Interactive CardioVascular and Thoracic Surgery (2017) 1–7
doi:10.1093/icvts/ivx229

ORIGINAL ARTICLE

Cite this article as: Colli A, Carrozzini M, Francescato A, Galuppo M, Comisso M, Toto F *et al.* Acute DeBakey Type I aortic dissection without intimal tear in the arch: is total arch replacement the right choice? Interact CardioVasc Thorac Surg 2017; doi:10.1093/icvts/ivx229.

VASCULAR

Acute DeBakey Type I aortic dissection without intimal tear in the arch: is total arch replacement the right choice?

Andrea Colli[a,*,†], Massimiliano Carrozzini[a,†], Annalisa Francescato[a], Marco Galuppo[a], Marina Comisso[a], Francesca Toto[a], Dario Gregori[b] and Gino Gerosa[a]

[a] Cardiac Surgery Unit, Department of Cardiac, Thoracic and Vascular Sciences, University of Padova, Padova, Italy
[b] Biostatistics, Epidemiology and Public Health Unit, Department of Cardiac, Thoracic and Vascular Sciences, University of Padova, Padova, Italy

* Corresponding author. Cardiac Surgery Unit, Department of Cardiac, Thoracic and Vascular Sciences, University of Padova, U.O.C. Cardiochirurgia, Policlinico Universitario, Via Giustiniani 2, 35128 Padova, Italy. Tel: +39-049-8212410; fax: +39-049-8212409; e-mail: andrea.colli@unipd.it (A. Colli).

Received 3 February 2017; received in revised form 4 June 2017; accepted 11 June 2017

Abstract

OBJECTIVES: Surgical management of acute DeBakey Type I aortic dissection without intimal tear in the aortic arch is controversial. This study compared short- and long-term outcomes of total arch replacement (TAR) versus limited ascending aorta/hemiarch replacement (no-TAR) in a consecutive series of patients.

METHODS: Between January 1998 and December 2015, 220 consecutive patients were operated for DeBakey Type I acute aortic dissection; 135 cases did not exhibit an intimal entry tear in the aortic arch and were subsequently selected to comprise the primary study cohort. A secondary subgroup analysis was made within these 135 cases, which comprised patients who received antegrade cerebral perfusion as the neuroprotective strategy of choice ($n = 45$).

RESULTS: Mean follow-up period was 5 ± 4 years. Among the patients selected, 21 (16%) underwent TAR. Thirty-day mortality was higher in the TAR group (38% vs 21%, $P = 0.04$). Postoperative complication rates were similar between the groups (61% vs 73%, $P = 0.31$). Long-term mortality and late aortic reintervention rates were also similar (7% vs 30%, $P = 0.36$ and 27% vs 14%, $P = 0.32$, respectively). From the subgroup of patients with antegrade cerebral perfusion, 14 (31%) underwent TAR and 31 (69%) had no-TAR. Mean follow-up-time was 3 ± 2 years. Thirty-day mortality was higher in the TAR group (50% vs 16%, $P < 0.01$), postoperative complications, long-term mortality and late aortic reintervention rates were similar (64% vs 69%, $P = 0.73$; 0% vs 19%, $P = 0.22$; 29% vs 8%, $P = 0.17$, respectively).

CONCLUSIONS: TAR was associated with higher 30-day mortality compared with the less extensive hemiarch replacement. In the long term, TAR showed a trend of improved survival and higher reintervention rate.

Keywords: Aortic dissection • Intimal tear • Aortic arch • Total arch replacement

INTRODUCTION

DeBakey Type I acute aortic dissection remains a major surgical challenge, associated with high rates of mortality and morbidity [1–5]. Optimal surgical management is still under debate, and one of the most discussed issues is the treatment of the dissected aortic arch [6]. A general agreement exists in applying total arch replacement (TAR) in patients presenting with an intimal tear in the aortic arch; however, this circumstance is detected only in the minority of cases (10–30%) [7, 8]. In the absence of a visualized intimal tear in the arch, many authors advocate a conservative approach limited to the ascending aorta or hemiarch replacement to reduce the risk inherent to more complex surgical procedures [9, 10]. The contrary stipulation of an incremental benefit of extending the aortic replacement to the arch is based on the purported advantage of obliterating the false lumen more effectively, thus preventing late aortic dilation and the associated risks of reintervention or sudden rupture [11–14].

