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Type 2 diabetes mellitus reduces clinical complications and mortality in Stanford type B aortic dissection after thoracic endovascular aortic repair: A 3-year follow-up study☆

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ABSTRACT

Background: Previous studies have demonstrated that type 2 diabetes mellitus (T2DM) is negatively correlated with the occurrence of aortic dissection (AD). This study aimed to investigate the effects of T2DM on the prognosis of Stanford type B AD (STBAD) patients after thoracic endovascular aortic repair (TEVAR).

Methods: STBAD patients (n = 141) who underwent TEVAR received an oral glucose tolerance test (OGTT) and were divided into a normal glucose (NG, n = 55) group, an abnormal glucose tolerance (AGT, n = 48) group and a T2DM (n = 38) group according to the results of the OGTT. Data on mortality, clinical complications, left ventricular (LV) remodeling and aortic remodeling were collected during the 3-year follow-up.

Results: Lower mortality and fewer clinical complications after TEVAR were found in the T2DM group than in the NG group. Multivariate linear regression analysis showed that 2-hour postprandial glucose (Glu-2h) was negatively correlated with mortality and the occurrence of clinical complications in STBAD patients after TEVAR. In addition, better LV remodeling, larger true lumen areas and smaller false lumen areas in both the proximal aortas and abdominal aortas were observed in the T2DM group than in the NG group. Furthermore, no significant differences in mortality or clinical complications after TEVAR were found between the NG group and the AGT group or between the T2DM group and the AGT group.

Conclusion: During the 3-year follow-up period, mortality and clinical complications in STBAD patients after TEVAR were significantly reduced in the T2DM group. For STBAD patients who undergo TEVAR, properly relaxing of blood glucose control requirements may be beneficial for their prognosis.

1. Introduction

Aortic dissection (AD) can be divided into Stanford type A aortic dissection (STAAD) and Stanford type B aortic dissection (STBAD) according to whether the aortic arch is included; this distinction provides important guidance for the clinical treatment of AD. STAAD is often treated by aortic replacement, which has the drawbacks of major trauma and high cost [1]. Meanwhile, STBAD is commonly treated by thoracic endovascular aortic repair (TEVAR), which significantly reduces short-term mortality; this procedure, first proposed for the purpose in 1999, quickly gained acceptance and is now widely applied in clinical practice [2,3]. However, since TEVAR can lead to a variety of serious clinical complications and has no obvious advantages in prognosis over conservative treatment in internal medicine, TEVAR has been increasingly questioned in recent years [4].

There are many known risk factors for AD, including hypertension, smoking, drug abuse, atherosclerosis, and genetic diseases [5,6]. Numerous studies have found that nearly 70% of AD patients suffer from

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hypertension, and most of these patients' blood pressure is outside the standard range; accordingly, hypertension has been shown to be the most important risk factor for AD [7]. Type 2 diabetes mellitus (T2DM) has been demonstrated to be closely related to the occurrence of hypertension, and the incidence of hypertension in T2DM patients is twice that in patients with normal glucose levels [8,9]. Given this information combined with the fact that T2DM is a risk factor for a variety of vascular diseases, T2DM was initially considered the most important pathogenic factor for AD, but later evidence refuted this belief. A single-center study published in the *Journal of the American Heart Association* (*JAHA*) in 2012 was the first to show a reduced incidence of T2DM in AD patients, suggesting that T2DM may be a protective factor against the occurrence of AD [10]. The same phenomenon was found in a small single-center sample of Spanish people [11]. In a subsequent meta-analysis published in the journal *Angiology*, the negative association between T2DM and thoracic AD was confirmed for the first time from the viewpoint of evidence-based medicine [12]. In a recent study published in *JAHA*, T2DM was once again shown to be negatively correlated with the subsequent occurrence of AD in the Swedish population when the influences of hypertension were excluded [13]. However, whether T2DM is associated with the prognosis of STBAD patients after TEVAR remains unknown. This study aimed to clarify the effects of T2DM on mortality, clinical complications and aortic remodeling in STBAD patients after TEVAR.

