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Thoracic Endovascular Aortic Repair for Type B Acute Aortic Dissection Complicated by Descending Thoracic Aneurysm

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WHAT THIS PAPER ADDS

This paper offers a specific analysis of thoracic aortic endovascular repair (TEVAR) for type B acute aortic dissection complicated by descending thoracic aneurysm present on admission after the clinical onset of the dissection. This condition has not been specifically analysed in previous cohorts.

Objectives: To analyse the results and review the literature about thoracic aortic endovascular repair (TEVAR) for type B acute aortic dissection (TBAAD) complicated by descending thoracic aortic aneurysm (DTA) in the hyperacute or acute phases.

Methods: This was a multicentre, observational descriptive study. Inclusion criteria were TBAAD with a DTA of $\geq$50 mm, TBAAD on an already known aneurysmal descending thoracic aorta, and TBAAD presenting with an enlarged aorta with a total diameter $<$50 mm, but with $>$50% diameter increase compared with a previous computed tomography angiography (CTA) showing a non-dissected aorta with normal sizing. Primary endpoints were early and long-term survival, freedom from TEVAR and aortic related mortality (ARM), and freedom from re-intervention.

Results: Twenty-two patients were included in the analysis. The mean aortic diameter was 66 $\pm$ 26 mm (range 42–130; IQR 51–64). The in hospital TEVAR related mortality was 14% ($n = 3$). The mean radiological follow-up was 56 $\pm$ 45 months (range 6–149; IQR 12–82), and the follow-up index 0.97 $\pm$ 0.1. All surviving patients were available for follow-up. During follow-up the cumulative mortality was 26% ($n = 5$) and TEVAR related mortality was 5% (n = 1). Overall the estimate of survival was 82% (95%CI: 61.5–93) at 1 year, and 64% at 5 years. Ongoing primary clinical success was 79% (re-intervention $n = 4$). Freedom from aortic related mortality was 86% (95%CI: 66–95) at 1 and 5 year, while freedom from re-intervention was 95% (95%CI: 75.5–95) at 1 year, and 77% (95% CI: 50–92) at 5 years.

Conclusions: In our experience, DTA is a frequent complication from the very beginning of the clinical onset of TBAAD. In this high-risk cohort, TEVAR showed satisfactory results, better than those predicted by the risk score for open repair, with favourable stability of the aortic diameter and no aortic related adverse events during follow-up.

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INTRODUCTION

Complicated type B acute aortic dissection (TBAAD) currently identifies cases with persistent symptoms despite the best medical treatment, impending or frank rupture, or malperfusion.[1] Specific mention of TBAAD complicated by descending thoracic aneurysm (DTA) has been made rarely in acute onset of the dissection, since they are more frequently seen in the chronic phase as a degeneration of an uncomplicated TBAAD.[2] In such an acute setting, thoracic aortic endovascular repair (TEVAR) has been suggested to bring potential advantages over open repair (OR), while concerns remain in the long-term period because of aortic-related events such as rupture or re-interventions of the untreated residual dissection.[3,4] The most recent published experiences analysed mainly chronic dissection related aneurysmal degeneration of the descending thoracic aorta.[5–17] The aim of this paper was to analyse our

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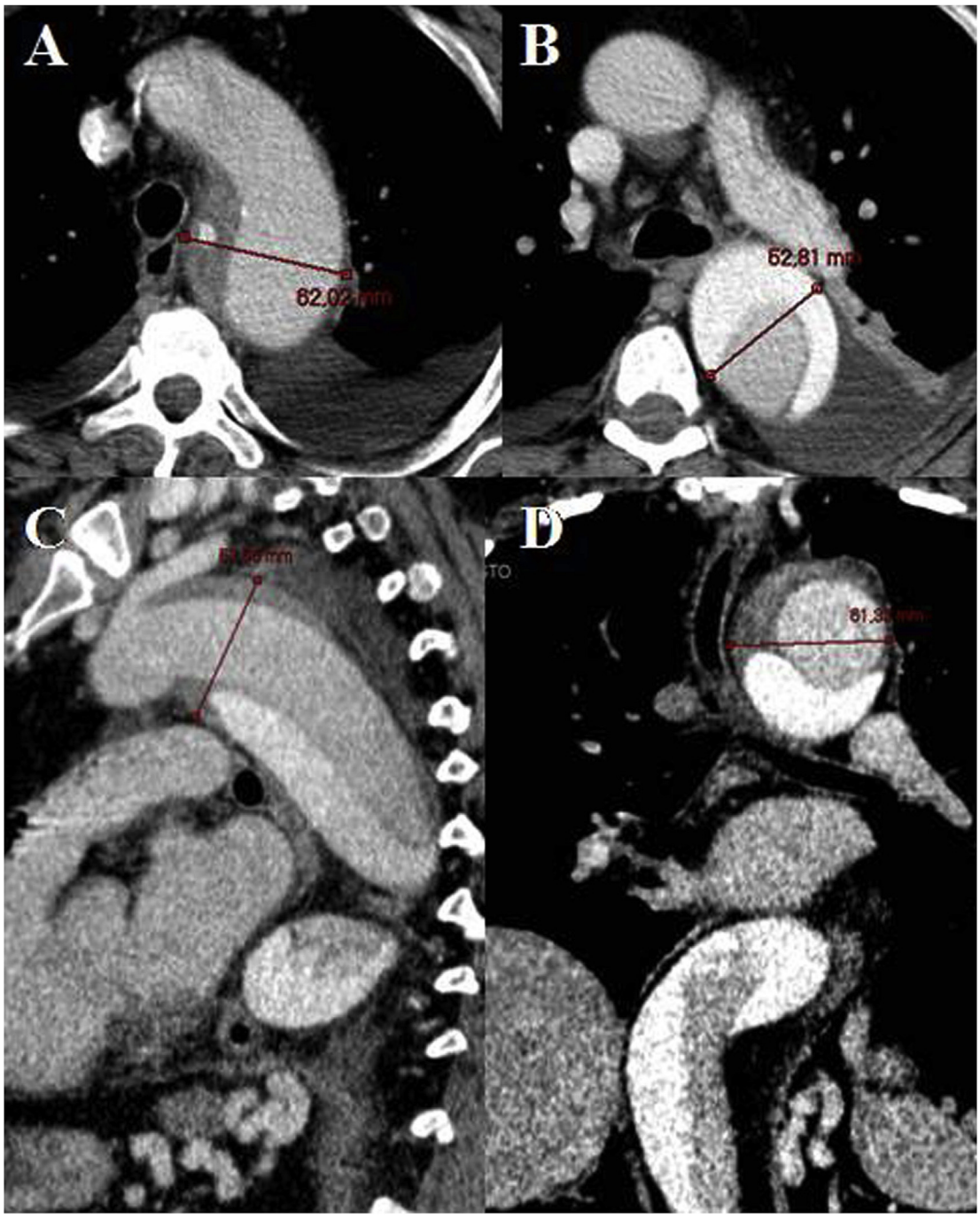