The aim of this study was to compare early and long-term outcomes of TAR versus ascending aorta/hemiarch replacement (no-TAR) in patients with DeBakey Type I acute aortic dissection without intimal tear in the arch.

MATERIALS AND METHODS

Patients and study design

Between January 1998 and December 2015, 220 patients were treated for an acute aortic dissection at our Institute; of these, 149 patients presented with a DeBakey Type I dissection without intimal tear in the arch and were included in the study.

†The first two authors contributed equally to this study.

To provide an unbiased analysis, 14 patients managed with deep hypothermic circulatory arrest, clamped distal anastomosis or unknown neuroprotection strategy were excluded, leading to our study population of 135 patients.

Follow-up data were derived from outpatient clinic records, telephone contact and municipal civil registries and were complete in 126 (93%) patients.

Study approval was granted by the Ethics Committee of the University of Padova; individual patient consent was waived.

Primary end-points were 30-day mortality and all-cause late mortality; secondary end-points were intensive care unit stay and hospital stay and rate of intraoperative death, postoperative procedure-related complication and late aortic reintervention.

In our series, both antegrade cerebral perfusion (ACP) and retrograde cerebral perfusion (RCP) were adopted as neuroprotection strategies. ACP is well established as the gold standard neuroprotection strategy in aortic arch surgery; therefore, to eliminate any confounding effect on outcomes, we decided to perform a secondary separate analysis on the patients who received ACP ($n = 45$).

Definitions

Aortic dissection definition was made in accordance with current international guidelines [15].

The presence of an intimal tear in the aortic arch was assessed by operative reports and by reviewing preoperatory computed tomography scan if not reported in surgical operative reports.

Obesity was body mass index >30 kg/m^2. Hypertension was considered as history of systolic pressure >140 mmHg or diastolic pressure >90 mmHg. Diabetes was defined as history of diabetes mellitus, regardless of the need for antidiabetic agents or duration of the disease. Dyslipidaemia included any lipid disorder. Chronic lung disease, peripheral vascular disease and renal impairment were defined according to 'EuroSCORE II'. The neurological status on admission was considered undefined when the patient was already under sedation. Cardiogenic shock was identified as persistent hypotension (systolic pressure <80 mmHg) in the presence of adequate elevated filling pressures. Postoperative complications were classified according to the latest consensus statement from the International Aortic Arch Surgery Study Group [16].

Operative technique

During the study period, the interventions were performed by 10 experienced surgeons.

