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Left ventricular remodeling in patients with acute type B aortic dissection after thoracic endovascular aortic repair: Short- and mid-term outcomes☆

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ABSTRACT

Background: Left ventricular (LV) remodeling remains unknown in patients with acute Type B aortic dissection (aTBAD) after thoracic endovascular aortic repair (TEVAR) during follow-up.
Methods: Between May 2004 and January 2016, 163 consecutive patients (136 males, mean preoperative age: 51.06 ± 10.79 years) with aTBAD underwent TEVAR. A linear mixed model was used to evaluate risk factor influencing on LV remodeling and investigate longitudinal changes in LV thickness, diameter, volume, function and mass at preoperation, postoperation, short- and mid-term follow-up.
Results: Median follow-up time was 48.0 months (quartiles 1–3, 31–84 months, maximum 147 months). LV thickness and mass followed a continuous downward trend over time. Interventricular septal thickness at end-diastole significantly decreased at mid-term follow-up (time, $p < 0.001$: preoperative 11.59 ± 0.14 mm vs mid-term 10.82 ± 0.15 mm, $p < 0.001$; postoperative 11.40 ± 0.14 mm vs mid-term 10.82 ± 0.15 mm, $p = 0.006$). LV posterior wall thickness at end-diastole was markedly reduced at mid-term follow-up (time, $p < 0.001$: preoperative 10.89 ± 0.11 mm vs mid-term 10.02 ± 0.11 mm, $p < 0.001$; postoperative 10.78 ± 0.13 mm vs mid-term 10.02 ± 0.11 mm, $p < 0.001$; short-term 10.56 ± 0.15 mm vs mid-term 10.02 ± 0.11 mm, $p = 0.021$). LV mass index markedly decreased during follow-up (time, $p = 0.001$: preoperative 129.60 ± 3.55 g/m^2 vs short-term 119.26 ± 3.19 g/m^2, $p = 0.009$; preoperative 129.60 ± 3.55 g/m^2 vs mid-term 115.79 ± 3.62 g/m^2, $p = 0.003$). LV function was improved, but not significantly so, during follow-up. Strict blood pressure control had no influence on LV remodeling. True lumen followed a continuous enlargement trend in terms of proximal thoracic aorta and celiac trunk level during follow-up.
Conclusions: TEVAR can reverse LV remodeling and LV hypertrophy in patients with aTBAD during follow-up.

1. Introduction

Type B aortic dissection (TBAD) involves the aorta distal to the left subclavian artery and accounts for 25–40% of aortic dissections, nearly 75% as uncomplicated TBAD [1] and approximately 80% with hypertension [2]. Because of adaptation to chronic pressure overload [3], acute TBAD (aTBAD) is often complicated with significant left ventricular (LV) hypertrophy (LVH), and a marked increase in LV posterior wall thickness (PWT) and interventricular septal thickness (IVST) [4]. Thoracic endovascular aortic repair (TEVAR) has advantages of minimal invasion, significant reduction of morbidity and mortality, and noticeable therapeutic benefit terms in comparison with open surgery for the treatment of complicated TBAD [5]. Given the high incidence of aortic-related adverse events associated with surgical intervention and poor long-term survival with medical treatment, TEVAR may even be recommended as optimal therapy for uncomplicated aTBAD patients [6,7]. It has been emphasized that endovascular aortic repair for thoracic and abdominal aneurysms can increase aortic stiffness and subsequently result in hypertension, further induce adverse cardiovascular remodeling

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including LVH [8]. To date, accumulating studies of clinical outcomes have mainly focused on stent graft design, treatment effects, and risk factor prediction; however, little research has investigated the influence of TEVAR on LV remodeling in patients with aTBAD during follow-up. The aim of this study was to evaluate longitudinal changes of LV remodeling in patients with aTBAD after TEVAR during follow-up, taking into consideration the influence of strict blood pressure control.

2. Methods

2.1. Study subjects

Between May 2004 and January 2016, 163 consecutive patients (136 males, mean age 51.06 ± 10.79 years) with aTBAD who underwent TEVAR at the People's Hospital of Xinjiang Uygur Autonomous Region were enrolled. Patients who had congenital heart disease, severe valvular heart disease, thoracic aortic pseudoaneurysm, or traumatic aortic injury, or who had undergone a previous aortic surgical procedure, were excluded. The study protocol was approved by the institutional ethics committee at the People's Hospital of Xinjiang Uygur Autonomous Region, and all subjects provided informed consent.