Figure 1. Acute type B aortic dissection complicated by descending aneurysm. Aneurysm involved the entire descending aorta (A—C), whereas the dissecting flap extended downstream to the abdominal aorta (D).

results of a consecutive cohort of patients treated by TEVAR for TBAAD complicated by the presence of DTA in the hyperacute or acute phase.

MATERIALS AND METHODS

Patient cohort

Since November 2000, all TBAADs were identified from a computerized database registry, which, at each centre, remained consistent over the study period. Information about demographics, comorbidities, medical and surgical history, operative details and post-operative events during the hospital stay, and follow-up were all registered. This was a multicentre, observational descriptive study with retrospective analysis of consecutive patients treated by TEVAR for TBAAD complicated by DTA in the hyperacute or acute phases. This experience includes patients observed from September 2001 to December 2014; for the final analysis, the end of the study was December 31, 2014.

Inclusion criteria were as follows:

- TBAAD with DTA of $\geq$50 mm (Fig. 1A—D)
- TBAAD upon an already known aneurysmal descending thoracic aorta (Fig. 2A, B)

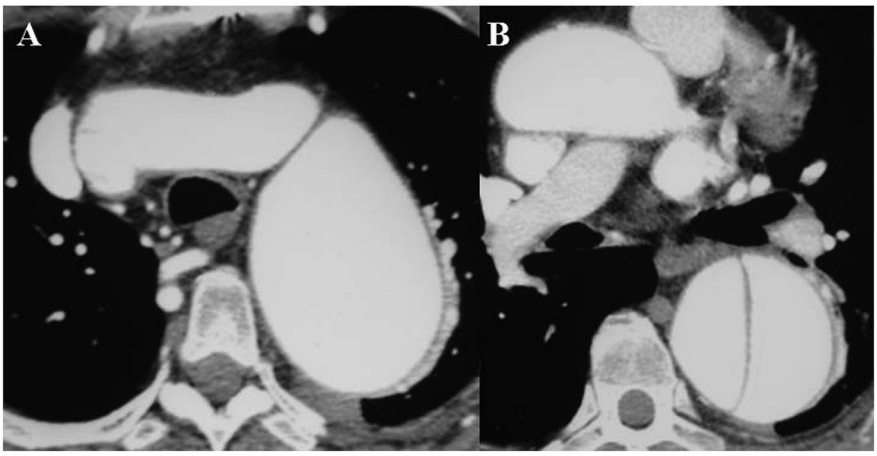

Figure 2. Acute dissection on an already aneurysmal descending thoracic aorta: the aneurysm involved the entire descending aorta starting just beyond the origin of the left subclavian artery (A), and the dissecting flap developed in the middle third of the descending thoracic aorta (B).

- TBAAD presenting with an enlarged aorta with a total diameter <50 mm, which, however, had >50% diameter increase compared with a previous computed tomography angiogram (CTA) performed before the dissection occurred and showing a normal sized aorta (Fig. 3A, B).

Exclusion criteria were as follows:

- residual type B aneurysmal degeneration after repair of type A aortic dissection
- thoraco-abdominal aneurysm caused by dissection
- dissection variants such as intramural haematoma and penetrating aortic ulcer
- acute dissection caused by blunt traumatic injury.

Informed consent was signed by each patient; retrospective analysis of the anonymised data did not require approval of the institutional review board. Each case was collegially reviewed by the coordinators (GP, PO, ST), and the cases were merged into one multicentre database for the retrospective analysis.