2. Methods

2.1. Study subjects

From May 2014 to May 2015, this study enrolled 158 consecutive patients who were diagnosed with STBAD according to their clinical symptoms and computed tomography angiography (CTA) results and underwent TEVAR at the People's Hospital of Guangxi Zhuang Autonomous Region and Beijing Anzhen Hospital. Among these 158 patients, 17 patients were excluded from the study because they had a history of other diseases that might affect the results of follow-up, including liver cancer ($n = 2$), lung adenocarcinoma (LA, $n = 1$), coronary artery disease (CAD, $n = 6$), valvular heart disease (VHD, n = 2), peripheral arterial disease (PAD, $n = 5$), and chronic heart failure (CHF, n = 1). The remaining 141 patients underwent an oral glucose tolerance test (OGTT) before TEVAR and were then divided into a normal glucose (NG, $n = 55$) group, an abnormal glucose tolerance (AGT, $n = 48$) group and a T2DM ($n = 38$) group based on their OGTT results (as shown in Supplementary Fig. 1). The patients or their families provided informed consent. This study protocol was approved by the Medical Ethics Committee of the People's Hospital of Guangxi Zhuang Autonomous Region and Beijing Anzhen Hospital.

2.2. Implementation of aortography and TEVAR

After surgery-related examination and preparation, patients lay flat on the operating table and received local anesthesia and skin disinfectant. Then, aortography was performed after puncture and intubation of the left brachial artery to obtain a comprehensive understanding of the lesion site, morphology and range of involved structures in each patient. In addition, the diameter of the left subclavian artery (LSA) opening proximal to the aorta and the distance between the primary rupture and the LSA opening were measured. Stents with a diameter was 10%–20% larger than that of the normal aorta at the proximal AD and a length of 10 cm were chosen and used in TEVAR. After the nearby skin was disinfected, one femoral artery was cut open, and a membrane-covered stent was placed through the true lumen space to block the primary rupture. The laminated part of the stent was located below the LSA opening ($n = 101$) when the distance from the original lesion to the LSA opening was > 1.5 cm, while the coated part sealed the LSA opening ($n = 40$) when the primary lesion was < 1.5 cm from the LSA opening. Further information on stent placement and LSA coverage is listed in Table 1.

Table 1
Peri-operative characteristics and postoperative complications in these 3 groups.

Characteristic	NG	AGT	T2DM
Proximal sent graft type			
Shanghai Aegis (n, %)	18 (32.7)	22 (45.8)	13 (34.2)
Jiangsu Vascore (n, %)	21 (38.2)	14 (29.2)	15 (39.5)
Shenzhen Grikin (n, %)	16 (29.1)	12 (25.0)	13 (26.3)
Distal bare stent type			
Aegis (n, %)	4 (7.3)	6 (12.5)	3 (7.9)
Aorta coverage distance (mm)	144 ± 131	152 ± 129	150 ± 134
LSA coverage			
Without coverage (n, %)	42 (76.4)	38 (79.1)	29 (76.3)
Partial coverage (n, %)	8 (14.5)	7 (14.6)	5 (13.2)
Complete coverage (n, %)	5 (9.1)	3 (6.3)	4 (10.5)
Postoperative complications			
Total (n, %)	20 (36.4)	12 (25.0)	5 (13.2)*,#
Proximal SINE (n)	1	1	1
Distal SINE (n)	1	1	0
Endoleak (n)	3	2	1
Ischemic stroke (n)	1	1	0
Spinal cord ischemia (n)	2	0	1
Acute renal failure (n)	2	1	0
ELEA (n)	1	2	1

SINE: stent graft-induced new entry; ELEA: embolism of lower extremity artery.
* $p < 0.05$ vs. NG group.
# $p < 0.05$ vs. AGT group.

2.3. Follow-up

To ensure the smooth progress of follow-up, we implemented two measures. First, all the patients who were enrolled in this study were required to return to the hospital for re-examination once a month after they had received effective treatment and were discharged from the hospital; second, we notified all patients by telephone 2–3 days before each follow-up time point. Patients were readmitted to the hospital and received CTA at 12, 24, and 36 months after TEVAR, and they underwent OGTT every 6 months. In addition, information on mortality, clinical complications and aortic remodeling was collected during the 3-year follow-up. The three follow-up time points (designated T1, T2, and T3) were defined, respectively, as 12 months, 24 months and 36 months after STBAD patients underwent TEVAR in this study.