2.2. Surgical techniques

Based on diagnostic measurements from computed tomography angiography (CTA), the surgeon was required to identify potential supra-aortic anomalies (lusorian artery), incomplete circle of Willis or dominant left vertebral artery in the case of intended occlusion of left subclavian artery (LSA). All intervention procedures were performed under general anesthesia in a hybrid operating room including fluoroscopic and angiographic guidance. Thoracic stent-graft devices were delivered through the femoral artery surgical cut down. Stent grafts were used both to scaffold up to 15–30 cm of dissected aorta and to seal major entries. Some patients required LSA coverage because of inadequate proximal landing zone (<15 mm). Angiography was performed immediately after deployment to confirm coverage of the entry tear and closure of aortic dissection. Balloon dilatation was performed if proximal wall apposition was incomplete or in the event of endoleak. Prophylactic cerebrospinal fluid drainage was not used unless clinical paraparesis occurred postoperatively. Controlled hypotension with systolic pressure was lowered to ≤90 mm Hg by urapidil during stent release. The size of the thoracic stent graft was selected according to the aortic diameter of the proximal landing zone assessed by intraoperative digital subtraction angiography (DSA). Oversize was limited to between 10% and 20%.

2.3. Postoperative management

All subjects were continuously monitored for at least 12 h in at the intensive care unit after TEVAR. Renal replacement therapy was emergently performed in case of acute renal failure. For patients with poor blood pressure control, we worked with the physician's team to determine the antihypertensive regimen. Oral antihypertensive agents included beta-blockers, calcium-channel blockers, angiotensin-converting enzyme inhibitor, and angiotensin II receptor blockers.

2.4. Echocardiography parameters and CTA examination

Transthoracic echocardiography was performed with commercially available ultrasound systems (Sonos 7500, IE33, EPIQ-7C; Philips Healthcare, Bothell, WA, USA) by two experienced sonographers. Interventricular septal thickness at end-diastole (IVST), LV posterior wall thickness at end-diastole (PWT), LV end-diastolic diameter (LVEDD) and LV end-systolic diameter (LVESD) were measured at left parasternal left ventricle long-axis transection. LV end-diastolic volume (LVEDV) and LV end-systolic volume (LVESV) were quantified from the apical 2- and 4-chamber views. LV ejection fraction (LVEF) was calculated using the Simpson biplane method [9]. Aortic regurgitation (AR) grade was evaluated by measurement of the regurgitant jet width relative to the LV outflow tract diameter or the jet area relative to the LV outflow tract area. LV mass (LVM) was calculated by the formula recommended by the American Society of Echocardiography (ASE) [10]. LV mass index (LVMI) was calculated as LVM divided by body surface area. LV hypertrophy (LVH) was defined by LVMI >115 g/m^2 for males and > 95 g/m^2 for females as recommended by the ASE [9]. Aortic remodeling was evaluated by a 64-slice multidetector CT scanner (Ingenuity CT; Philips Medical Systems, Cleveland, OH, USA), a 320-slice CT scanner (Aquilion ONE TSX-301A; Toshiba Medical Systems, Tochigi, Japan). Status of the false lumen (FL) was defined as patent, partial thrombosis, complete thrombosis, and obliteration. This study mainly focused on the true lumen (TL) area and the FL area at the proximal thoracic aorta level approximately 2 cm distal to the LSA (proximal segment of the stent) and at the celiac trunk level.

2.5. Patient follow-up

All patients who underwent TEVAR were subject to inpatient review. Clinical examination, laboratory tests, and transthoracic echocardiography were performed at preoperation, postoperation, 3 and 12 months' follow-up after the initial procedure and annually thereafter. CTA was measured preoperatively, at 3 and 12 months after initial procedure, and annually thereafter. Short-term follow-up was defined as 3 months after TEVAR. Mid-term follow-up was defined as 3–5 years after TEVAR.