Operative details

All TBAADs included in this cohort were diagnosed in the hyperacute or acute phases by spiral or computed tomography angiography, and immediately managed by best medical treatment. TEVAR was performed in the operating theatre, with general anaesthesia for all patients. Usually, stent graft (SG) oversizing was 10% at most, according to the diameter of the normal aorta in the proximal landing zone, while at the planned distal landing zone (LZ) the sizing was performed considering the overall aortic diameter which included both the true (TL) and false lumen (FL). The aim of the TEVAR was firstly to seal the proximal aortic entry tear to abolish antegrade flow into the FL aneurysm, and secondly to cover the entire descending thoracic aorta to seal off the DTA. When ≥2 SGs were planned to be implanted, the sequence of SG deployment was always

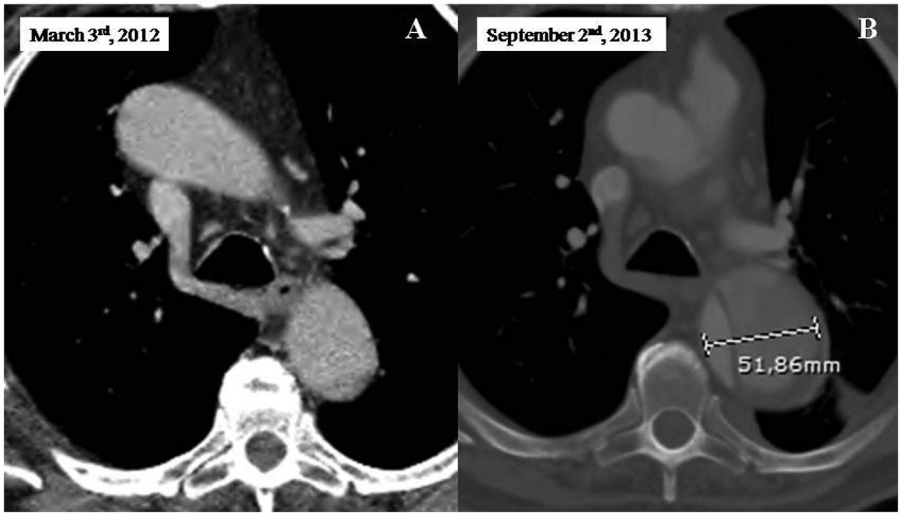

Figure 3. Acute type B aortic dissection presenting with a total diameter <55 mm (B) but with >50% diameter increase compared with a previous computed tomography angiography (CTA) showing a normal aortic sizing (A).

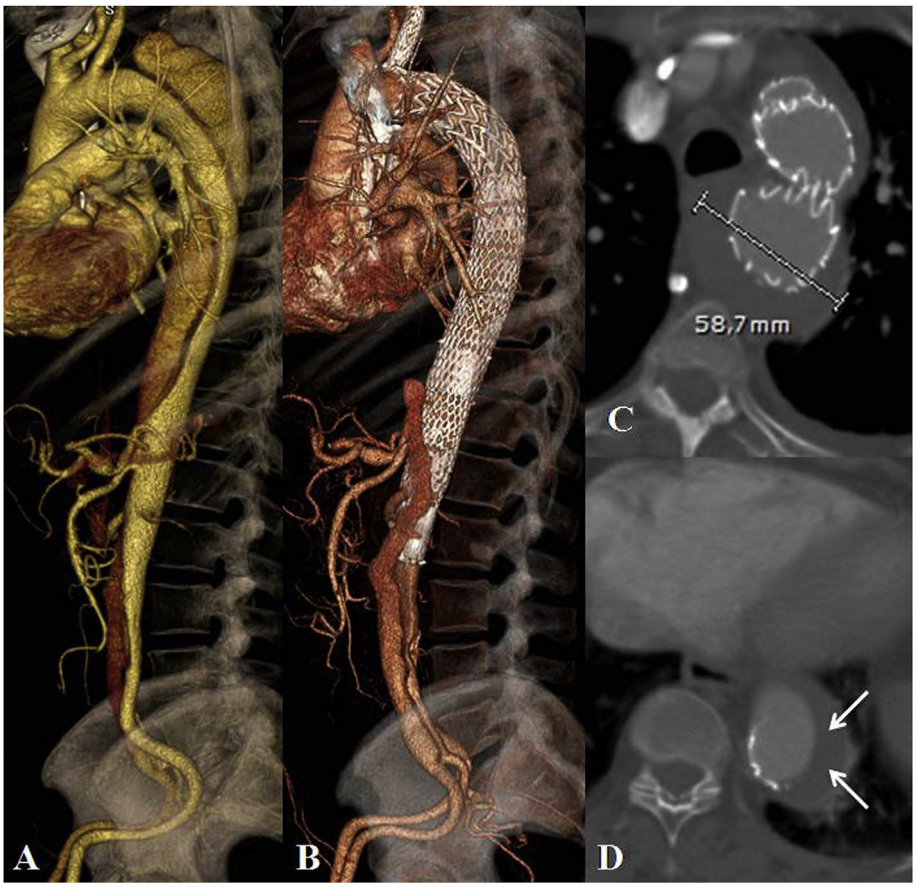

Figure 4. Follow-up CTA after TEVAR plus uncovered stent along the visceral segment for acute type B aortic dissection complicated by aneurysm. False lumen thrombosis obtained along the thoracic endograft (class 1, according to Parsa *et al.*,[6] A, B) and in the total descending aorta (class 2, according to Parsa *et al.*,[6] C, D).

proximal first, and then distally. Balloon angioplasty of the proximal and distal landing zones was not performed on a routine basis. In those cases with planned intentional coverage of the left subclavian artery (LSA), adjunctive prophylactic revascularisation was performed in selected cases (e.g. in stable haemodynamic conditions, when aortic coverage was $\geq$20 cm, when the patient had a previous abdominal aortic aneurysm repair, in those whose coronary circulation was supplied by the left internal mammary, in the presence of a hypoplastic contralateral vertebral artery, left handed professionals, and left arm arteriovenous fistula for dialysis). Cerebrospinal fluid drainage was not performed on a routine basis but selectively (spinal cord bleeding risk from ongoing anticoagulant or antiplatelet therapy, when thoracic aortic coverage was $\geq$20 cm with previous/synchronous abdominal aortic aneurysm repair, and/or LSA coverage).[18,19] Graft materials and manufacturers are reported in Appendix I.

