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Pursuit of the Changes of the Descending Aortic False Lumen after Surgical Repair of Acute DeBakey Type 1 Aortic Dissection

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ABSTRACT

Background: After surgical repair of acute DeBakey type 1 aortic dissection (AIAD), most survivors suffer from persistent false lumen in the descending aorta that adversely affects the long-term prognosis, and the need for secondary intervention is prompted. **Objective:** This study was aimed at providing control data to determine the actual benefits of more aggressive or newer approaches by investigating the changes in the descending aortic false lumen over 1-year follow-up. **Patients and Methods:** This retrospective study included 83 patients with AIAD ± significant aortic incompetence (AI). On emergency basis, all the patients had undergone aggressive resection of the intimal tear and replacement of the ascending aorta ± proximal or total aortic arch replacement, and aortic valve (AV) repair, replacement, valve sparing re-implantation of the AV or composite replacement of the aortic root (depending on the encountered pathology). Multi-slice CT aortography (MSCT) at 6-months and 1-year was done for segmental analysis of the distal aortic diameter at fixed levels and false lumen status evaluation. **Results:** The mean age was 47.18±4.87 years. Mortality was 8(9.64%) intraoperatively, 10(12.05%) operative mortality, no late mortality and the overall 1-year survival rate was 65(78.31%). The overall hospital complications rate was 17(22.67%). The descending aortic false lumen remained patent in most [56(86.15%)] survivors. There was no dilatation of the descending aorta at 6-months. At 1-year, there was statistically significant ($p<0.001$) dilatation of the descending aorta of >10 mm in 24(42.86%) and <10 mm in 32(57.14%) in patients with persistent false lumen. Patients <40 years expressed statistically significant ($p<0.001$) total resolution of the false lumen and >10 mm dilatation of the descending aorta. Multivariable logistic regression analysis showed that the significant predictive risk factors for persistence of the false lumen in the descending aorta were age <50 year (OR: 3.10 (95% CI: 0.846-19.320); $p= 0.041$), Bentall operation (OR: 0.35 (95% CI: 0.026-2.087); $p= 0.021$) and long-term anticoagulation (OR: 0.19 (95% CI: 0.025-1.289); $p= 0.043$), and those for thrombosis of the false lumen were age >60 years (OR: 6.13 (95% CI: 5.123-34.162); $p= 0.031$) and valve-sparing aortic root reconstruction (OR: 4.23 (95% CI: 1.480-14.053); $p= 0.007$). **Conclusion:** Residual intimal tear in the distal descending aorta is the major cause of continued false lumen patency. More extensive aortic replacement and avoidance of surgical reconstruction procedures needing long-term anticoagulation could diminish the prevalence of patent false lumen and its adverse sequelae.

Keywords: Dilated descending aorta, Patent false lumen, Persistent false lumen, MSCT, Aortic dissection, Marfan syndrome.

INTRODUCTION

Markedly obvious and steady improvements have occurred over the last two decades in the postoperative surgical outcomes for repair of acute DeBakey type 1 aortic dissection (AIAD) [(1)].

Persistent false lumen post-conventional surgery in the downstream aorta has affected most AIAD candidates, which may have a negative impact on their long-term prognosis [(2)].

While several previous research investigated the descending aortic false lumen and risk factors for aneurysmal dilatation, very few focused on differences or changes that began with immediate postoperative features and extended to subsequent, longer-term alterations. Furthermore, most earlier research used antiquated methods such as limited replacement of the ascending aorta solely and anastomosis under aortic cross-clamping without complete circulatory arrest (TCA), even when an aortic arch rupture was present[(3)].

Thus, we lack sufficient control data to establish the real value of more aggressive or novel treatments, such as regular aortic arch replacement or insertion of a frozen elephant trunk, which are believed to enhance long-term prognosis.

This study was aimed at providing such control data by investigating the changes in the descending aorta false lumen and its differences over one-year follow-up.

PATIENTS AND METHODS

This retrospective observational non-randomized study included a total of 83 patients with AIAD with or without significant aortic incompetence (AI), attending at Department of Cardiothoracic Surgery, Cairo University Hospitals. This study was conducted between June 2018 to May 2023.

The patients mean age was 47.18±4.87 years (range: 34-62). On emergency basis surgery, all the

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patients had undergone aggressive resection of the intimal tear and replacement of the ascending aorta. Depending on the pathology encountered, the proximal repair included resuspension of the AV, aortic valve replacement (AVR) or repair, valve sparing re-implantation of the AV, composite replacement of the aortic root, replacement of the proximal aortic arch, or total arch replacement. Distal anastomoses were performed during a period of total circulatory arrest (TCA) when the aortic arch was included in the repair or without TCA if repair was confined to the ascending aorta only.