Intraoperative transoesophageal echocardiography was used for the confirmation of diagnosis and evaluation of the aortic valve. Invasive haemodynamic monitoring was achieved with arterial lines in both right and left radial arteries, and the use of cerebral and somatic regional oxygen saturation monitoring (INVOS^{TM}). All patients underwent median sternotomy. Cannulation site for cardiopulmonary bypass (CPB) depended on dissection extent, the need for emergent cannulation, planned neuroprotection strategy and flow requirements. Arterial cannulation was carried out through direct cannulation or grafted perfusion of either the femoral or the right subclavian artery. Venous return was achieved via right atrial or bicaval cannulation; few cases required initial femoral cannulation for emergent CPB institution. The left ventricle was vented through the right superior pulmonary vein or the main pulmonary artery trunk. Temperature was continuously measured in the nasopharynx, oesophagus and rectum. Desired target temperature increased during the study period to achieve moderate hypothermia between 24°C and 28°C. During cooling, the ascending aorta was cross-clamped, the aortotomy performed and antegrade cold blood cardioplegia was administered directly into the coronary ostia. The aortic root, aortic valve and coronary arteries were then inspected and the proximal anastomosis was performed, as needed. Once the target temperature was reached, a period of circulatory arrest followed, allowing for cross-clamp removal and inspection of the aortic arch, followed by an open-end distal anastomosis. RCP or ACP, according to Kazui *et al.*'s [17] protocol, was adopted as neuroprotection strategy: RCP was performed with a perfusion flow of 150–450 ml/min via the superior vena cava, maintaining a central venous pressure of 20–30 mmHg. ACP was established through a selective cannulation of the supra-aortic branches. In case the subclavian artery was used for CPB, the brachiocephalic trunk was tightened, and cerebral perfusion was performed through the right subclavian artery and selective cannulation of the left common carotid artery and left subclavian artery. Perfusion flow for ACP was 10–15 ml/kg/min, maintaining a right radial artery pressure of 40–70 mmHg. In patients receiving TAR, after cross-clamp removal, the aortic arch was completely excised up to the origin of the supra-aortic vessels and replaced using a tri- or quadrifurcated graft; the distal anastomosis was performed first. Following reinstitution of CPB through the graft, the supra-aortic branches were anastomosed separately. Rewarming was achieved at 1°C/3 min, not exceeding a 10°C gradient between blood temperature and nasopharyngeal or rectal temperature.

In cases of malperfusion after cross-clamping the ascending aorta, and as soon as the desired target temperature was reached, the cross-clamp was removed and the distal repair was performed first, during circulatory arrest with RCP or ACP, followed by reinstitution of CPB through the graft and finally the completion of the proximal anastomosis.

Detailed operative data are presented in Table 3.

Statistical analysis

Continuous variables were expressed as average; standard deviation was used as measure of variability. Comparison among groups was based on the Wilcoxon or χ^2 test, depending on the characteristics of the variables involved. Cumulative survival curves were generated with the Kaplan–Meier method and compared using the log-rank test. Mean survival time was estimated using Tsiatis estimate for the baseline hazard and a Cox proportional hazard model. The statistical significance was set at P-value <0.05. The R-System statistical package and Harrell's regression modelling strategies libraries were used for analysis.

RESULTS

Overall population

From our total study cohort ($n = 135$), 114 (84%) patients were no-TAR and 21 (16%) patients were TAR. Baseline patients' characteristics, risk factors (Table 1) and clinical presentation (Table 2) were comparable between the 2 groups.

Table 1: Patient characteristics and risk factors

Variables	Overall patients ($n = 135$)			ACP patients ($n = 45$)		
	No-TAR ($n = 114$)	TAR ($n = 21$)	*P*-value	No-TAR ($n = 31$)	TAR ($n = 14$)	*P*-value
Age (years)	63 ± 12	63 ± 13	0.89	64 ± 11	60 ± 11	0.29
Male	49 (43)	9 (43)	0.99	9 (29)	6 (43)	0.36
BSA (m^2)	1.7 ± 0.6	1.5 ± 0.8	0.74	1.8 ± 0.4	1.9 ± 0.2	0.43
BMI (kg/m^2)	26 ± 4	27 ± 5	0.56	26 ± 5	27 ± 4	0.84
Obesity	20 (15)	6 (29)	0.24	8 (27)	4 (28)	0.84
Systemic hypertension	61 (69)	15 (83)	0.21	18 (64)	8 (73)	0.62
Diabetes mellitus	6 (7)	0 (0)	0.27	1 (4)	0 (0)	0.55
Dyslipidaemia	10 (13)	3 (25)	0.26	4 (15)	2 (20)	0.71
Smoking	13 (15)	1 (6)	0.49	6 (21)	1 (10)	0.44
Aortic diseases familiarity	1 (1)	1 (6)	0.19	0 (0)	1 (10)	0.09
Bicuspid aortic valve	4 (5)	1 (6)	0.87	1 (4)	1 (9)	0.51
Marfan syndrome	3 (3)	0 (0)	0.44	0 (0)	0 (0)	1
Chronic lung disease	3 (3)	0 (0)	0.44	1 (0)	0 (0)	0.55
Peripheral vascular disease	6 (8)	2 (17)	0.31	2 (7)	2 (20)	0.27
Renal impairment	5 (6)	0 (0)	0.31	1 (4)	0 (0)	0.55
Prior CVA	4 (5)	0 (0)	0.72	0 (0)	0 (0)	1
CAD	4 (4)	1 (6)	0.52	1 (4)	0 (0)	0.55
Prior AMI	3 (3)	1 (6)	0.66	0 (0)	1 (10)	0.09
Prior cardiac surgery	3 (3)	2 (12)	0.14	0 (0)	1 (10)	0.09
Prior aortic surgery	7 (8)	3 (18)	0.23	4 (15)	2 (20)	0.71