Aortic assessment and follow-up

After intervention, the follow-up imaging protocol included triple phase spiral/CTA performed at 1, 6, and 12 months post-operatively, and on an annual basis thereafter. Attention was focused on the sealing zones, FL thrombotic status, and aortic measurements. Computed tomography scans were reviewed by vascular surgeons and interventional radiologists. False lumen patency was defined by the presence of contrast within the FL on the arterial or venous phase of the CTA. The short axis diameter of the TL, FL, and total aorta was quantified on the pre-operative and the most recent post-operative CTA at different levels (distal aortic arch just below the LSA take off, at the bifurcation of the left and right pulmonary arteries, the diaphragmatic aortic hiatus, and the infrarenal aorta). Aortic diameter was measured adventitia to adventitia; the estimation of the TL/FL ratio before and after TEVAR was measured just below the LSA, and at the distal edge of the SG.

Definition

Type B aortic dissection was classified according to identification of the clinical symptom onset: hyperacute (0—24 hours), acute (2—7 days), subacute (8—30 days), and chronic (30 days).[20] Comorbidities were defined according to the Society of Thoracic Surgeons adult database.[21] Morphological characteristics and outcomes were defined according to the European Association for Cardiothoracic Surgery/European Society for Cardiovascular Surgery (EACTS/ESCVS) best practice guidelines for reporting treatment results in the thoracic aorta, and/or the Society for Vascular Surgery (SVS) ad hoc committee on TEVAR reporting standards.[22,23] Primary technical success was defined as coverage of the proximal entry tear in the

absence of surgical conversion to open repair or death ≤24 hours. Follow-Up Index (FUI) described follow-up completeness at the given study end date as the ratio between the investigated and the potential follow-up periods.[24] TEVAR related mortality included deaths as a result of aneurysm rupture, surgical conversion, or complications of TEVAR unsolved by additional procedures. Favourable aortic remodelling was defined as FL thrombosis in the whole descending thoracic segment or longer, and/or an aneurysm diameter reduction of at least 5 mm. False lumen thrombosis was defined according to Parsa et al.[6]: class 0, designates some retrograde FL perfusion along the SG; class 1, designates thrombosis along the length of SG; class 2, designates thrombosis of FL throughout the thoracic aorta (Figs. 4A, 1, 2); class 3, designates complete thrombosis of FL throughout the entire native aorta (Figs. 4B, 1, 2). The analysis here evaluated early and long-term survival, freedom from aortic related mortality (ARM), and freedom from re-intervention.

Statistical analysis

Clinical data were prospectively recorded and tabulated in Microsoft Excel (Microsoft Corp, Redmond, WA, USA): statistical analysis was performed with SPSS, release 23.0 for Windows (IBM SPSS Inc.; Chicago, IL, USA). Categorical variables were presented as frequencies and percentages, continuous variables as mean ± standard deviation (SD) and interquartile (IQR) range; otherwise medians with range were applied. Wilcoxon's signed-rank test was used to evaluate the difference in TL/FL ratio before and after TEVAR. A p value $< .05$ was considered to be significant. Cumulative survival, freedom from ARM and freedom from re-intervention rates were estimated using the Kaplan-Meier method with 95% confidence interval (CI).

RESULTS

Cohort data

Twenty-two patients were treated, representing 16% of the 179 type B acute aortic dissections observed during the study period, of which 41.5% presented with complications in the hyperacute or acute periods. Connective tissue disorder related DTA was not observed. Demographic data and risk factors are presented in Table 1. All the patients were symptomatic with thoracic pain at presentation; malperfusion was additionally present in two patients (9%), while refractory/recurrent pain was observed in 10 (45.4%). The pre-operative mean total aortic diameter was 66 ± 26 mm (range 42—130; IQR 51—64). Thrombus in the FL was never observed on admission. Free rupture with haemothorax was present in three (14%) patients. Dissection was limited to the descending thoracic aorta in seven (32%) patients, but involved the thoraco-abdominal aorta in 15 (68%).

Operative details

Operative details are reported in Table 2. Urgent (<24 h) intervention was performed in 12 (54.5%) patients: the rest were treated after a mean of 11 ± 4 days (range 7—16; IQR 11—14). Mean aortic length coverage was 22 ± 6 cm (range 10—35; IQR 20—25). Cerebrospinal fluid drainage was used in 5 (23%) patients. All patients were admitted to the

Table 1. Demographic data, comorbidities, and risk factors.

Variable	*n* (%)
Demographic data	
M:F (ratio)	15:7
Age (mean ± SD)	67 ± 8
Risk factors	
Hypertension	22 (100)
COPD	7 (31.8)
Hyperlipidaemia	7 (31.8)
Obesity (BMI >30)	5 (22.7)
Ischaemic heart disease	3 (13.6)
Diabetes	3 (13.6)
Previous proximal aortic surgery[a]	3 (13.6)
Renal insufficiency (GFR <30 mL/min)	2 (9.1)
EuroSCORE II, (mean ± SD)	23 ± 18 (IQR 13—25)

M = male; F = female; COPD = chronic obstructive pulmonary disease; BMI = body mass index; GFR = glomerular filtration rate.
[a] Non-dissection surgery.

