vasc Surg* 2025;81:566–73.
8. Takeda Y, Sakata Y, Ohtani T, et al. Endovascular aortic repair increases vascular stiffness and alters cardiac structure and function. *Circ J* 2014;78:322–8.
9. Samman B, Deng MX, Chung JCY, et al. Understanding thoracic aortic disease in women. *Circ Res* 2025;136:606–17.
10. Deery SE, Shean KE, Wang GJ, et al. Female sex independently predicts mortality after thoracic endovascular aortic repair for intact descending thoracic aortic aneurysms. *J Vasc Surg* 2017;66:2–8.
11. Zhang Y, Zhang Y, Wang Y, et al. Sex-based outcomes after thoracic endovascular aortic repair: a systematic review and meta-analysis. *J Vasc Surg* 2024;80:1303–1313.e8.
12. Lomazzi C, Mascoli C, de Beaufort HWL, et al. Gender related access complications after TEVAR: analysis from the retrospective multicentre cohort GORE® GREAT Registry Study. *Eur J Vasc Endovasc Surg* 2020;60:203–9.
13. Marcaccio CL, O'Donnell TFX, Dansey KD, et al. Disparities in reporting and representation by sex, race, and ethnicity in endovascular aortic device trials. *J Vasc Surg* 2022;76:1244–1252.e2.
14. Patel J, Pallapothu S, Langston A, et al. A systematic review of the recruitment and outcome reporting by sex and race/ethnicity in stent device development trials for endovascular abdominal aortic aneurysm repair. *Ann Vasc Surg* 2023;89:353–61.
15. Coutinho T. Arterial stiffness and its clinical implications in women. *Can J Cardiol* 2014;30:756–64.
16. Jiang DJ, Pugar JA, Babrowski TA. Current management practices for thoracic aortic aneurysms is associated with increased symptomatic presentation in females. *J Vasc Surg* 2025;82:1966–71.
17. Aimo A, Vergaro G, González A, et al. Cardiac remodelling — Part 2: Clinical, imaging and laboratory findings. A review from the Study Group on Biomarkers of the Heart Failure Association of the European Society of Cardiology. *Eur J Heart Fail* 2022;24:944–58.

18. Đorđević DB, Koračević GP, Đorđević AD, et al. Hypertension and left ventricular hypertrophy. *J Hypertens* 2024;42:1505–15.
19. Dernellis J, Panaretou M. Aortic stiffness is an independent predictor of progression to hypertension in nonhypertensive subjects. *Hypertension* 2005;45:426–31.
20. Cavalcante JL, Lima JA, Redheuil A, et al. Aortic stiffness: current understanding and future directions. *J Am Coll Cardiol* 2011;57:1511–22.
21. Protogerou A, Blacher J, Stergiou GS, et al. Blood pressure response under chronic antihypertensive drug therapy: the role of aortic stiffness in the REASON (Preterax in Regression of Arterial Stiffness in a Controlled Double-Blind) study. *J Am Coll Cardiol* 2009;53:445–51.
22. Kuo MC, Meena RA, Ramos CR, et al. Stent graft oversizing is associated with an increased risk of long-term left ventricular wall thickening in young patients following thoracic endovascular aortic repair. *Ann Vasc Surg* 2021;76:66–72.
23. Du Y, Aizezi M, Lin H, et al. Left ventricular remodeling in patients with acute type B aortic dissection after thoracic endovascular aortic repair: short- and mid-term outcomes. *Int J Cardiol* 2019;274:283–9.
24. Moulakakis KG, Kadoglou NPE, Antonopoulos CN, et al. Changes in arterial stiffness and N-terminal pro-brain natriuretic peptide levels after endovascular repair of descending thoracic aorta. *Ann Vasc Surg* 2017;38:220–6.
25. Ohyama Y, Teixeira-Tura G, Ambale-Venkatesh B, et al. Ten-year longitudinal change in aortic stiffness assessed by cardiac MRI in the second half of the human lifespan: the multi-ethnic study of atherosclerosis. *Eur Heart J Cardiovasc Imaging* 2016;17:1044–53.