Values are represented as *n* (%) or mean ± SD. Percentages are calculated as the number of events on total number of patients with available data for the variable under examination for each subgroup.

ACP: antegrade cerebral perfusion; AMI: acute myocardial infarction; BMI: body mass index; BSA: body surface area; CVA: cerebrovascular accident; CAD: coronary artery disease; SD: standard deviation; TAR: total arch replacement.

Table 2: Clinical presentation

Variables	Overall patients ($n = 135$)			ACP patients ($n = 45$)		
	No-TAR ($n = 114$)	TAR ($n = 21$)	*P*-value	No-TAR ($n = 31$)	TAR ($n = 14$)	*P*-value
Chest pain	58 (64)	11 (61)	0.79	19 (66)	9 (75)	0.55
Back pain	18 (20)	3 (17)	0.74	5 (17)	2 (17)	0.97
Migrating pain	3 (3)	1 (6)	0.65	1 (3)	1 (8)	0.51
Abdominal pain	12 (13)	3 (17)	0.71	4 (14)	3 (25)	0.39
Any pulse deficit	5 (6)	2 (11)	0.38	1 (3)	2 (17)	0.14
Limb ischaemia	5 (6)	0 (0)	0.31	2 (7)	0 (0)	0.35
Visceral malperfusion	4 (4)	0 (0)	0.36	0 (0)	0 (0)	1
Syncope	10 (11)	3 (17)	0.52	4 (14)	1 (8)	0.63
Neurological symptomatology	31 (34)	3 (17)	0.12	7 (23)	2 (17)	0.63
Any neurological deficit	17 (18)	3 (17)	0.83	6 (21)	2 (17)	0.77
Coma	5 (5)	0 (0)	0.31	0 (0)	0 (0)	1
Undefined	9 (10)	0 (0)	0.17	1 (3)	0 (0)	0.52
Cardiac symptomatology	17 (18)	2 (11)	0.40	3 (10)	1 (8)	0.87
Ongoing AMI	2 (2)	0 (0)	0.52	1 (3)	0 (0)	0.51
Cardiogenic shock	8 (9)	1 (6)	0.66	1 (3)	1 (8)	0.49
Cardiac tamponade	7 (8)	1 (6)	0.76	1 (3)	0 (0)	0.52
Dissection involving supra-aortic branches	58 (42)	14 (67)	0.45	14 (45)	11 (79)	0.26
Dissection involving coronary arteries	10 (9)	1 (5)	0.06	4 (13)	1 (7)	0.26

Values are represented as *n* (%) or mean ± SD. Percentages are calculated as the number of events on total number of patients with available data for the variable under examination for each subgroup.

ACP: antegrade cerebral perfusion; AMI: acute myocardial infarction; SD: standard deviation; TAR: total arch replacement.

In TAR patients, both CPB time and ACP time were significantly longer compared to the no-TAR group ($P = 0.01$ and $P < 0.01$, respectively), whereas other procedural data were not statistically different (Table 3).