2.4. Data collection

The patients' clinical characteristics, such as gender, age, smoking, systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR), creatinine (CREA), white blood cell (WBC) count, D-dimer, and C-reactive protein (CRP), were derived from the medical records made on admission. Fasting glucose and 2-hour postprandial glucose (Glu-2h) were obtained from the results of the OGTT. Data regarding the LV structure and function, including LV posterior wall thickness (PWT), LV end-diastolic diameter (LVEDD), LV end-systolic diameter (LVESD), LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV) and LV ejection fraction (LVEF), were collected from the echocardiographic results. Data on the structure of the aorta, including the true lumen and the false lumen, were derived from the results of CTA.

2.5. Statistical analyses

All the data in the present study were analyzed using SPSS 22.0. Data that conformed to the normal distribution were expressed as the mean ± standard deviation (SD), and differences between the means

Table 2
Clinical characteristics in different groups.

Characteristic	NG	AGT	T2DM
Male gender (n, %)	41 (74.5)	34 (70.8)	31 (81.6)
Age (years)	46 (37, 57)	52 (39, 61)*	56 (42, 65)*,#
Smoking (n, %)	21 (38.2)	19 (39.6)	9 (23.7)
HBP (n, %)	37 (67.3)	26 (54.2)	22 (57.9)
SBP (mmHg)	157 (139, 168)	151 (142, 164)	150 (137, 168)
DBP (mmHg)	92 (82, 118)	93 (86, 114)	95 (76, 118)
HR (bpm)	81 (62, 98)	74 (69, 94)	78 (66, 97)
CREA (μmol/L)	89 (74, 116)	98 (82, 124)*	107 (92, 139)*,#
WBC ($\times 10^9$/L)	15.1 (10.7, 22.6)	13.2 (9.5, 16.6)*	11.7 (8.6, 15.9)*,#
D-dimer (μg/ml)	8.9 (2.1, 17.9)	7.0 (1.8, 13.4)*	6.1 (1.1, 11.9)*,#
CRP (mg/ml)	14.1 (1.7, 25.4)	13.7 (1.3, 19.6)*	9.8 (0.7, 17.1)*,#
Metformin use	0 (0)	0 (0)	24 (63.2)*,#

NG: normal glucose; AGT: abnormal glucose tolerance; T2DM: type 2 diseases mellitus; HBP: blood pressure not up to standard; SBP: systolic blood pressure; DBP: diastolic blood pressure; WBC: white blood cell; HR: heart rate; CREA: creatinine; WBC: white blood cell; CRP: C-reactive protein.
* $p < 0.05$ vs. NDM group.
# $p < 0.05$ vs. AGT group.

Table 3
Summary of death in different groups.

Months	NG	AGT	T2DM	Cause of death
3	1	–	–	Retrograde dissection causing AIS
4	–	1	–	Retrograde dissection causing TAAD
6	1	–	–	Renal artery dissection causing ARF
7	–	1	–	Retrograde dissection causing AIS
10	1	–	–	Thoracic aortic rupture
14	1	–	–	Retrograde dissection causing AIS
16	1	–	–	Retrograde dissection causing ALHF
17	–	1	–	Abdominal aortic aneurysm ruptured
18	1	–	–	Renal artery dissection causing ARF
19	–	–	1	Abdominal aortic aneurysm ruptured
20	1	–	–	Abdominal aortic rupture
24	1	1	–	Retrograde dissection causing AIS/thoracic aortic rupture
29	1	–	–	Abdominal aortic aneurysm ruptured

AIS: acute ischemic stroke; TAAD: Stanford type A aortic dissection; ARF: acute renal failure; ALHF: acute left heart failure.

of two groups were compared by Student's *t*-test. Differences between the means of multiple groups were compared by one-way analysis of variance (ANOVA) followed by Tukey's multiple comparison test. Categorical variables are expressed as percentages and were analyzed by a chi-squared test. The Kaplan-Meier method and log-rank test were used to compute and analyze the survival rate and the incidence of complications. A p value < 0.05 was considered significant.