The survivors had serial multi-slice CT aortography (MSCT) at 6 months and 1 year postoperatively for segmental analysis of distal aortic diameter at fixed levels and false lumen status evaluation.

Inclusion and Exclusion Criteria

All cases of AIAD with tear in the ascending aorta, with or without AI, and with or without pericardial collection were included. Patients having prior open-heart surgery (redo patients), recent preoperative myocardial infarction (MI) or cardiomyopathy, compromised cardiac functions, and organic valvular abnormalities (rheumatoid or active endocarditis) were excluded [LV ejection fraction per cent (LVEF%) less than 40%].

Management Regimen

Preoperatively

Preoperative factors that were evaluated included assessing a patient's medical history, age, sex, hypertension, diabetes mellitus (DM), renal problems, smoking, dyslipidemia, and history of stroke or MI. Clinical examination: vital signs (blood pressure and pulses of upper and lower limbs for hypertension, big pulse volume, absent femoral pulsations), Laboratory investigations: complete blood count, liver and kidney functions, coagulation profile, cardiac examination for evidence of newly onset AI murmur, chest and neurological examination, resting 12-leads electrocardiogram (ECG) for heart rate and any associated ischemia or arrhythmias, Investigations following a fixed scan regimen, including chest X-ray (CXR), duplex of carotid and femoral arteries, echocardiography, and radiological investigations[transthoracic (TTE) and/or transesophageal (TEE)] to assess left ventricular dimensions and function [LVEDD, LVESD, and LVEF%], AV morphology and degree of AI [graded from 1 to 4 based on the ratio of the jet height to LVOT height] as well as the measurements of the aortic root (aortic annulus, mid sinuses diameter, sinu-tubular junction diameter, ascending aortic diameter).

(In 2D, M-mode, PW, CF mapping studies using parasternal long and short axis and apical 4 chamber views, the commercially available echo-Doppler system is a 2.5 mega/Hz transducer); additionally, magnetic resonance imaging (MSCT) of the thoracic aorta (for ascending, arch, and descending aorta diameter) and coronary arteries (to detect any associated coronary lesions; for patients with renal impairment, magnetic resonance angiography (MRA) was utilized).

Intraoperatively

The assessed operative variables included intraoperative mortality, determination of the intimal tear site, type of the operative procedure, total operative time, CPB time, total aortic cross clamping time and TCA time.

Operative Technique

It was a median sternotomy. After the patient was put on CPB, which was accomplished by femoro-atrial cannulation, moderate hypothermia, which reached 24°C, was brought on. For retrograde cerebral perfusion, cerebral protection was achieved through the superior vena cava (SVC); for antegrade cerebral perfusion, it was accomplished by the axillary artery. When TCA was required, cardioplegic perfusion via the coronary sinus was used to keep the myocardium's temperature below 15°C (59°F), and it was kept at 28°C otherwise. This shielded the heart during the whole process. By decompressing the left ventricle and releasing air through the left superior pulmonary vein or artery, ventricular distention was prevented. When the nasopharyngeal temperature hit 20°C and the EEG became isoelectric, the pump was turned off. The SVC was then used to initiate retrograde cerebral perfusion. The location, size, and involvement of the aortic arch were examined in order to determine whether an intimal disruption necessitating repair had occurred in the ascending aorta. The ascending aorta was opened and transected in close proximity to the innominate artery using a longitudinal technique. When the aortic arch was involved, patients had either the proximal or entire arch replaced; if there was evidence of rupture or fragmentation of the intima, the entire arch was replaced. The adventitia and intima were sutured together using fine 4/0 and 5/0 polypropylene sutures if the aortic arch was clear of re-entry.

A gelatin or collagen woven Dacron graft was end-to-end sutured to the strengthened proximal aortic arch, and 4/0 polypropylene pledgeted sutures were used for internal and external reinforcement. Retrograde cerebral perfusion was halted at the point of distal anastomosis completion, and CPB was recommenced through the femoral artery. This cleared the brachiocephalic vessels of any air and debris. The graft was constricted close to the innominate artery's origin. Clamping the graft at the innominate artery's origin allowed blood flow to return to the brain and the body. One useful technique for treating aortic dissection was hypothermic TCA. One significant risk connected with this treatment is air embolism, which can result in difficulties when major arteries are emptied because it allows air to enter. Therefore, we took care to ensure

that the patient's head was depressed rather than raised, allowing blood to flow into the head veins and pushing air (upward) to the periphery. This was really important. If there was no sign of aortic root dilatation and the AV was suspended with 4/0 polypropylene pledgeted sutures, then everything was okay. The aorta's adventitia and intima were strengthened from within the graft and sutured together above the coronaries.