Three (14%) patients died intraoperatively in the TAR group and 6 (5%) patients in the no-TAR group ($P = 0.13$). Causes of intraoperative death were cardiac failure (TAR, $n = 1$; No-TAR, $n = 4$), bleeding (TAR, $n = 1$; no-TAR, $n = 1$) and descending aorta rupture (TAR, $n = 1$; no-TAR, $n = 1$). Among the remaining patients, mean intensive care unit stay (TAR: 6 ± 8 days vs no-TAR: 6 ± 6 days; $P = 0.72$) and hospital stay (TAR: 14 ± 13 days vs no-TAR: 15 ± 10 days; $P = 0.74$) were comparable between the

Table 3: Procedural data

Variables	Overall patients (n = 135)			ACP patients (n = 45)		
	No-TAR (n = 114)	TAR (n = 21)	P-value	No-TAR (n = 31)	TAR (n = 14)	P-value
Rectal temperature (°C)	20 ± 4	22 ± 3	0.16	24 ± 3	22 ± 3	0.05
CPB time (min)	234 ± 84	285 ± 87	**0.01**	263 ± 84	323 ± 77	**0.02**
Cross-clamp time (min)	131 ± 54	157 ± 65	0.06	155 ± 55	186 ± 58	0.06
RCP time (min)	29 ± 14	26 ± 17	0.66			
ACP time (min)	36 ± 23	88 ± 52	**<0.01**	36 ± 23	88 ± 52	**<0.01**
Arterial cannulation			0.37			0.56
Subclavian artery	24 (20)	8 (38)		15 (48)	5 (36)	
Femoral artery	73 (65)	11 (52)		10 (32)	7 (50)	
Other	17 (15)	2 (10)		6 (20)	2 (14)	
Venous cannulation			0.08			0.79
Atriocaval	21 (18)	8 (38)		14 (45)	6 (43)	
Bicaval	75 (66)	7 (33)		8 (26)	5 (36)	
Other	18 (16)	6 (29)		9 (29)	3 (21)	
AVR	39 (34)	6 (29)	0.61	14 (45)	5 (36)	0.55
Valve-sparing procedure	3 (3)	0 (0)	0.45	2 (6)	0 (0)	0.33
Aortic root replacement	26 (23)	6 (29)	0.57	7 (23)	5 (36)	0.36
CABG	7 (6)	2 (10)	0.57	2 (6)	1 (7)	0.93

Values are represented as n (%) or mean ± SD. Percentages are calculated as the number of events on total number of patients with available data for the variable under examination for each subgroup.

Boldface significance is 0.05.

ACP: antegrade cerebral perfusion; AVR: aortic valve replacement; CABG: coronary artery bypass grafting; CPB: cardiopulmonary bypass; RCP: retrograde cerebral perfusion; SD: standard deviation; TAR: total arch replacement.

Table 4: Postoperative complications

Variables	Overall patients (n = 135)			ACP patients (n = 45)		
	No-TAR (n = 114)	TAR (n = 21)	P-value	No-TAR (n = 31)	TAR (n = 14)	P-value
Global neurological deficit	21 (20)	5 (28)	0.47	5 (18)	3 (27)	0.51
Focal neurological deficit	13 (13)	2 (11)	0.87	2 (7)	1 (9)	0.84
Spinal neurological deficit	3 (3)	1 (6)	0.56	1 (4)	0 (0)	0.53
Myocardial ischaemia	3 (3)	0 (0)	0.47	2 (7)	0 (0)	0.36
LCO syndrome	4 (4)	1 (6)	0.74	1 (4)	1 (9)	0.48
Arrhythmia	26 (25)	4 (22)	0.81	10 (36)	3 (27)	0.61
Pericardial effusion	5 (5)	1 (6)	0.89	2 (7)	1 (9)	0.84
Respiratory parenchymal complication	20 (19)	4 (22)	0.77	5 (18)	2 (18)	0.98
Respiratory pleural complication	9 (9)	2 (11)	0.74	4 (14)	1 (9)	0.66
Renal dysfunction	18 (17)	3 (17)	0.95	3 (11)	2 (18)	0.53
Gut or hepatobiliary complication	6 (6)	1 (6)	0.97	2 (7)	0 (0)	0.36
Postoperative bleeding	21 (20)	4 (22)	0.84	5 (18)	2 (18)	0.98
Wound complication	2 (2)	1 (6)	0.36	0 (0)	1 (9)	0.11
Infection (other than wound)	9 (9)	2 (11)	0.74	3 (11)	1 (9)	0.88