3. Results

3.1. Comparison of clinical characteristics among the NG, AGT and T2DM groups

No differences in clinical characteristics, including gender, smoking, high blood pressure (HBP), SBP, DBP and HR, were detected among the three groups. Age and CREA had a positive relationship with D-dimer and CRP in the NG, AGT and T2DM groups. In addition, the WBC count of the T2DM group was higher than that of the NG group and lower than that of the AGT group. The clinical data for each group are listed in Table 2.

3.2. The T2DM group had the lowest mortality and the fewest clinical complications

During the 3 years of follow-up, 9 patients, 4 patients and 1 patient died in the NG, AGT and T2DM groups, and the final survival rates were 83.6%, 91.7% and 97.4%, respectively. The final survival rate in the T2DM group was significantly higher than that of in the NG group but not higher than that of the AGT group, and no difference in survival was observed between the AGT and NG groups (Fig. 1A). Retrograde dissection and aortic rupture after AD were the most common causes of death in these patients, and the time and cause of death in each deceased patient are listed in Table 3. In addition, the incidence of clinical complications followed a similar trend to the survival rate (Fig. 1B). The clinical complications in each group are listed in Table 1.

3.3. Glu-2h was independently associated with the occurrence of death and clinical complications in STBAD patients after TEVAR

To investigate whether Glu-2h was independently associated with the occurrence of death in STBAD patients after TEVAR, we performed simple linear regression analysis and subsequent multivariate linear regression analysis. Simple linear regression analysis showed that smoking, CRP, Glu and LSA coverage exhibited trends ($p < 0.05$) toward an association with mortality. These variables were used to perform multivariate linear regression analysis, and the results demonstrated that elevated Glu-2h and smoking were independently associated with the occurrence of death in STBAD patients after TEVAR. The β values, 95% CIs and p values are shown in Table 4. The same methods were used to determine whether Glu-2h was independently associated with the occurrence of clinical complications, and the results showed that both age and Glu-2h were independently associated with the occurrence of clinical complications in STBAD patients after TEVAR. The β value, 95% CI and p value are shown in Table 5.

3.4. The T2DM group exhibited better LV remodeling than the NG group

No differences in PWT, LVEDD, LVESD, LVEDV, LVESV and LVEF values obtained before and after TEVAR were observed in the three groups (Fig. 2A–F). From T1 to T3, lower PWT, LVEDD, LVESD, LVEDV

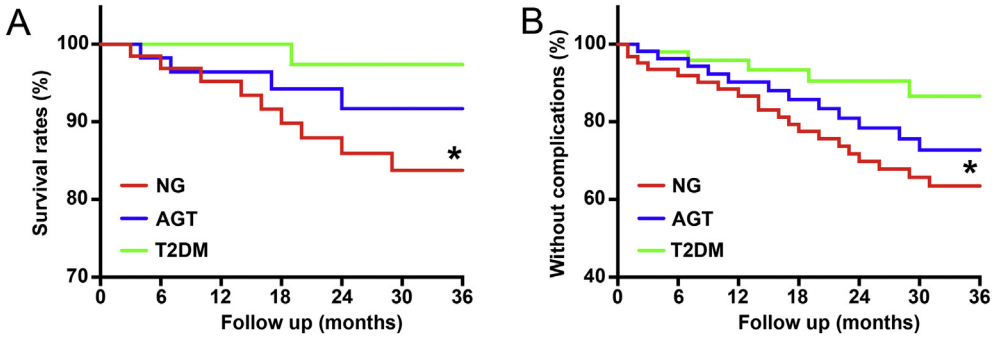

Fig. 1. Effects of T2DM on survival and the incidence of complications. (A). Kaplan-Meier curve for survival in the NG, AGT and T2DM groups. (B). Kaplan-Meier curve for complications in these three groups. *$P < 0.05$ vs. NG group.

Table 4
Association between clinical characteristics and death in STBAD patients after TEVAD were assessed by simple linear regression analysis and subsequent multivariate linear regression analysis.