2.6. Statistical analysis

All statistical analyses were performed using SPSS 24.0 for Windows (SPSS, Chicago, IL, USA). Continuous variables are presented as means ± standard deviation; categorical variables are given as frequencies and percentages. We used an Independent-Samples *t*-test to compare continuous variables. Categorical variables were compared by Chi-square test or the Fisher exact test. Survival and freedom from reoperation was analyzed using the Kaplan–Meier curves. Furthermore, a linear mixed model was applied to assess longitudinal changes in LV thickness, diameter, volumes, mass and function. Follow-up time (pre-operation, post-operation, short- and mid-term follow-up) and strict blood pressure control (below 140/90 mm Hg) were incorporated in the model as fixed variables. An unstructured covariance matrix was applied. The estimated marginal mean ± standard error of the mean was presented. Post hoc analyses were estimated using the Bonferroni test to correct for multiple comparisons. All tests were double-sided, a p value of <0.05 was considered statistically significant.

3. Results

3.1. Baseline characteristics

A total of 163 consecutive patients underwent TEVAR; 138 (84.7%) patients with uncomplicated aTBAD were identified from hospital records. The baseline characteristics and instrumental data are summarized in Table 1. Hypertension (74.8%), smoking (31.9%), and hyperlipidemia (20.2%) were more frequent in patients with aTBAD. Moreover, LVH (72.4%), coronary artery disease (14.7%), and lacunar infarction (16.0%) were more common in individuals with aTBAD. In addition, subjects with aTBAD had an increased burden of renal cyst (16.0%) and hepatic cyst (9.2%).

Table 1
Baseline clinical characteristics.

Variable	n = 163
Men (%)	136(83.4)
Age (years)	51.06 ± 10.79
Smoking(%)	52(31.9)
Drinking(%)	21(12.9)
Hypertension(%)	122(74.8)
LVH	118(72.4)
Diabetes mellitus (%)	7(4.3)
Hyperlipidemia (%)	33(20.2)
Coronary artery disease(%)	24(14.7)
Abdominal aortic aneurysm(%)	3(1.8)
Previous hematencephalon(%)	3(1.8)
Previous Lacunar infarction(%)	26(16.0)
Previous renal atrophy(%)	5(3.1)
Chronic renal insufficiency(%)	12(7.4)
Renal cyst(%)	26(16.0)
Hepatic cyst(%)	15(9.2)
Chronic cholecystitis(%)	18(11.0)
Nodular goiter(%)	1(0.6)
Dissection limited to thoracic aorta (%)	22(13.5)
Pre-operative complication	
Hypoxic ischemic encephalopathy(%)	5(3.1)
Mesenteric ischemic disease(%)	4(2.5)
Acute renal infarction(%)	4(2.5)
Limb ischemic disease(%)	12(7.4)
Previous surgery history	
Previous coronary artery bypass grafting(%)	2(1.2)
Previous percutaneous coronary intervention(%)	3(1.8)
Previous lung cancer radical mastectomy(%)	1(0.6)
Previous radical mastectomy(%)	1(0.6)
Previous surgery for abdominal trauma(%)	2(1.2)
Previous cholecystectomy(%)	8(4.9)
Previous appendectomy(%)	7(4.3)
Previous hernia surgery(%)	3(1.8)
Previous thyroidectomy(%)	3(1.8)
Previous cesarean section(%)	4(2.5)
Previous myomectomy(%)	1(0.6)
Previous hysterectomy(%)	2(1.2)

Data are presented as mean ± standard deviation, or as number (percentage). LVH, left ventricular hypertrophy.

Table 2
Surgical procedure and peri-operative characteristics.

Variable	n = 163
Proximal sent graft type	
Medtronic Talent stent graft (%)	108(66.3)
Ankura thoracic stent graft (%)	55(33.7)
Distal bare stent type	
Medtronic Endurant stent graft (%)	10(6.1)
Distance of the aorta covered(mm)	150.95 ± 120.41
LSA coverage	
Without coverage (%)	117(71.8)
Partial coverage (%)	18(11.0)
Complete coverage (%)	28(17.2)
Concomitant procedures	
CABG (%)	1(0.6)
Femoral-femoral artery bypass (%)	1(0.6)
Postoperative complications	29(17.8)
Acute cerebral infarction (%)	1(0.6)
Hypoxic/ischemic encephalopathy (%)	2(1.2)
Spinal cord ischemia (%)	3(1.8)
Lower-extremity ischemia (%)	1(0.6)
Acute renal failure (%)	1(0.6)
Obstructive jaundice (%)	1(0.6)

Data are presented as mean ± standard deviation, or as number (percentage). LSA, left subclavian artery; CABG, coronary artery bypass grafting.