Values are represented as n (%) or mean ± SD. Percentages are calculated as the number of events on total number of patients with available data for the variable under examination for each subgroup.

ACP: antegrade cerebral perfusion; LCO: low cardiac output; SD: standard deviation; TAR: total arch replacement.

2 groups. The rate of postoperative complications (61% vs 73%; P = 0.31) was similar (Table 4).

Thirty-day mortality was significantly higher in TAR patients (38% vs 21%; P = 0.04). Causes of death (excluding intraoperative deaths) were cardiac failure (TAR, n = 2; no-TAR n = 7), fatal cerebral event (TAR, n = 2; no-TAR, n = 4), septicaemia (TAR, n = 1; no-TAR, n = 3), bleeding (TAR, n = 0; no-TAR, n = 2), descending aorta rupture (TAR, n = 0; no-TAR, n = 1) and bowel ischaemia (TAR, n = 0; no-TAR, n = 1).

Mean follow-up time was 5 years (range 0.1–15 years) in the no-TAR group and 4 years (range 0.7–9 years) in the TAR group (P = 0.09). Late all-cause mortality was higher in the no-TAR group (7% vs 30%; P = 0.36, Figure 1), though not significantly. There was no significant difference in the rate of late aortic reintervention between groups (27% vs 14%; P = 0.32). The types of reintervention were as follows: thoracic endovascular aortic repair (TAR, n = 2; no-TAR, n = 1), aortic arch replacement (TAR, n = 0; no-TAR, n = 4), abdominal aorta replacement (TAR, n = 1; no-TAR, n = 4), aortic arch and abdominal aorta replacement (TAR, n = 0; no-TAR, n = 1), carotid–subclavian artery bypass and descending aorta replacement (TAR, n = 0; no-TAR, n = 2). Of the 12 patients, 5 (42%) patients in the no-TAR group died

Table 5: Early and late outcomes

Variables	Overall patients ($n = 135$)			ACP patients ($n = 45$)		
	No-TAR ($n = 114$)	TAR ($n = 21$)	*P*-value	No-TAR ($n = 31$)	TAR ($n = 14$)	*P*-value
ICU stay (days)	6 ± 6	6 ± 8	0.72	5 ± 7	4 ± 5	0.69
Hospital stay (days)	15 ± 10	14 ± 13	0.74	15 ± 13	9 ± 9	0.14
Postoperative complications	78 (73)	11 (61)	0.32	20 (69)	7 (64)	0.73
Intraoperative deaths	6 (5)	3 (14)	0.13	3 (10)	4 (29)	0.11
Thirty-day deaths	24 (21)	8 (38)	**0.04**	5 (16)	7 (50)	**<0.01**
Late deaths	27 (30)	1 (7)	0.36	5 (19)	0 (0)	0.22
Late aortic reintervention	12 (14)	3 (27)	0.32	2 (8)	2 (29)	0.17

Values are represented as *n* (%) or mean ± SD. Percentages are calculated as the number of events on total number of patients with available data for the variable under examination for each subgroup.
Boldface significance is 0.05.
ACP: antegrade cerebral perfusion; ICU: intensive care unit; SD: standard deviation; TAR: total arch replacement.