Variables	Univariate			Multivariate		
	β	95% CI	p	β	95% CI	p
Male	0.098	−0.047 to 0.149	0.221			
Age	0.178	0.064 to 0.292	0.116			
Smoking	0.319	0.158 to 0.480	0.009	0.189	0.097 to 0.281	0.032
HBP	0.298	0.142 to 0.454	0.108			
HR	0.010	−0.106 to 0.084	0.215			
CREA	−0.094	−0.192 to −0.04	0.689			
WBC	0.118	0.026 to 0.210	0.484			
D-dimer	0.182	0.069 to 0.295	0.126			
CRP	0.374	0.196 to 0.552	0.046	0.219	0.111 to 0.327	0.218
Glu	−0.424	−0.699 to −0.169	0.001	−0.263	−0.375 to −0.151	0.007
LSA	−0.205	−0.514 to −0.104	0.028	−0.112	−0.305 to −0.081	0.106
Metformin use	−0.223	−0.541 to −0.081	0.141			

and LVESV and higher LVEF values were observed in the T2DM group than in the NG group (Fig. 2A–F). Furthermore, no significant differences in PWT, LVEDD, LVESD, LVEDV, LVESV and LVEF values measured from T1 to T3 were found between the NG group and the AGT group or between the T2DM group and the AGT group (Fig. 2A–F).

3.5. The development of the aortic false lumen was slowest in the T2DM group

Regarding the area of the true lumen in the proximal thoracic aorta, no difference was found before TEVAR among the NG, AGT and T2DM groups, but a difference gradually emerged from T1 to T3 (Fig. 3A). The true lumen areas in the T2DM group were larger than those in the NG group at the same follow-up time points but not larger than those in the AGT group, although no difference was observed between the NG and AGT groups (Fig. 3A). The area of the true lumen in the proximal thoracic aorta showed similar trends (Fig. 3B). Regarding the area of the false lumen in the proximal thoracic aorta, no difference was found among these three groups before TEVAR. The false lumen areas were all favorably reduced at T1, with that of the T2DM group decreasing most dramatically (Fig. 3C). From T2 to T3, the false lumen areas rapidly increased and were even larger than they had been before TEVAR; the increase was least pronounced in the T2DM group, while no differences were observed between the NG and AGT groups (Fig. 3C). The false lumen areas in the celiac trunk, gradually increased in all three groups during the follow-up and was significantly larger in the T2DM group than in the NG group, but there was no significant difference between the T2DM and AGT groups in this respect (Fig. 3D).

4. Discussion

In the present study, we found that the incidence of clinical complications and the rate of mortality in STBAD patients after TEVAR were significantly lower in those with T2DM than in those with normal glucose. Multivariate linear regression analysis showed that Glu-2h was independently associated with the occurrence of both death and clinical complications in STBAD patients after TEVAR. In addition, better LV remodeling was observed in T2DM group than in the NG group, but LV remodeling was not better than that in the AGT group. Furthermore, we also detected changes in the true and false lumen areas in both the proximal thoracic aorta and the proximal thoracic aorta at different time points. The results showed that the postoperative true lumen areas of the proximal and proximal thoracic aorta in the T2DM group both increased more quickly than those of other groups, while the opposite was true of the false lumen areas. These data may suggest that T2DM reduces the occurrence of clinical complications and mortality in STBAD patients after TEVAR. In patients with STBAD, it may be beneficial to relax the control of blood glucose after TEVAR.

The occurrence of AD is the result of the interaction of a variety of complicated pathological processes, and various pathological factors and risk factors can affect its prognosis [14–18]. T2DM is a risk factor for a variety of vascular diseases and was once considered a risk factor for AD because T2DM can lead to hypertension, which promotes the development of AD [19]. However, surprisingly, clinical observation showed that the incidence of AD in T2DM patients was significantly lower than that in patients with normal glucose and linear regression also showed that the occurrence of AD was negatively correlated with T2DM [10–13]. Furthermore, increasing evidence from clinical

Table 5
Association between clinical characteristics and complication occurrence in STBAD patients after TEVAD were assessed by simple linear regression analysis and subsequent multivariate linear regression analysis.