3.2. Procedural details of initial endovascular repair

Endovascular coverage of the primary entry tear was achieved in all cases, and no patient required conversion to open repair. A total of 173 stents were used in 163 acute cases (55 Ankura thoracic stent grafts, 108 Medtronic Talent grafts, and 10 Medtronic Endurant stent grafts). The mean distance of the aorta covered was 150.95 ± 120.41 mm.

The proximal landing zones for the endovascular repair were classified according to the position with respect to the great vessels. There was complete coverage of the LSA in 17.2% (28/163), partial coverage of LSA in 11.0% (18/163), and no coverage of LSA in 71.8% (117/163) of patients. Concomitant procedures included one femoral-femoral artery bypass and one coronary artery bypass grafting procedure. Acute ischemic stroke occurred in one patient who had partial LSA coverage. Hypoxic/ischemic encephalopathy after TEVAR was observed in two patients, including one with complete LSA coverage. Three patients experienced spinal cord ischemia; one with paraplegia had complete LSA coverage. Other complications are outlined in Table 2. All complications were managed by active internal physicians.

3.3. Mortality

Follow-up was completed in 93.3% of patients (152/163) with a median duration of 48.0 months (quartiles 1–3, 31–84 months, maximum 147 months). Short-term and mid-term mortality rates were 1.2% (2/163) and 14.1% (23/163), respectively. Common causes of death included stroke, aortic rupture, and malignant tumor (Supplementary Table 1).

3.4. Survival outcomes

The 5-year survival rate was 73.26 ± 6.00%, and mean survival 120.02 ± 4.94 months; (95% confidence interval, 110.33–129.70) (Fig. 1A). Re-interventions were performed in 6 patients during follow-up. The freedom from re-interventions after 5-year follow-up was 93.47 ± 3.06% (Fig. 1B). Reasons for re-interventions included proximal and distal stent graft-induced new entry (SINE) in one patient, distal SINE in two patients, aneurysmal dilatation of thoracic aorta induced by endoleak in one patient, aneurysmal dilatation of abdominal aorta induced by endoleak in one patient, and aneurysmal dilatation of thoracoabdominal aorta induced by endoleak in one patient.

During the follow-up period, two patients underwent coronary artery bypass grafting procedure and another patient underwent percutaneous coronary artery intervention. No deaths among these patients were recorded during the study period.

3.5. Left ventricular remodeling outcomes

Longitudinal changes of LV thickness, diameter, volume and mass in aTBAD patients after TEVAR are depicted in Fig. 2. For echocardiography, 76.7% (125/163) of patients were available at short-term follow-up and 57.1% (93/163) at mid-term follow-up. Overall, LV remodeling and LVH was reversed after TEVAR.

IVST showed a continuous downward trend, further decreased at mid-term follow-up (time, $p < 0.001$: preoperative 11.59 ± 0.14 mm vs mid-term 10.82 ± 0.15 mm, $p < 0.001$; postoperative 11.40 ± 0.14 mm vs mid-term 10.82 ± 0.15 mm, $p = 0.006$). In addition, strict blood pressure control had no influence on IVST regression (blood pressure control, $p = 0.539$) (Fig. 2A).

PWT showed a continuously reducing tendency up until mid-term follow-up (time, $p < 0.001$: preoperative 10.89 ± 0.11 mm vs mid-term 10.02 ± 0.11 mm, $p < 0.001$; postoperative 10.78 ± 0.13 mm vs mid-term 10.02 ± 0.11 mm, $p < 0.001$; short-term 10.56 ± 0.15 mm vs mid-term 10.02 ± 0.11 mm, $p = 0.021$). Moreover, strict blood pressure control did no impact on PWT regression (blood pressure control, $p = 0.882$) (Fig. 2B).

LVEDD demonstrated a slightly downward trend at short-term follow-up, with partial regain at mid-term follow-up (time, $p = 0.419$: preoperative 49.47 ± 0.56 mm vs mid-term 49.19 ± 0.67 mm, $p > 0.05$). Strict blood pressure control had no effect on LVEDD regression (blood pressure control: $p = 0.082$) (Fig. 2C).