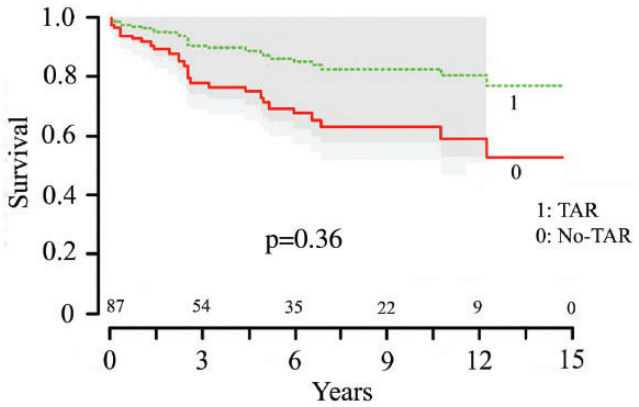

Figure 1: Kaplan–Meier survival curve of the overall patient analysis. TAR: total arch replacement; no-TAR: limited ascending aorta/hemiarch replacement.

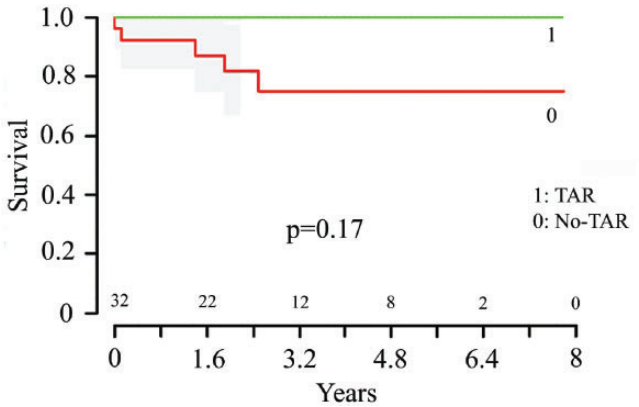

Figure 2: Kaplan–Meier survival curve of the antegrade cerebral perfusion patient analysis. TAR: total arch replacement; no-TAR: limited ascending aorta/hemiarch replacement.

during reintervention; there was no reoperative death in the TAR group.

Early and late outcomes are summarized in Table 5.

Antegrade cerebral perfusion population analysis

Forty-five patients received ACP; among those, 31 (69%) patients were no-TAR and 14 (31%) patients were TAR.

Preoperative data remained comparable between the 2 groups (Tables 1 and 2). CPB time (TAR: 323 ± 77 vs no-TAR: 263 ± 84; $P = 0.02$) and ACP time (TAR: 88 ± 52 vs no-TAR: 36 ± 23; $P < 0.01$) were significantly higher in the TAR group (Table 3).

Early and long-term outcomes of TAR versus no-TAR were identified as follows (Tables 4 and 5): intraoperative mortality was 29% in TAR and 10% in no-TAR ($P = 0.11$); 30-day mortality remained significantly higher in the TAR patients (50% vs 16%; $P < 0.01$) with a comparable rate of postoperative complications (64% vs 69%; $P = 0.73$). Mean follow-up time was 3 years (range 0.1–8 years) in the no-TAR group and 3 years (range 0.7–4 years) in the TAR group ($P = 0.54$). Late all-cause mortality (0% vs 19%; $P = 0.22$) and rate of aortic reintervention (29% vs 8%; $P = 0.17$) were not statistically different between groups (Figure 2).

DISCUSSION

One of the most controversial issues in aortic surgery is the management of the aortic arch in the setting of acute DeBakey Type I dissections. In fact, increased understanding of the pathophysiology and improved surgical techniques and perioperative care allowed for a safer approach to aortic arch surgery; however, it remains unclear whether a more complex, extensive surgery translates to better patient outcomes [6]. Especially in the presence of arch dissection but without an intimal tear in it, there is no consensus among surgeons: some prefer TAR based on the belief of achieving a more radical resolution of the aortic disease, reducing the rate of late false lumen patency; others tend to limit the reconstruction to the ascending aorta, willing to minimize the operative risk [9–14]. In our study, we focused on this specific scenario and reviewed our experience aiming to compare these 2 surgical strategies. All included patients presented an acute Type I dissection without a demonstrable intimal tear in the aortic arch with similar patient characteristics, risk factors and clinical presentation among TAR and no-TAR groups. To avoid additional confounding related to operative technique, we excluded patients managed with deep hypothermic circulatory arrest, clamped distal anastomosis or those

with undocumented neuroprotection strategy. Finally, accounting for a potential bias due to the cerebral protection used, we performed a subanalysis of ACP patients only.