Variables	Univariate			Multivariate		
	β	95% CI	p	β	95% CI	p
Male	0.145	0.062 to 0.228	0.174			
Age	0.189	0.098 to 0.280	0.005	0.081	0.042 to 0.120	0.044
Smoking	0.209	0.126 to 0.292	0.014	0.101	0.068 to 0.134	0.128
HBP	0.119	0.037 to 0.201	0.029	0.058	0.026 to 0.090	0.364
HR	−0.104	−0.216 to −0.008	0.621			
CREA	−0.122	−0.282 to −0.038	0.098			
WBC	0.227	0.178 to 0.276	0.162			
D-dimer	0.198	0.108 to 0.288	0.315			
CRP	0.222	0.138 to 0.306	0.044	0.108	0.072 to 0.144	0.582
Glu	−0.292	−0.647 to −0.063	0.007	−0.136	−0.398 to −0.126	0.019
LSA	0.109	−0.084 to 0.134	0.418			
Metformin use	−0.240	−0.508 to 0.006	0.056			

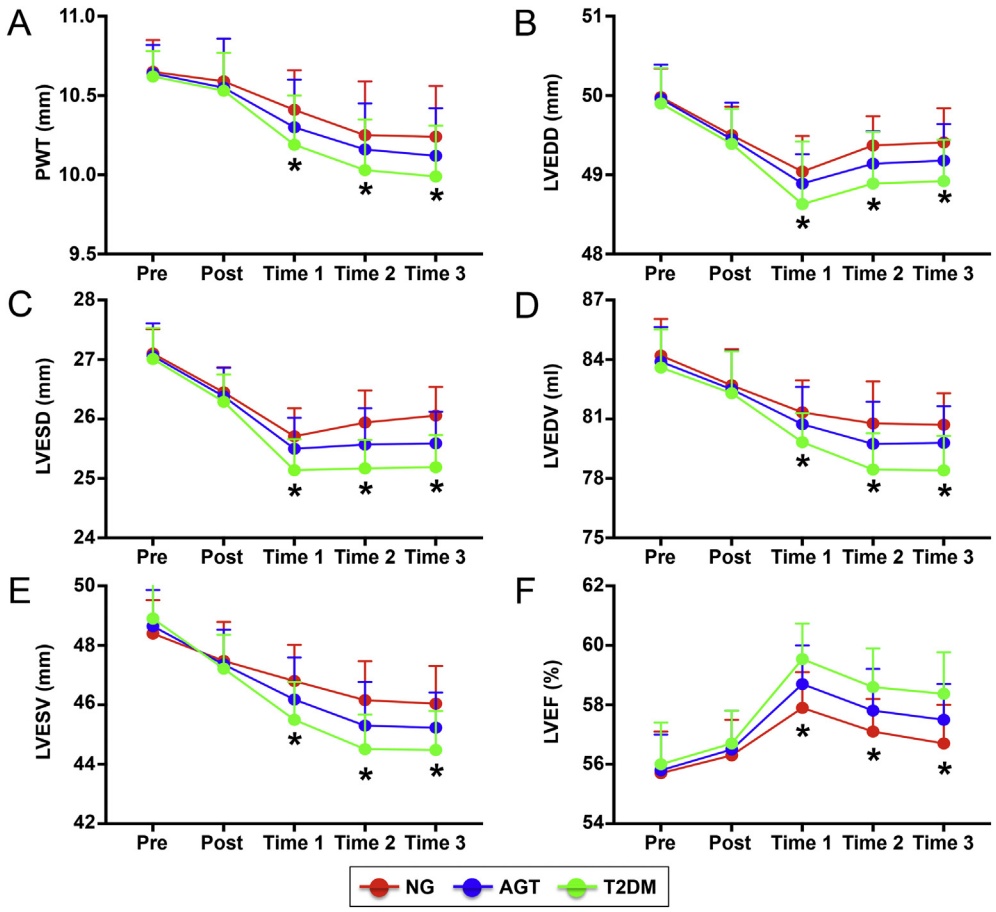

Fig. 2. LV structure and function in each group. (A). PWT, (B). LVEDD, (C). LVESD, (D). LVEDV, (E). LVESV and (F). LVEF in the NG group, AGT group and T2DM group. *P < 0.05 vs. the NG group.