LVESD sharply shrank postoperatively, with no further significant decrease during follow-up (time, $p = 0.017$: preoperative 27.41 ± 0.58 mm vs postoperative 26.13 ± 0.55 mm, $p = 0.045$; preoperative

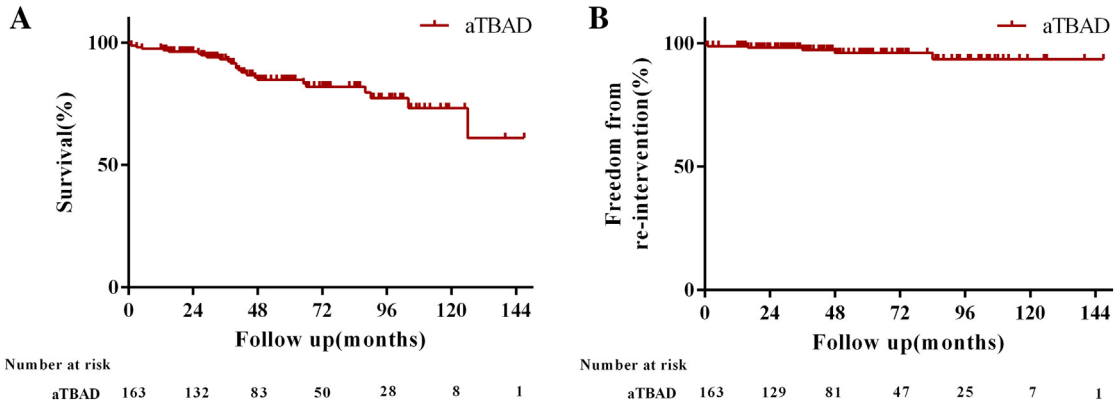

Fig. 1. Kaplan–Meier curves for survival and freedom from re-intervention.