Table 2. Operative data.

Variable	*n* (%)
Operative data	
General anaesthesia	22 (100)
Femoral access	22 (100)
Landing zone	
1	2 (9.1)
2	13 (59.1)
3	7 (31.8)
Aortic coverage, (cm ± SD)	
<20	4 (18.2)
≥20	18 (81.8)
Intervention (min ± SD)	192 ± 142
Blood loss (median, mL)	265
Contrast (median, mL)	150
PRBC (median, units)	1
Stent graft, (manufacturer)	
Free-flo	8 (30.8)
Diameter (mm ± SD)	37 ± 3
Open adjunctive procedures	
Carotid-subclavian by-pass	6
Left hemiarch de-branching	2
Chest drain	2
Endovascular adjunctive procedures	
LSA embolisation	4
LSA "chimney"	2
PETTICOAT	2
SMA stenting	1
RAAs stenting	1
LCIA stenting	1
EVAR	1

PRBC = packed red blood cell; LSA = left subclavian artery; PETTICOAT, Provisional Extension to Induce Complete Attachment; SMA = superior mesenteric artery; RRA = right renal artery LCIA = left common iliac artery; EVAR = endovascular abdominal aortic repair.

Table 3. Post-operative complication (SVS grading score[a]).

Complication[a]	n (%)	Treatment
Mild		
Acute kidney injury	2	Conservative
Atrial fibrillation	1	Amiodarone
Acute lung injury	2	C-PAP
Paraparesis	1	KT
Severe		
Acute mesenteric ischaemia	2	Hartman (1); AMS stent (1)
Acute lung injury	1	OTI
Cardiogenic shock	1	Sudden death

KT = kinesiotherapy; SMA = left subclavian artery; SMA = superior mesenteric artery OTI = orotracheal intubation.
[a] See Fillinger et al. (2010).[23]

intensive care unit with a median length of stay of 4 days (range 1–33; IQR 1–4).

Early results

Primary technical success was 91% (n = 20/22): primary conversion was never performed. In hospital TEVAR related mortality was 14% (n = 3): causes of death included cardiogenic shock (n = 1; no aortic rupture), mesenteric ischaemia (n = 1, visceral malperfusion at admission), and multiple organ failure (n = 1). Spinal cord ischaemia occurred in 1 (4.5%) patient (non-disabling paraparesis); retrograde type A aortic dissection (RTAD) was not observed. TEVAR related complications and their treatments are reported in Table 3. Mean hospitalisation was 12 ± 11 days (range 3–50; IQR 5–15): all patients who survived were discharged home, without permanent or definitive physical disability.

Late outcomes

All surviving patients were available for follow-up. The mean follow-up was 56 ± 45 months (range 6–149; IQR 12–82): the mean FUI was 0.97 ± 0.1. During follow-up the cumulative mortality was 26.3%: five patients died after a mean period of 34 months (range 6–84). During follow-up, the TEVAR related mortality was 5% (n = 1): this patient died because of multiple organ failure after OR of a type IV thoraco-abdominal aortic aneurysm. Further causes of death included acute myocardial infarction (n = 1), cancer (n = 1), chronic respiratory insufficiency (n = 1), and sepsis caused by pneumonia (n = 1). Stent graft breakage, migration, or infection was not seen. Kaplan-Meier estimate of survival was 82% (95%CI: 61.5–93) at 1 year, and 64% (95%CI: 41–82) at 5 year (Fig. 5). Seventeen (89%) patients had CTA at 1 year, 12 (63%) at 3 years, and seven (37%) at ≥5 years. Among follow-up CTA evaluations, aneurysm sac enlargement was not observed, while 2 (10.5%) patients had significant aneurysm diameter reduction. Seventeen (89.5%) patients had class 2 thrombosis, but no patient presented with class 3 FL thrombosis. The TL/FL ratio was significantly improved after TEVAR just below the LSA origin (0.7 ± 0.2 vs. 0.9 ± 0.1, p = .002) and the distal edge of the SG (0.7 ± 0.2 vs. 0.9 ± 0.2, p = .025). During the follow-up, ongoing primary clinical success was 79%: four patients required secondary aortic intervention (Table 4). Freedom from ARM at 1 and 5 year was 86% (95%CI: 66–95), while freedom from re-intervention was 95% (95%CI: 75.5–95) at 1 year, and 77% (95%CI: 50–92) at 5 year (Fig. 5).

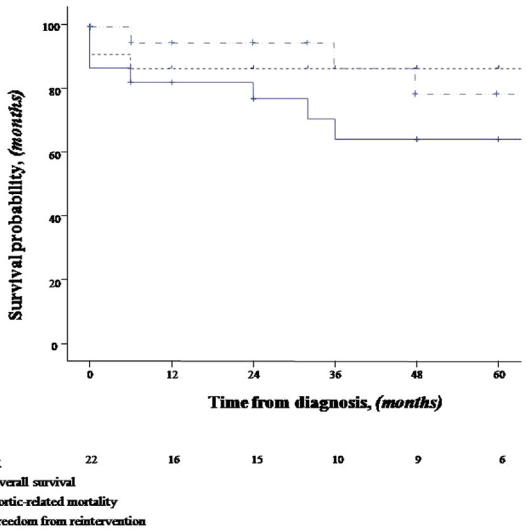

Figure 5. Kaplan-Meier estimates of major aortic outcomes: overall survival, aortic related mortality, and freedom from aortic re-intervention.