experiments has shown in recent years that T2DM can unexpectedly reduce the incidence of both STAAD and STBAD [11–13,20]. However, the effect of T2DM on the prognosis of acute STBAD patients after TEVAR remains unreported. In the present study, we divided the STBAD patients who received TEVAR into three groups according to OGTT results and found that the rates of mortality and clinical

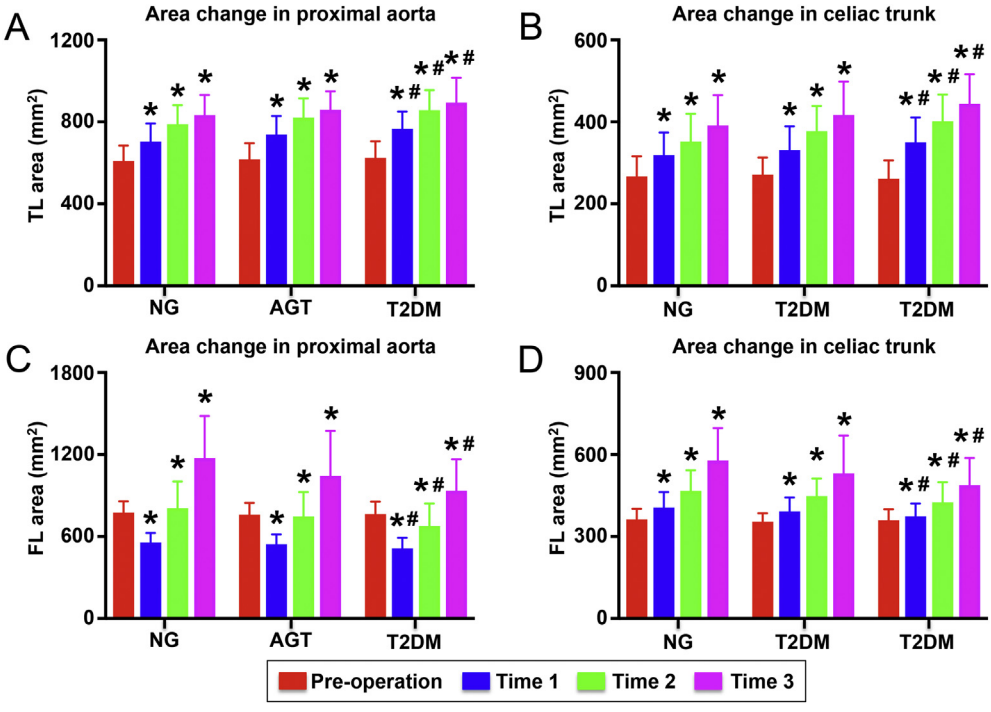

Fig. 3. True and false lumen areas in the NG, AGT and T2DM groups. (A, B). True lumen areas in the proximal aorta and celiac trunk were measured preoperatively and at Time 1, Time 2 and Time 3; (C, D). False lumen areas in the proximal aorta and celiac trunk were measured at different time points. *P < 0.05 vs. previous time points; #P < 0.05 vs. NG group.

complications were both significantly lower in the T2DM group than in the NG group. These results indicate that T2DM can significantly improve the prognosis of acute STBAD patients after TEVAR. In addition, we found that the of in the AGT group was not significantly different from that of the T2DM group and NG group. The reason for this phenomenon may be that the sample size was relatively small, and further expansion of the sample size may be necessary. AD and abdominal aortic aneurysm share the same pathological mechanisms. Numerous studies have demonstrated that the oral hypoglycemic drug metformin significantly reduces the occurrence and progression of abdominal aortic aneurysm [21,22]. These results seem to be contrary to the previous conclusions. One possible reason for these findings is that the anti-aneurysm effect of metformin is not achieved by lowering the blood glucose level but is related to its anti-inflammatory effect, the reduction of inflammatory cell infiltration into the aortic wall and its protective effect against aortic smooth muscle cell injury. Sulfonylurea therapy did not exhibit an anti-aneurysm effect and may even play an opposite role, partly explaining these contrary results [22].