A composite valve graft was inserted if the root or the AV were dilated. Reattaching the coronary arteries involved the use of a button or a modified Cabrol method. Restoring valvular competence and allowing the aortic leaflets to resuscitate may have only needed to decompress the artificial lumen when AI was present. Pledgeted sutures were often used to resuscitate the commissures after the two layers of the dissected aorta wall were approached. Prosthetic AVR could also be required in some circumstances. And if distal anastomosis was done on TCA, another graft was needed for the proximal anastomosis. Then the proximal and distal grafts were sutured together. If the dissected root was dilated with healthy leaflets, valve sparing re-implantation of the AV was performed. Defibrillation was used to return the patient to sinus rhythm once the surgery was finished. Next, CPB was weaned off the patient.

Postoperatively

Acute respiratory distress syndrome (ARDS), pulmonary infection, pulmonary atelectasis, bleeding complications (re-exploration to control bleeding or relieve cardiac tamponade), respiratory complications (pulmonary infection, pulmonary atelectasis, acute respiratory distress syndrome [ARDS] and respiratory failure: prolonged ventilation >48 hours postoperatively, re-intubation or tracheostomy), pleural or pericardial effusions, and operative mortality (defined as death in the first 30 days following surgery) were among the variables assessed postoperatively, The criteria for determining the overall hospital complication rate (ARF) were as follows: an increase in the creatinine level (absolute ≥ 0.3 mg/dl, percentage ≥50%), the need for renal replacement therapy or dialysis (excluding patients who required dialysis prior to the surgery), deep wound infections that occur within the first 30 days following surgery and extend beyond the deep tissue plane with positive bacterial cultures and purulent discharge, and superficial wound infections, total hospital stay and radiological follow-up for the discharged survivors: serial follow-up MSCT at 6 months and 1 year duration for (1) segmental analysis of the descending aortic diameter at fixed points including proximal descending aorta after origin of the left subclavian artery, descending aorta diameter at the pulmonary artery bifurcation level and diameter at the level of the diaphragm, and (2) evaluation of the false lumen patency status as patency of the false lumen after surgery is a risk factor for expansion of the descending aortic diameter and need for further intervention. Patent/Persistent false lumen is defined as equal contrast enhancement of both true lumen and false lumen with no thrombus formation while thrombosed and partially thrombosed false lumen is defined as absent or weak contrast enhancement of false lumen with thrombus formation.

Ethical Approval

Written informed consent of all patients was obtained preoperatively. Approvals of the Scientific Committee of the Department of Cardiothoracic Surgery, the Scientific Committee of the Faculty of Medicine, and the Research Ethics Committee of the Faculty of Medicine, Cairo University were all obtained, and its registration number is N-317-2023 on 20-09-2023. The Helsinki Declaration was followed throughout the study's conduct.

Statistical Analysis

The collected data was arranged, tabulated, and statistically examined using SPSS V. 21.0. Fischer's exact test or the appropriate Chi-square test were used to calculate the frequency and percent distributions for the qualitative data. The quantitative data's mean, standard deviation, lowest and maximum values were compared using the t-student test. The correlation between the parameters was ascertained using the Spearman's rank correlation coefficient. Multivariable logistic regression analysis was used to analyse the predicted risk variables for either thrombosis or persistence (patency) of the distal false lumen in the descending aorta. When necessary, Bonferroni adjustments were employed for multiple tests. When the p-value was equal to or less than 0.05, it was deemed significant.

RESULTS

Preoperative Data: (Table 1)

The study involved 83 patients. Their mean age was 47.18±4.87 years (range: 34-62). All of them had chest pain at the presentation. None had undergone previous cardiac surgery or associated valvular pathological affection other than the AV. All of them were admitted to the ICU on an emergency basis for urgent surgical preparation.

Table (1): Preoperative data.

Gender:	
Males (%)	56(67.47%)
Females (%)	27(32.53%)
Age groups distribution:	
>40 years old (%)	53(63.86%)
<40 years old (%)	30(36.14%)
Risk factors:	
Hypertension (%)	69(83.13)
Smoking (%)	44(53.01)
DM (%)	48(57.83)
Dyslipidemia (%)	36 (43.37)
History of stroke (%)	11(13.25)
COPD (%)	3(3.61)
AF (%)	12 (14.45)
History of chronic renal disease (%)	5(6.02)
Peripheral vascular disease (%)	2(2.41)
Marfan syndrome (%)	21(25.30)
Laboratory findings:	
Mean FBG level (mg/dl)	157.20±31.09
Mean creatinine level (mg/dl)	1.44±0.89
Mean Hb (g/dl)	11.5±2.69
Mean INR	1.3±0.7
Echocardiography findings:	
AI (None/Mild/Moderate /Severe) (%)	[3(3.61)/1(1.20)/35(42.17)/44(53.01)]
BAV (%)	8(9.64)
Mean aortic annulus (mm)	23.81±2.05
Mean aortic root (mm)	47.9±12.9
Mean LVEF%	50.98±10.13
Mean LVEDD (mm)	60.12±8.09
Mean LVESD (mm)	42.03±5.87
Pericardial effusion (%)	49(59.04)
SWMAs (%)	15(18.07)
Electrocardiogram findings:	
Normal sinus rhythm (%)	63(75.90)
Strain pattern (%)	18(21.69)
Old MI (%)	2(2.41)
MSCT findings:	
Mean ascending aorta diameter (mm)	59.03±12.08
Flap extent into ascending aorta (%)	1(1.20)
Flap extent into aortic arch (%)	4(4.82)
Flap extent into descending aorta (%)	78(93.97)