Table 4. Follow-up: re-intervention.

Secondary aortic intervention	Months	Intervention	Outcome
Distal SINE with saccular aneurysm	3	TEVAR	Alive
>60 mm type IV TAAA	6	Open repair	Dead
Type 1a endoleak	36	Arch de-branching	Alive
>60 mm type IV TAAA	108	FEVAR	Alive

SINE = stent graft induced new entry site; TAAA = thoraco-abdominal aortic aneurysm; TEVAR = thoracic endovascular aortic repair; LSA, left subclavian artery; FL, false lumen FEVAR, fenestrated endovascular aortic repair.

DISCUSSION

The main findings of this study are the prevalence of DTA as a complication of TBAAD in the hyperacute or acute phases, which is underreported in the literature, and the acceptable early and late results of TEVAR as management, especially when compared with OR.

In the hyperacute or acute phases, complicated TBAAD has been defined by the presence of aortic rupture, major organ malperfusion, or persistent symptoms despite best medical treatment.[1] Few series specifically mentioned DTA ≥5 cm as sudden complication of TBAAD on admission in the early setting. Despite its prevalence being reported in the range of 20–32%, the results of TEVAR for such condition have not been specifically analysed.[1,2,5–17,25] In our

Table 5. Literature summary of TEVAR for dissection related DTA.

Author	Pts (number)	AD delay (months)	TAAD (%)	DB-IIIa (%)	AoCo $\geq$ 20 cm (% or mm $\pm$ SD)	Op M (%)	SCI (%)	AKI (%)	RTAD (%)	FU (months)	FLT (%)	SAI (%)	Redo EVR (%)	Redo M (%)	FFR (%)	ARM (%)	Survival (@ years)
Xu et al.[7]	84	14 $\pm$ 22	n.s.r.	n.s.r.	n.s.r.	1.2	0	4.5	1.2	33 $\pm$ 19	n.s.r.	4.8	57.1	0	n.s.r.	3.6	84.4 @ 5y
Czerny et al.[8]	14	19	14.3	n.s.r.	19	0	n.s.r.	n.s.r.	0	34	35.7	7.1	n.s.r.	n.s.r.	n.s.r.	7.1	n.s.r.
Oberhuber et al.[9]	19	36	n.s.r.	n.s.r.	n.s.r.	0	5.2	0	0	13	5.2	47.3	77.7	0	n.s.r.	n.s.r.	n.s.r.
Kang et al.[10]	47	25	22	n.s.r.	n.s.r.	5	0	2	3.9	24	39	22	n.s.r.	n.s.r.	n.s.r.	n.s.r.	80 @ 3y
Mani et al.[11]	58	29 $\pm$ 31	13.8	24.1	22 $\pm$ 10	5.2	1.7	n.s.r.	6.9	38 $\pm$ 28	38	21.8	50	n.s.r.	71 @ 3y	3.6	64 @ 3y
Nozdrzykowski et al.[12]	32	3.2	18.8	n.s.r.	n.s.r.	6.2	9.3	34.4	n.s.r.	n.s.r.	n.s.r.	28.1	n.s.r.	n.s.r.	n.s.r.	n.s.r.	87.5 @ 3y
Scali et al.[13]	80	16	9	24	78.2	2.5	10	1.2	1.2	18	51	29	17.3	0	70 @ 3y	0	70 @ 5y
Leshnower et al.[14]	31	47 $\pm$ 44	33	39	22 $\pm$ 4	0	0	0	0	21 $\pm$ 20	80.6	19	66.6	n.s.r.	76.9 @ 5	n.s.r.	80 @ 5y
Hughes et al.[15]	32	32 $\pm$ 44	n.s.r.	n.s.r.	23 $\pm$ 6	0	0	n.s.r.	6.3	54	84.4	21.8	80	0	n.s.r.	0	71.2 @ 6y
Nathan et al.[16]	47	54 $\pm$ 50	71.4	57.4	78.7	4.3	6.4	6.4	0	31 $\pm$ 18	85.2	20	88.8	11.1	54 @ 5y	2.2	89 @ 5y
van Bogerijen et al.[17]	32	20 $\pm$ 28	43.7	59.4	71.9	0	0	3.1	0	34.8	53.1	15.6	80	0	87.5 @ 3y	n.s.r.	78.1 @ 5
Overall (mean)	43	27	28.2	44.6	76.2	2.2	3.2	6.4	1.9	30	52.5	21.5	64.7	1.8		3	

DTA = descending thoracic aneurysm; Pts = patients; AD = aneurysm onset from acute dissection; TAAD = acute type A aortic dissection; AoCo = aortic coverage, meaning extent of total descending thoracic aortic coverage; Op M = operative mortality; SCI = spinal cord ischaemia; CVA = cerebrovascular accident; AKI = acute kidney injury; RTAD = retrograde acute type A aortic dissection; FLT = false lumen thrombosis; SAI = secondary aortic intervention; EVR = endovascular repair; Redo M = mortality of redo endovascular repair; FFR = freedom from re-intervention; ARM = aortic related mortality; SCI = spinal cord ischaemia; n.s.r. = not specifically reported.

experience, aortic dilatation was more common than other types of complications at the clinical onset of TBAAD: although it involved only 16% of all TBAADs, it represented 41.5% of the complication events and was the most frequent indication for immediate operative repair.