The pathogenesis of AD is a dramatic progression of hemodynamic change, and the main clinical treatment measures, including surgical treatment and conservative treatment, are aimed at eliminating abnormal hemodynamics, which may directly affect the prognosis of AD [5,23,24]. The effects of hypertension or anti-hypertensive drugs on the prognosis of AD are thought to be achieved by influencing hemodynamics [19,20]. The constant onslaught of abnormal blood flow is especially likely to aggravate hemodynamic disorder after the occurrence of AD in the elderly because the compliance of the aortic wall deteriorates with age [25,26]. In AD patients, the changes in both the true and false lumen areas are closely related to hemodynamics, and the smooth recovery of the true lumen is crucial to reduce the overload pressure on the heart and promote the recovery of cardiac function [27], while the area of the false lumen directly affects mortality and the incidence of clinical complications [28]. In addition, a large number of clinical studies have suggested that the long-term prognosis of STBAD patients after TEVAR is related to aortic remodeling, and partial thrombus of the false lumen is an independent risk factor affecting the long-term prognosis of STBAD patients after TEVAR [29–31]. A study based on the VIRTUE registry indicated that aortic remodeling is more favorable in the acute stage and the subacute stage than in the chronic stage, and the difference is reflected in the degree of narrowing of the false lumen areas [32]. The ideal outcome of aortic remodeling after TEVAR treatment is that the true lumen is fully dilated, the false lumen completely disappears, and the histological continuity of the medial membrane is restored. Therefore, we also determined the effect of T2DM on AD. We began by examining fasting glucose and Glu-2h at different time points after TEVAR, and we found that there was no significant difference in fasting glucose between the NG and AGT groups, while both had lower levels than the T2DM group, although the fasting glucose levels in the T2DM group gradually decreased after treatment (Supplementary Fig. 2A). Glu-2h increased gradually in the NG, AGT and T2DM groups across all time points (Supplementary Fig. 2B). The multivariate linear regression analysis also demonstrated that Glu-2h was independently associated with the occurrence of clinical complications and mortality in STBAD patients after TEVAR. These results suggest that a better prognosis due to T2DM may be associated with better aortic remodeling, especially slowing the growth of the false lumen, in STBAD after TEVAR. The true lumen area, the functional area of the aorta, is closely related to the structure and function of the heart [27]. In STBAD patients, the false lumen area is increased, which significantly decreases the true lumen area. This can aggravate the pressure load on the heart, leading to changes in the structure and function of the heart, which are manifested as cardiac hypertrophy, LV remodeling and decreased cardiac function [27,28]. To further illustrate our conclusions, we observed the changes in cardiac structure and function in STBAD patients after TEVAR, even in patients who did not exhibit typical symptoms. The results showed that at 12 months after TEVAR, the cardiac structure and function were significantly improved in both the NG and T2DM groups, but the T2DM group showed a greater improvement and corresponding changes in the true lumen areas. At 12 to 36 months after TEVAR, the cardiac structure and function exhibited a deteriorating trend, even though the true lumen areas continued to recover. These results suggest that a sudden increase in false lumen area may affect cardiac structure and function. Therefore, the goal of treatment for STBAD patients is not only to restore the true lumen but also to delay the progression of the false lumen.

However, the evidence on the role of T2DM in AD is not completely consistent, and a recent study showed that T2DM was an independent risk factor for death in STBAD patients who did not receive interventional therapy [22]. In addition, data from clinical experiments and animal studies indicate that some hypoglycemic drugs can also reduce the incidence of AD [33]. There may be a factor that is not yet known in the complex microenvironment of T2DM, and further research is necessary. In STBAD patients after TEVAR, lower blood sugar control requirements may be beneficial for reducing complications and improving survival rates.

5. Conclusions

In STBAD patients after TEVAR, T2DM may lead to better aortic remodeling and an improved prognosis; appropriate relaxation of glucose management may be necessary.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.lfs.2019.05.055.

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