DM: diabetes mellitus; COPD: chronic obstructive pulmonary disease; AF: atrial fibrillation; FBG: fasting blood glucose; Hb: hemoglobin; INR: international neutralized ratio; AI: aortic incompetence; BAV: bicuspid aortic valve; LVEF%: left ventricular ejection fraction per cent; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; SWMAs: segmental wall motion abnormalities; MI: myocardial infarction; MSCT: multi-slice CT aortography.

Operative Data: (Table 2)

The intimal tear site was found in the ascending aorta in 70(84.34%) patients and in the aortic arch in 13(15.66%) patients. In all patients, the ascending aorta was replaced by a synthetic graft in addition to AV surgery (AVR or AV repair). 50(60.24%) patients had undergone Bental operation, 30(36.14%) patients had undergone Tirone David operation, and 3(3.61%) patients had undergone supra-coronary conduit operation. For 59(71.08%) patients, replacement was limited to the ascending aorta till the innominate artery and for the rest 24(28.92%) patients, the hemiarch was replaced.

Table (2): Operative Data.

Mean operative time (min.)	433.20±88.80
Mean aortic cross clamping time (min.)	141.60±22.80
Mean CPB time (min.)	208.40±24.60
Mean TCA time (min.)	19.88±4.50
Intraoperative mortality (%)	8(9.64)
Mortality in age group >40 years old (%)	5(6.02)
Mortality in age group <40 years old (%)	3(3.61)

CPB: cardiopulmonary bypass; TCA: total circulatory arrest.

Postoperative Data: (Table 3)

All the operative mortalities happened during the ICU period. The remaining 65(78.31%) survivors were discharged to the usual ward after stabilizing their hemodynamics and stopping inotropic supports safely. In-hospital postoperative echocardiography revealed mean LVEF% 48.78±7.69, 52(80%) patients without residual AI, 6(9.23%) patients with residual trivial AI, 7(10.77%) patients with residual mild AI, and non with residual more grade of AI.

Table (3): Postoperative Data.

Mean duration of mechanical ventilation (hours)	29.08±14.53
Mean duration of inotropic support (hours)	49.20±12.87
Mean total blood loss (ml)	940.50±720.50
Mean total duration of ICU stay (hours)	87.68±22.19
CVAs (%)	14(18.67)
Bleeding complications (%)	7(9.33)
ARF (%)	15(20.00)
Prolonged ventilation >48 hours (%)	7(9.33)
Superficial wound infections (%)	8(10.67)
Deep wound infections (%)	2(2.67)
Pericardial effusion (%)	14(18.67)
Sternal dehiscence	2(2.67)
The overall hospital complication rate (%)	17(22.67)
Operative mortality (%)	10(12.05)
Mortality in age group > 40 years old (%)	5(6.02)
Mortality in age group < 40 years old (%)	5(6.02)
Mean total duration of hospital stay (days)	13.28±5.09

ICU: intensive care unit; CVAs: cerebrovascular accidents; ARF: acute renal failure.

Notably, there was statistically significant difference regarding total resolution of the false lumen in the descending aorta and the mean diameter of the descending aorta at 1-year follow-up, no dilatation of the descending aorta was noticed at 6-months, the thrombosed false lumen showed minimal retraction 0.2 mm in its diameter or no dilatation, and patients with persistent false lumen at 1-year MSCT follow-up expressed statistically significant dilatation of the descending aorta of more than 10 mm in 24(42.86%) and less than 10 mm in 32(57.14%) **(Table 4)**.

Table (4): Changes of the false lumen and descending aorta dilatation at 6-months and 1-year postoperatively by MSCT.

	At 6-months (n=65)	**At 1-year (n=65)**	***P* value**
Still patent (Persistent) (%)	60(92.31)	56(86.15)	0.385
Thrombosed (%)	5(7.69)	3(4.61)	0.432
Totally resolved (%)	0	6(9.23)	**<0.001**
Mean diameter of the descending aorta (mm)	29.10±2.98	36.25±5.87	**<0.001**
***Descending aorta dilatation:**			
>10 mm (%)	0	25(38.46)	**<0.001**
<10 mm (7 mm or less) (%)	0	37(56.92)	**<0.001**
Not dilated or minimally retracted (0.2 mm) (%)	0	3(4.61)	**<0.001**

***** Measurement done at the maximal dilatation level.

MSCT at one-year follow-up showed that all patients with Marfan syndrome expressed dilatation of the descending aorta >10 mm with patent (persistent) false lumen. It also showed that patients aged <40 years expressed statistically significant higher rates of both total resolution of the false lumen and more than 10 mm dilatation of the descending aorta. Meanwhile, patients aged >40 years expressed statistically significant higher rates of both thrombosed false lumen and consequent minimal retraction 0.2 mm in the descending aorta diameter or no dilatation and less than 10 mm dilatation of the descending aorta **(Table 5)**.