According to our data, TAR surgery showed a significantly higher early mortality both in the overall study and in the ACP patient subanalysis. Reports in the literature are controversial and often biased by selection criteria and patient heterogeneity between groups [6]. Some authors showed that aortic arch surgery could be performed without increased early mortality and morbidity [10, 18–22], whereas others reported higher rates of early deaths after TAR surgery [23, 24]. The small number of events enabled us to statistically compare different causes of death; moreover, rates of postoperative complications resulted comparable among groups, thus we could not relate the difference in mortality to a specific complication. Nevertheless, TAR patients received a significantly longer CPB and ACP time, both well-known risk factors for early death [4, 22, 25, 26], possibly explaining our findings.

In the long term, overall survival was similar for both groups with a trend towards better outcomes in patients who received TAR. With the limits of our sample size, these results are consistent with the present literature [6] and can be explained considering the unfavourable impact of false lumen patency on late outcomes. It is well established that false lumen patency represents a risk factor for aneurysmal degeneration and late death [14, 27], being associated with up to 20% reduced survival [12, 28]. As recent reports showed, a patent false lumen in the arch and proximal descending aorta can be found in 65% and 75%, respectively, of patients managed with no-TAR [19, 21, 29]; similarly, TAR surgery was demonstrated to allow false lumen thrombosis in up to 86% of cases [23]. Thus, our observed results in late survival in the TAR cohort is probably mainly due to a reduction in the false lumen patency, with a decreased risk of fatal events related to the residual dissection.

In our experience, rates of late reintervention on the remaining portion of the dissected aorta, including open and endovascular surgery, were not statistically different between the 2 groups, though more frequent in TAR patients. Of note, 42% of patients who underwent reintervention in the no-TAR group died versus no deaths in the TAR group. These results are probably due to the need for more extensive and technically demanding procedures when approaching the aorta in patients who previously underwent a proximal repair, leading to a higher reoperative risk in these patients. For the same reason, TAR surgery in our series represented a more favourable substrate for late aortic procedures.

To better manage the descending aorta following Type I aortic dissection, several authors are advocating even more aggressive primary operations, by adding to TAR a routine use of classic or frozen elephant trunk [11, 13, 19, 29, 30]. These techniques are more effective in obliterating the false lumen and facilitate further late endovascular interventions.

Limitations

We presented the results of 2 different surgical strategies, with the potential confounding effect of the individual surgeon preference in choosing one or the other. Only an analysis of randomized patients could overcome this bias, but, in the real world, it is impossible to design such a study in the setting of emergent operations.

The main limitation of our study was the small number of TAR patients reported, with an imbalance between the 2 groups considered that could have influenced the analysis. Our findings need to be confirmed in a larger patient population, ideally from a multicentre study.

As postoperative computed tomography scan was not available in all cases, we were not able to further analyse the impact of repair extension on false lumen patency and long-term outcomes.

CONCLUSIONS

Our analysis showed that TAR versus no-TAR was associated with higher early mortality. In the long term, TAR presented a tendency towards an improved survival and seemed to provide a more favourable substrate for late aortic reinterventions. On this basis, in the setting of an acute Type I aortic dissection without intimal tear in the arch, both no-TAR and TAR strategies can be pursued and recommended. The first will be mainly aimed to reach the primary goal of this emergency: to save the patient life. The latter will be justified by the intention to treat more radically the aortic disease and to better manage the remaining dissected descending aorta.

ACKNOWLEDGEMENTS

Pandis Dimosthenis, Department of Cardiovascular Surgery, Mount Sinai Medical Center, New York, USA.

Conflict of interest: none declared.

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