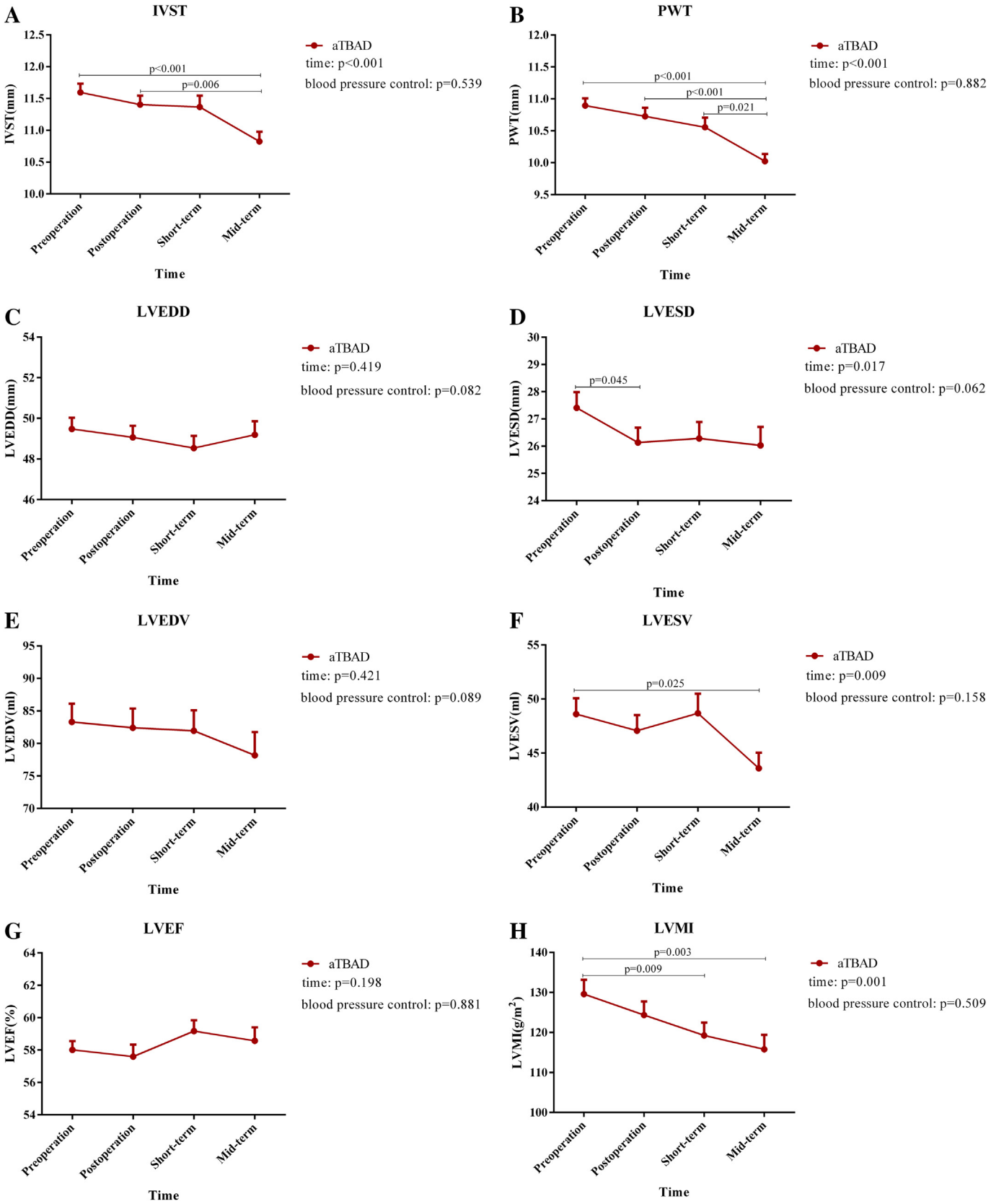

Fig. 2. Left ventricular (LV) remodeling over time in aTBAD. Data are displayed as estimated marginal means ± standard error of the mean. aTBAD, acute Stanford Type B aortic dissection; IVST, interventricular septal thickness; PWT, posterior wall thickness; LVEDD, LV end-diastolic diameter; LVESD, LV end-systolic diameter; LVEDV, LV end-diastolic volume; LVESV, LV end-systolic volume; LVEF, LV ejection fraction; LVMI, LV mass index.

27.41 ± 0.58 mm vs mid-term 26.03 ± 0.67 mm, p > 0.05). Strict blood pressure control had no effect on LVESD regression (blood pressure control: p = 0.062) (Fig. 2D).

LVEDV gradually shrank over time, but without significant difference at all time points (time, p = 0.421: preoperative 83.30 ± 2.82 mL vs mid-term 78.18 ± 3.55 mL, p > 0.05). Strict blood pressure control did not affect LVEDV regression (blood pressure control: p = 0.089) (Fig. 2E).

LVESV marginally shrank postoperatively, but noticeably so at mid-term (time, p = 0.009: preoperative 48.61 ± 1.46 mL vs mid-term 43.59 ± 1.44 mL, p = 0.025). However, strict blood pressure control was not a factor in LVESV regression (blood pressure control: p = 0.158) (Fig. 2F).

LVEF slightly declined postoperatively, but did not significantly improve during follow-up (time, p = 0.198: preoperative 58.01 ± 0.55% vs mid-term 58.57 ± 0.83%, p > 0.05). Strict blood pressure control had no influence on LVEF improvement (blood pressure control: p = 0.881) (Fig. 2G).

LVMI showed a substantial decreasing trend, markedly so at short- and mid-term follow-up (time, p = 0.001: preoperative 129.60 ± 3.55 g/m^2 vs short-term 119.26 ± 3.19 g/m^2, p = 0.009; preoperative 129.60 ± 3.55 g/m^2 vs mid-term 115.79 ± 3.62 g/m^2, p = 0.003). Again, LVMI regression was not affected by strict blood pressure control (blood pressure control: p = 0.509) (Fig. 2H).

3.6. Prevalence of aortic regurgitation

The prevalence of AR preoperatively, postoperatively, and during follow-up is displayed in Supplementary Fig. 1. AR mainly presented as mild in aTBAD given that the intimal flap did not involve the aortic valve. The prevalence of mild AR (grade 1) 44.2% (72/163) prior to operation, immediately decreased postoperatively to 31.5% (47/149), then remained stable at short-term 29.6% (37/125) and mid-term 28.0% (26/93) (p = 0.008).