Table (5): Changes of the false lumen and descending aorta dilatation in different age groups at 1-year postoperatively.

	Age <40 years (n=22)	**Age >40 years (n=43)**	***P* value**
Still patent (Persistent) (%)	18(81.81)	38(88.37)	0.190
Thrombosed (%)	0	3(6.98)	**<0.001**
Totally resolved (%)	4(18.18)	2(4.65)	**<0.001**
***Descending aorta dilatation:**			
>10 mm (%)	16(72.73)	9(20.93)	**<0.001**
<10 mm (7 mm or less) (%)	6(27.27)	31(72.09)	**<0.001**
Not dilated or minimally retracted (0.2 mm) (%)	0	3(6.98)	**<0.001**

***** Measurement done at the maximal dilatation level.

Multivariable logistic regression analysis showed that the significant predictive risk factors for persistence (patency) of the distal false lumen in the descending aorta were age less than 50 years old, Bentall operation and long-term anticoagulation, and the significant predictive risk factors for thrombosis of the false lumen were age more than 60 years old and valve-sparing aortic root reconstruction procedure **(Table 6)**.

Table (6): Predictors of persistence and thrombosis of the distal false lumen in the descending aorta by multivariable logistic regression analysis.

Risk Factor	OR	*p* Value	95% CI
Persistent (patent) distal false lumen			
Age <50 years	3.10	0.041	0.846–19.320
Bentall operation	0.35	0.021	0.026–2.087
Long-term anticoagulation	0.19	0.043	0.025–1.289
Thrombosed distal false lumen			
Age >60 years	6.13	0.031	5.123–34.162
Valve-sparing aortic root reconstruction procedure	4.23	0.007	1.480–14.053

OR: odds ratio; CI: confidence interval.

The whole duration of the study was 4.92 years. No late mortality was recorded over one-year follow-up and the overall survival rate was 65(78.31%). At one-year follow-up MSCT, there was 1(1.54%) survivor who developed renal failure and was in need of regular dialysis sessions had persistent false lumen. Also, there were 2(3.08%) more survivors with lower limb

ischemic changes. One had persistent false lumen and the other had thrombosed false lumen.

DISCUSSION

By replacing the ascending aorta and preventing its sudden rupture, treating AIAD, and guiding blood flow into the actual aortic lumen to prevent malperfusion, the primary goals of emergency surgery are to preserve the patient's life [4]. The dissection involves the aortic arch extending beyond the ascending aorta in 70% of patients [5]. Although the gold standard operation for AIAD is hemiarch replacement, descending (distal) aortic dilatation and need for reoperation was reported by multiple previous studies. The presence of a false lumen in the descending aorta following surgical correction of AIAD reduces distal aortic diameter and necessitates additional surgery. It was ascribed to the existence of unresected primary tears in the aortic arch, which resemble(s) a highly important risk factor for poor long-term prognosis [6].

Although the etiology of descending aortic dilatation is multifactorial [3], the primary reason for persistent false lumen patency is thought to be the persisting intimal rip in the descending aorta. **Ikeno *et al.*** [7] demonstrated that remaining descending aortic dilatation was caused in part by an anastomotic leak that occurred after hemiarch replacement for AIAD. **Heo *et al.*** [8] shown that aortic adverse events are higher when there is persistent postoperative supra-aortic re-entry in the arch vessels.

Post-surgical perfusion of the false lumen is the most significant risk factor for continued dilatation of the dissected descending aorta after AIAD repair. The distal (descending) aortic segments are thought to include the greater total number of contacts between lumina, which explains the situation. increased false lumen perfusion in the distal aortic segments as a result. The aortic arch does not widen as quickly as the proximal descending aorta because it is stabilized cranially by the supra-aortic arteries and proximally by the distal anastomosis [9]. The cumulative number of connections in the aortic arch and the rate of dilatation of the dissected descending aorta were found to be positively correlated by **Rylski *et al.*** [10]. This result emphasizes the need for more thorough aortic arch repair and careful communication identification, along with close CT monitoring that is required to lower the risk of reoperation away from the graft.