Open repair for complicated TBAAD is associated with a significant in hospital mortality rate, in the range of 11.5—29.3%.[1,2,5] Large data set analysis showed that TEVAR for thoracic aortic dissection is associated with a significantly lower mortality rate and a greater number of patients discharged home.[3] In our series, TEVAR related mortality was 14%: this is a higher rate than the average 2.2% reported in the literature summary (Table 5), but lower than the 23% predicted mortality for OR.[26] Moreover, almost all series in the literature analysed a mixed cohort of dissecting aneurysms, and did not specify which of the cases were TBAAD complicated by DTA since the beginning of clinical onset.

Promoting thrombosis of the FL and aortic remodelling are recognised benefits of TEVAR when treating TBAAD.[3,4,14,15] Recently, data from the ADSORB trial showed significantly better remodelling at 1 year induced by the best medical treatment plus SG in the acute setting: in particular, 57% FL thrombosis, 63% of freedom from aortic dilatation, and no rupture.[27] Although our cohort represents a different clinical setting, and specific volumetric analysis was not performed, in this challenging context TEVAR yielded similar positive results, such as the cumulative 89.5% FL thrombosis rate, a significant improvement in the TL/FL ratio, and the absence of thoracic aortic enlargement. This satisfactory outcome could be explained by the combination of variables, such as effective abolition of the antegrade flow induced by the proximal aortic entry tear closure, the smoothness and plasticity of the lamella that may be completely remodelled, and the absence of FL thrombosis at presentation.

Secondary aortic interventions have commonly been described in TEVAR series reporting on TBAADs, usually with a higher prevalence compared with re-intervention after OR.[6—17] Nevertheless, 65% of re-interventions identified in the present literature summary were managed endovascularly, and the redo related mortality was lower than the mortality rate of the first intervention. In the present series, the cumulative 79% freedom from aortic re-intervention rate is similar to the mean rate noted in the literature summary. Furthermore, secondary aortic interventions were performed predominantly with endovascular SG, and ARM was only associated with the need of open conversion (graft replacement of a type IV TAAA).

Although the estimated overall survival was as low at 64% at 5 years in our series, the estimated ARM was 86%, which is in line with the 70—89% survival range at 5 years reported in the literature summary. A clear explanation for the worse survival of patients with a TBAAD and a dilatation at admission is difficult to find. Firstly, our cohort included only TBAAD treated in the urgent setting because of a complication of the disease in the acute phase, which describes a higher-risk cohort, while the literature summary reported only subacute and/or chronic patients. Secondly, the high operative risk of this cohort does not find meaningful comparison because the literature lacks references to specific operative risk stratification, or homogeneity in the follow-up period. In addition, differences could be related to the high follow-up compliance (>60% at 3 years had follow-up CTA), which was an issue described in other experiences.[10,11,13,16] Lastly, the present data reflect strict observation of the outcome definitions, which is often not present or reported heterogeneously in other series.[16] Future studies comparing TEVAR results in TBAAD with and without DTA/significant aortic enlargement will be useful to better define the risk profile and the mid-/long-term outcome of such cohorts.

Limitations

The present study has clear limitations. First, it was a retrospective study with sampling bias: neither medical therapy alone nor OR arms were included for comparison. Multivariate analysis would have been misleading with this cohort size and thus was not performed. Consequently, results may be underpowered to demonstrate meaningful associations between pre-operative predictors and post-operative outcomes. Lastly, no volumetric analysis was performed, which may have offered a more comprehensive and precise assessment of aortic remodelling for dissection related pathology. Despite these limitations, inclusion criteria resulted in a homogeneous cohort in terms of epidemiological and demographic data. Follow-up was performed at approximately 5 years and included clinical visits and radiological examinations. Outcomes adhered systematically to the proposed guidelines, which compare well with those of other studies.

CONCLUSION

In our experience, DTA is a frequent complication from the very beginning of the clinical onset of TBAAD. In this high-risk cohort of patients, TEVAR showed satisfactory results, better than those predicted by the risk score for OR, with favourable stability of the aortic diameter and no aortic related adverse events during follow-up.

CONFLICT OF INTEREST

None.

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APPENDIX A. SUPPLEMENTARY DATA

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.ejvs.2017.02.022.

REFERENCES

1 Trimarchi S, Nienaber CA, Rampoldi V, Myrmel T, Suzuki T, Bossone E, et al. IRAD Investigators. Role and results of surgery in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). *Circulation* 2006;**114**(1 Suppl):I357–64.

2 Bozinovski J, Coselli JS. Outcomes and survival in surgical treatment of descending thoracic aorta with acute dissection. *Ann Thorac Surg* 2008;**85**(3):965–70.

3 Conrad MF, Ergul EA, Patel VI, Paruchuri V, Kwolek CJ, Cambria RP. Management of diseases of the descending thoracic aorta in the endovascular era: a medicare population study. *Ann Surg* 2010;**252**(4):603–10.

4 Wilkinson DA, Patel HJ, Williams DM, Dasika NL, Deeb GM. Early open and endovascular thoracic aortic repair for complicated type B aortic dissection. *Ann Thorac Surg* 2013;**96**(1):23–30.

5 Pujara AC, Roselli EE, Hernandez AV, Vargas Abello LM, Burke JM, Svensson LG, et al. Open repair of chronic distal aortic dissection in the endovascular era: implications for disease management. *J Thorac Cardiovasc Surg* 2012;**144**(4):866–73.

6 Parsa CJ, Schroder JN, Daneshmand MA, McCann RL, Hughes GC. Midterm results for endovascular repair of complicated acute and chronic type B aortic dissection. *Ann Thorac Surg* 2010;**89**(1):97–102.