3.7. Aortic remodeling outcomes

Aortic remodeling outcomes after TEVAR are plotted in Supplementary Fig. 2. For CTA, 63.8% (104/163) of patients were available at short-term follow-up, and 42.3% (69/163) patients at mid-term follow-up. Regarding proximal thoracic aorta level (Supplementary Fig. 2A), TL displayed an increasing tendency, being significantly larger at short- and mid-term (preoperative 534.44 ± 27.89 mm^2 vs short-term 739.74 ± 30.56 mm^2, p < 0.001; preoperative 534.44 ± 27.89 mm^2 vs mid-term 836.06 ± 37.38 mm^2, p < 0.001). FL markedly shrank up to short-term follow-up, then dilated remarkably by mid-term (preoperative 799.71 ± 77.70 mm^2 vs short-term 497.17 ± 54.88 mm^2, p = 0.023; short-term 497.17 ± 54.88 mm^2 vs mid-term 1365.92 ± 232.21 mm^2, p = 0.013). Status of FL was as follows: at short-term—patent 3.8% (4/104), partial thrombosis 14.4% (15/104), complete thrombosis 31.7% (33/104), obliteration 50.0% (52/104); at mid-term—patent 2.9% (2/69), partial thrombosis 7.2% (5/69), complete thrombosis 15.9% (11/69), obliteration 73.9% (51/69).

With regard to celiac trunk level (Supplementary Fig. 2B), TL manifested a consistently increasing tendency, strikingly expanded at short- and mid-term follow-up (preoperative 264.01 ± 12.14 mm^2 vs short-term 302.17 ± 11.47 mm^2, p = 0.002; preoperative 264.01 ± 12.14 mm^2 vs mid-term 369.49 ± 16.45 mm^2, p < 0.001; short-term 302.17 ± 11.47 mm^2 vs mid-term 369.49 ± 16.45 mm^2, p < 0.001). FL exhibited a considerable dilatation trend at short- and mid-term follow-up (preoperative 384.29 ± 19.83 mm^2 vs short-term 481.80 ± 31.77 mm^2, p = 0.004; preoperative 384.29 ± 19.83 mm^2 vs mid-term 616.93 ± 67.89 mm^2, p = 0.003). Status of FL as at short-term: patent, partial thrombosis, complete thrombosis, and obliteration was 56.7% (59/104), 26.9% (28/104), 8.7% (9/104), and 7.7% (8/104) respectively, at short-term follow-up and 33.3% (23/69), 37.7% (26/69), 15.9% (11/69), and 13.0% (9/69), respectively, at mid-term follow-up.

4. Discussion

The main findings of the present study can be summarized as follows. (1) Patients with acute TBAD commonly suffer LVH as a preoperative complication. (2) TEVAR can reverse LV remodeling and LVH. (3) LV thickness and mass showed a continuous regression trend overall during follow-up. (4) LV function was improved during follow-up. (5) Mild aortic regurgitation was significantly reduced after the operation, but remained stable during follow-up. (6) Strict blood pressure control had no influence on LV remodeling.

As a risk factor, the high prevalence of hypertension in our investigation (74.8%) was similar to the results reported by the Sino-RAD [11]. Long-term hypertension leads to cardiac anatomic and functional changes characterized as LVH because of adaptation to chronic pressure overload [3]. A previous investigation confirmed that patients with aTBAD demonstrated significant LVH with PWT and IVST obviously thickened [4]. IRAD data further suggested a high prevalence of LVH in black (44.2%) and white (20.1%) patient cohorts [12]. Our research indicated that the prevalence of LVH (72.4%) was higher, likely because nearly half of the subjects in previous reports had type A aortic dissection. Their results would be more plausible if follow-up observation was included. Accumulating clinical evidence supports antihypertensive treatment being able to regress LVH [3]. In our results, 60.1% (98/163) of patients underwent strict blood pressure control (below 140/90 mm Hg); however, a linear mixed model indicated that antihypertensive treatment cannot reverse LVH. In fact, TEVAR itself can attenuate hypertension in patients with aTBAD [13]. Previous reports confirmed that strict blood pressure control can promote perioperative LV ejection fraction for patients with aTBAD, although their results would be more convincing if taking follow-up outcomes into consideration [14].