Furthermore, **Zhang *et al.*** [11] computational fluid dynamic (CFD) analysis demonstrated that the distal (descending) aortic segments had a greater pressure differential between the genuine and false lumina than the proximal ones. In a previous work, **Tse *et al.*** [12] used CFD simulations to infer that additional dilatation of the dissected aorta might be caused by a large inter-luminal pressure gradient. Therefore, from a biomechanical perspective, the descending aorta should be more prone to dilatation than the aortic arch.

Our study population cohort- with a relatively suitable sized sample- represents a homogenous illustrative sector of those patients carrying the pathology of AIAD with or without significant AI associated with multiple comorbidities and risk factors. Our cohort showed comparable results to other reported cohorts. **Rylski *et al.*** [10] reported on 86 survivors. **Kallenbach *et al.*** [13] reported their results on 22 patients, **Rios *et al.*** [14] reported on 87 patients, **David *et al.*** reported on more cohorts: 167 patients in one study [15] and 289 patients in another one [16]. **Rylski *et al.*** [5] reported on older patients and demonstrated even higher incidence of morbidity and mortality rates reaching up to 35.5% in age groups over 80 years. The prevalence of aortic dissection in young male patients is thought to be explained by gender-related alterations in aorta geometry, they added. However, the ratio normalises across genders when patients age beyond 70 years. Intraoperatively, in our study population, the dissection flap was seen in the ascending aorta in only 1(1.20%) patient, extending to the aortic arch in 4(4.82%) patients while in the rest 78(93.97%) patients, the flap extended to the descending aorta. However, **Kallenbach *et al.*** [13] in their smaller sample-sized cohort study reported finding the dissection flap in the ascending aorta in 3/22(13.64%) patients or in both the ascending aorta and the aortic arch in the remaining 19/22(86.36%) patients. **Rylski *et al.*** [10], of the patients included, the descending thoracic and abdominal aortas were dissected in 100% and 76% of the cases, respectively, and 155/271 (57.20%) had dissection extending at least into the aortic arch. In the aorta, we discovered many intimal tear sites. Thirteen (15.66%) patients had it in the aortic arch, and 70 (84.34%) patients had it in the ascending aorta. However, the outcome wasn't markedly different in relation to the intimal tear. In addition to AV surgery (AVR or AV repair), the ascending aorta was replaced by a synthetic graft in all patients, depending on the kind of procedure. Composite graft replacement of ascending aorta (Bental) was performed in 50 (60.24%) patients, AV re-implantation procedure (Tirone David) in 30 (36.14%), and supra-coronary conduit in 3 (3.61%).

In 59 (71.08%) patients, just replacement of the ascending aorta till the innominate artery was performed and for the rest 24(28.92%) patients, the hemiarch was replaced. That was similar to other reports. **Rios *et al.*** [14] for example reported isolated replacement of the ascending aorta to be the most frequently (78.2%) performed procedure. Again, the difference in the outcome of false lumen and dilatation of the distal descending aorta was insignificantly remarkable among different types of operations. Worthy to note that the definite decision of aortic root reconstruction was made intraoperatively after measuring the aortic annulus.

AVR was preferred in patients with Marfan syndrome, BAV, in dilated aortic annulus more than 27 mm and diseased AV leaflets. Although some surgeons prefer to use pericardial strips within the anastomosis to ensure adequate obliteration of the false lumen and

others propose inserting a short elephant trunk during aortic arch replacement to prevent tiny intimal rips at the anastomosis [17], we didn't attempt such procedures. In consistent with our evolved results, other investigators have documented an inverse association between the degree of aortic replacement and the occurrence of false lumen patency. They discovered that following arch replacement, the proximal descending thoracic aortic false lumen was less commonly patent than after ascending aorta replacement alone [3,5,7,8,10,14].

In our study, we had a mean operative time of 433.20±88.80 min., a mean CPB time of 208.40±24.60 min., a mean aortic cross clamping time of 141.60±22.80 min. and a mean TCA time of 19.88±4.50 min. Our results were similar and even a bit shorter than other authors' reports. Of whom, **Kallenbach *et al.*** [13] who reported a mean CPB time of 212±56 min. (range: 134–352 min.), a mean aortic cross clamping time of 157±24 min. (range: 114– 205 min.) and a mean TCA time of 35±18 min. (range: 11–75 min.), and **Leyh *et al.*** [18] who reported a mean CPB time of 212±20 min., a mean aortic cross clamping time of 143±18 min. and a mean TCA time of 29±9 min. However, **Rios *et al.*** [14] reported a shorter mean CPB time of 173±52 min. and a longer mean TCA time of 35±11 min. that was performed in 55.2% of their cohort.

Our results concerning the mean mechanical ventilation time (mean: 29.08±14.53 hours), mean total ICU stay time (mean: 87.68±22.19 hours) and mean total duration of hospital stay (mean: 13.28±5.09 days) were comparably like others'. **Kallenbach *et al.*** [13] reported shorter both mean mechanical ventilation time of 20±8 hours and mean total ICU stay time of 50±16 hours, but longer mean total duration of hospital stay of 21±14.4 days as well as **Graeter *et al.*** [19] who reported mean mechanical ventilation time of 24±6 hours, mean total ICU stay time of 60±15 hours and mean total duration of hospital stay of 14±6 days. However, **Rios *et al.*** [14] reported longer durations of mean mechanical ventilation time of 59.6±2.17 hours, mean total ICU stay time of 151.2±290.4 hours and mean total duration of hospital stay of 13.2±13.7 days. Although our average total duration of hospital stay was 7 to 19 days, 3(4%) patients stayed for 28 days [2(2.66%) patients owing to neurological complications and 1(1.33%) patient due to sternal dehiscence and he had undergone sternal re-wiring].

Our immediate postoperative adverse events showed comparable results resembling what other investigators reported [3,5,7,8,10,11,13,14,15,16,18]. The most frequently encountered adverse effects were renal, neurological and pericardial effusions. We had 14(18.67%) patients with neurological complications in the form of delayed recovery more than 24 hours in 7(9.33%) patients and TIAs with complete resolution in 6(8%) patients and cerebrovascular stroke with residual hemiplegia in 1(1.33%) patient. 15(20%) patients of our population had renal insult in the form of elevated serum creatinine. 5(6.66%) patients of them needed one or two sessions of dialysis and resolved completely and only 1(1.33%) patient became in need for regular dialysis sessions. 8(10.67%) patients had superficial sternotomy wound infection. Of them, 2(2.67%) patients turned to deep sternotomy wound infection and needed re-operation for sternal re-wiring while the other 6(8%) patients needed only frequent dressing and antibiotics according to culture and sensitivity. 14(18.67%) patients had mild to moderate pericardial effusion, but none was a cause of hemodynamic significance for interventional evacuation. We had to re-explore 7(9.33%) patients for hemorrhagic complications. **David *et al.*** reported rates of re-exploration for mediastinal bleeding of 7% [15], and 8.5% [16] in two different studies. **Ninomiya *et al.*** [20] in another comparative study between different techniques showed that the rate of re-exploration for bleeding was 20%. The high rates of re-exploration for bleeding in our study and the comparable ones might be due to the prolonged CPB time and its subsequent coagulopathic effects. The operative mortality -according to the recent registry- is still approximately 10–20% [21,22]. However, some authors advocate higher rates of 19-32% [23,24]. We had 8(9.64%) intraoperative mortality comparably similar to others' reports. **Rios *et al.*** [14] reported 11(17.5%) intraoperative mortality. Our operative mortality rate was 10(12.05%) which mimicked others' reports. **Ikeno *et al.*** [7] reported 37/267(13.86%). However, **Rios *et al.*** [14] reported higher rate of operative mortality of 18(20.7%).

In our study, we found most of our cohort having a persistent patent false lumen in the descending aorta with statistically insignificant different value (p=0.385) at both 6-months and 1-year follow-up. However, statistically significant different value (p<0.001) was found regarding total resolution of the false lumen in the descending aorta and the mean diameter of the descending aorta at 1-year follow-up. Again, at 1-year follow-up, we found the majority of patients [56(86.15%)] with persistent false lumen, 3(4.61%) patients with thrombosed false lumen and 6(9.23%) patients with totally resolved false lumen. Our findings agree with other authors' reports. **Zhang *et al.*** [11] reported 70% patent false lumen, **Tse *et al.*** [12] reported 91% patent false lumen and 9% thrombosed one, **Moore *et al.*** [25] reported patency of the false lumen in the descending aorta at ranges of 50-80% and **Gariboldi *et al.*** [26] reported remaining of the false lumen in 69% of their cohort.

At 6-months follow-up, there was no dilatation of the descending aorta. At one-year follow-up, we found statistically significant (p<0.001) dilatation of the descending aorta of >10 mm in 24(42.86%) and <10 mm in 32(57.14%) in those patients with persistent false lumen, and of >10 mm in 25(38.46%) and <10 mm in 37(56.92%) in the whole survivors. Moreover, we found that patients with thrombosed false lumen showed minimal retraction 0.2 mm in its diameter or

even no dilatation. We believe that the descending aorta dilatation was attributed to enlargement of the false lumen. In agreement with our findings what was reported by **Tanaka *et al.*** [3]. They reported that in 47.5% of their cohort, there was increase of the maximal diameter of the descending aorta by 10 mm or more. **Halstead *et al.*** [27] reported that the median maximum diameter of the descending aorta was 37 mm, with a 1 mm/year growth rate. They also indicated that if the descending aorta's beginning diameter is 40 mm or more, quicker growth is to be expected. **Heinemann *et al.*** [28] **& Zierer *et al.*** [29] reported aneurysmal dilatation of the descending aorta of >10 mm in 15-60% of the patients within 5 years follow-up.