Given the specific anatomic structure and peculiar hemodynamic changes, much literature has demonstrated obvious pressure overload before TEVAR in patients with TBAD: pressure gradient increasing between ascending aorta and descending aorta, and continuously increasing during follow-up [15]; ascending flow resistance [16]; apparent energy losses and increased hydraulic resistance felt by the heart [17]; elevated augmentation index of the carotid artery [18]; and FL pressure exceeding TL at peak systole in distal dissection [19]. In fact, TEVAR itself can attenuate the pressure gradient in TBAD [13] by occluding the primary tear, correcting the shape of the collapsed TL, overcoming the pressure load from within the FL, and reducing the pressure gradient along the TL [16,20]. Accumulated evidence has further confirmed that TEVAR can induce LV unloading by means of pressure unloading: decreasing FL pulse pressure, even constantly so during follow-up [21]; reduced FL pressure in the thrombosed FL [22]; sustained increasing pressure gradient between TL and FL during follow-up after TEVAR [23]; and pressure gradient greatly declining along the distal arch region and stented region after TEVAR [16]. Furthermore, many clinical investigations and experimental records have confirmed LVH reversal following LV unloading [24]. To some extent, TEVAR can be a trigger factor for LV remodeling and LVH reversal for patients with aTBAD. It was postoperatively that LV thickness, diameter, volume and mass began to decrease, although without significant difference. Furthermore, our results implied that aortic remodeling was a maintaining factor for LV remodeling and LVH reversal: LV remodeling followed aortic remodeling during follow-up. In this study, TL showed a continuous increased trend in proximal thoracic aorta level and celiac trunk level during follow-up. It was during follow-up that LV thickness and mass significantly decreased. It seems that LV continuous unloading induced by aortic remodeling accounts for LV remodeling and LVH reversal. This finding was further complemented by a previous computational fluid dynamics study in which the pressure gradient along the stented

segment and thoracoabdominal aorta was reported to further decrease along with sustained TL enlargement and constriction removal [16,25].

It should be noted that the FL continually dilated and then compressed the TL in our results, which may further induce TL collapse and increase LV afterload [26]. The adverse FL remodeling may be unfavorable to LV remodeling and LVH reversal. To some degree, TEVAR may only delay the process of aortic aneurysm expansion. The aim of TEVAR for aTBAD is not only to cover the primary entry tear but also to promote aortic remodeling and further prevent extension of dissection. This can infer that patients with aTBAD may achieve preferable clinical outcomes by adopting aggressive TEVAR strategies such as extension of aortic coverage including the streamliner multilayer flow modulator (SMFM) stent and provisional extension to induce complete attachment (PETTICOAT) procedure [29], and by following auxiliary strategies for residual FL including placing coils, Amplatzer vascular plugs, visceral artery stent grafts, or glues [30]. These strategies can further eliminate FL and promote aorta remodeling, but also can further attenuate the pressure gradient along TL and consistently reverse LVH, although previous studies have found that increasing aortic stiffness after TEVAR can result in hypertension and adverse cardiovascular remodeling [8]. However, their conclusions may be not applicable to aTBAD given patient population heterogeneity (e.g., thoracic or abdominal aneurysms, thoracic aorta trauma) [27]. It can be inferred that a stent graft may reach its maximum extent once implanted for thoracic or abdominal aneurysms, and thus increase aortic stiffness, diminish the windkessel function, and further induce LVH [28]. For patients with aTBAD after TEVAR, segments of the stent graft showed a continuous increasing trend during follow-up and pressure gradient gradually being reduced alongside sustained increasing TL [16,25], while LV continuous unloading promoted LV remodeling and LVH reversal during follow-up.

5. Study limitations

The present study has several limitations, based mainly on its retrospective nature. First, not all of the patients are followed up systematically; especially with respect to CTA; this may affect our results regarding aorta remodeling. Second, the small sample size is a major issue. Third, aortic gradients were not purposefully evaluated by transthoracic echocardiography given that the intimal flap did not involve aortic valve; in fact aortic gradients are important factors in evaluating LV afterload. Moreover, the duration of follow-up was not long enough to confirm whether TEVAR can continuously reverse LVH in patients with aTBAD during long-term follow-up.

6. Conclusions

Our study suggests that TEVAR can reverse LV remodeling and LVH in patients with aTBAD during follow-up. Although the freedom from re-interventions remained high in patients with aTBAD after TEVAR, patients managed with TEVAR still showed aortic expansion and disease progression during follow-up. More effective elimination of the FL can improve postoperative survival, further attenuate LV remodeling, and induce LVH regression.

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Disclosures

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