Comparison of the false lumen fates between 1-year survivors above and under 40 years of age showed statistically insignificant patency of the false lumen being persistent in most patients, statistically significant (p<0.001) higher rates of both total resolution of the false lumen and >10 mm dilatation of the descending aorta in patients <40 years old, and statistically significant (p<0.001) higher rates of both thrombosed false lumen and consequent minimal retraction 0.2 mm in the descending aorta diameter or no dilatation and <10 mm dilatation of the descending aorta in patients >40 years old. Moreover, the descending aorta was dilated more than 10 mm in all 21 Marfan syndrome patients, and there was a patent false lumen. Of these, 14 patients (66.67%) were under 40 years old. We hypothesized that the greater frequency of younger patients with Marfan syndrome among the survivors in our cohort was the cause of the noticeably higher incidence of descending aortic dilatation among younger patients. Furthermore, we hypothesized that younger patients were more susceptible to the effects of hypertension on aortic dilatation because most of our patients required long-term antihypertensive drugs after surgery. Our findings supported earlier studies that found younger age groups comprised the majority of patients with aneurysmally-dilated descending aortas [3,5,7,8,10,13,14,15,16,26,27,28].

We had no late mortality over one-year follow-up and the overall survival rate was 65(78.31%) in our study that lasted for 4.92 years. **Ikeno *et al.*** [7] reported 8/267(3.5%) late mortality in the first year and overall 48/267(17.98%) death rate of their follow-up that lasted for 6.5±4.6 years (range 0.1–18.8 years). They further reported that the 5- and 10-year survival were 84.1±3.6% and 70.8±5.3% respectively while the 5- and 10-year survival reported by **Rios *et al.*** [14] in their study that took 10 years were 52.8±5.5% and 37.3±6.2% respectively. **Gariboldi *et al.*** [26] reported overall survival at 1,5 and 10 years was 96%, 80% and 65% in their follow-up study averaged 4.45 years. For patients complicated with renal failure 1(1.54%) and lower limb ischemia 2(3.08%), we found no statistical significance between renal failure and/or lower limb ischemia and the false lumen persistency.

The main risk factors for false lumen patency include uncontrolled hypertension, Marfan syndrome, young age, need for anticoagulation, retrograde type 1 dissection with major distal tears, and initial preoperative descending aortic diameter >4.5 cm [3,4,5,7,8,9,10,13,14,18,20,25,28]. By multivariable logistic regression analysis, we found that patients <50 years, Bentall operation and long-term anticoagulation were significant predictive risk factors for persistent false lumen in the descending aorta, and patients >60 years and valve-sparing aortic root reconstruction procedure were significant predictive risk factors for false lumen thrombosis. Our findings were similar to what was reported by **Gariboldi *et al.*** [26] who reported that younger aged patients <50 years, DeBakey type III retrograde dissections, the Bentall procedure, and long-term oral anticoagulation were significant risk factors for descending aorta false lumen patency, and older patients >70 years old and valve-sparing aortic root reconstruction (Tirone David or Yacoub procedures) were significant predictors of descending aorta false lumen thrombosis.

Evangelista *et al.* [30] reported that the baseline maximal diameter of the descending aorta and the proximal entry tear site and size were the significant predictors of patent descending aorta false lumen. **Suzuki *et al.*** [31] reported that younger age, greater diameter of the descending aorta, nonprescription of beta-blockers and primary entry in the ascending aorta were the significant predictors of patent descending aorta false lumen. **Santamaria *et al.*** [32] reported the dramatic importance of aortic diameter in determining the time for complete thrombosis of the false lumen, where a larger preoperative diameter of the ascending aorta is associated with chronic partial thrombosis and a larger preoperative diameter of the descending aorta is associated with a longer time for complete thrombosis.

CONCLUSION

Residual intimal tear in the distal descending aorta is thought to be the major cause of continued false lumen patency. More extensive aortic replacement and avoidance of surgical reconstruction procedures needing long-term anticoagulation could diminish the prevalence of patent false lumen and its adverse sequelae. These findings can be used as control data for determining the benefit of more extensive or new surgical approaches.

STUDY LIMITATIONS

There were several limitations to our investigation. It came from a single center and was retroactive. The follow-up period was insufficient to demonstrate descending aortic alterations over the long run. Due to the very limited number of patients who received arch replacement, we were unable to compare the outcomes of ascending aortic replacement with those of arch replacement. The degree of aortic replacement would have affected the false lumen patency rates in this

comparison. However, we think that the information we gathered from our study will be helpful in modifying the descending aortic false lumen and the descending aorta's behavior regarding its diameter and expansion following surgery that follows contemporary guidelines, which call for open anastomosis under TCA and the resection of all aortic arch tears.

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