7 Xu SD, Huang FJ, Yang JF, Li ZZ, Yang S, Du JH, et al. Early and midterm results of thoracic endovascular aortic repair of chronic type B aortic dissection. *J Thorac Cardiovasc Surg* 2010;**139**(6):1548–53.

8 Czerny M, Roedler S, Fakhimi S, Sodeck G, Funovics M, Dumfarth J, et al. Midterm results of thoracic endovascular aortic repair in patients with aneurysms involving the descending aorta originating from chronic type B dissections. *Ann Thorac Surg* 2010;**90**(1):90–4.

9 Oberhuber A, Winkle P, Schelzig H, Orend KH, Muehling BM. Technical and clinical success after endovascular therapy for chronic type B aortic dissections. *J Vasc Surg* 2011;**54**(5):1303–9.

10 Kang WC, Greenberg RK, Mastracci TM, Eagleton MJ, Hernandez AV, Pujara AC, et al. Endovascular repair of complicated chronic distal aortic dissections: intermediate outcomes and complications. *J Thorac Cardiovasc Surg* 2011;**142**(5):1074–83.

11 Mani K, Clough RE, Lyons OT, Bell RE, Carrell TW, Zayed HA, et al. Predictors of outcome after endovascular repair for chronic type B dissection. *Eur J Vasc Endovasc Surg* 2012;**43**(4):386–91.

12 Nozdrzykowski M, Etz CD, Luehr M, Garbade J, Misfeld M, Borger MA, et al. Optimal treatment for patients with chronic Stanford type B aortic dissection: endovascularly, surgically or both? *Eur J Cardiothorac Surg* 2013;**44**(3):e165–174.

13 Scali ST, Feezor RJ, Chang CK, Stone DH, Hess PJ, Martin TD, et al. Efficacy of thoracic endovascular stent repair for chronic type B aortic dissection with aneurysmal degeneration. *J Vasc Surg* 2013;**58**(1):10–7.

14 Leshnower BG, Szeto WY, Pochettino A, Desai ND, Moeller PJ, Nathan DP, et al. Thoracic endografting reduces morbidity and remodels the thoracic aorta in DeBakey III aneurysms. *Ann Thorac Surg* 2013;**95**(3):914–21.

15 Hughes GC, Ganapathi AM, Keenan JE, Englum BR, Hanna JM, Schechter MA, et al. Thoracic endovascular aortic repair for chronic DeBakey IIIb aortic dissection. *Ann Thorac Surg* 2014;**98**(6):2092–7.

16 Nathan DP, Shalhub S, Tang GL, Sweet MP, Verrier ED, Tran NT, et al. Outcomes after stent graft therapy for dissection-related aneurysmal degeneration in the descending thoracic aorta. *J Vasc Surg* 2015;**61**(5):1200–6.

17 van Bogerijen GH, Patel HJ, Williams DM, Yang B, Dasika NL, Eliason JL, et al. Propensity adjusted analysis of open and endovascular thoracic aortic repair for chronic type B dissection: a twenty-year evaluation. *Ann Thorac Surg* 2015;**99**(4):1260–6.

18 Matsumura JS, Lee WA, Mitchell RS, Farber MA, Murad MH, Lumsden AB, et al. Society for Vascular Surgery. The Society for Vascular Surgery Practice Guidelines: management of the left subclavian artery with thoracic endovascular aortic repair. *J Vasc Surg* 2009;**50**(5):1155–8.

19 Etz CD, Weigang E, Hartert M, Lonn L, Mestres CA, Di Bartolomeo R, et al. Contemporary spinal cord protection during thoracic and thoracoabdominal aortic surgery and endovascular aortic repair: a position paper of the vascular domain of the European Association for Cardio-Thoracic Surgery. *Eur J Cardiothorac Surg* 2015;**47**(6):943–57.

20 Booher AM, Isselbacher EM, Nienaberca, Trimarchi S, Evangelista A, Montgomery DG. IRAD Investigators. The IRAD classification system for characterizing survival after aortic dissection. *Am J Med* 2013;**126**:19–24.

21 Society of Thoracic Surgeons, Committee to develop a national database for thoracic surgeons. Definitions of terms of the Society of Thoracic Surgeons national cardiac surgery database. *Ann Thorac Surg* 1994;**58**:271–3.

22 Turina MI, Shennib H, Dunning J, Cheng D, Martin J, Muneretto C, et al. EACTS/ESCVS committee. EACTS/ESCVS best practice guidelines for reporting treatment results in the thoracic aorta. *Eur J Cardiothorac Surg* 2009;**35**(6):927–30.

23 Fillinger MF, Greenberg RK, McKinsey JF, Chaikof EL. Society for Vascular Surgery Ad Hoc Committee on TEVAR Reporting Standards. Reporting standards for thoracic endovascular aortic repair (TEVAR). *J Vasc Surg* 2010;**52**(4):1022–33.

24 von Allmen RS, Weiss S, Tevaearai HT, Kuemmerli C, Tinner C, Carrel TP, et al. Completeness of follow-up determines validity of study findings: results of a prospective repeated measures cohort study. *PLoS One* 2015;**10**(10):e014081.

25 Tolenaar JL, Froehlich W, Jonker FH, Upchurch Jr GR, Rampoldi V, Tsai TT, et al. Predicting in-hospital mortality in acute type B aortic dissection: evidence from international registry of acute aortic dissection. *Circulation* 2014 Sep 9;**130**(11 Suppl 1). S45–50.

26 Nashef SA, Roques F, Sharples LD, Nilsson J, Smith C, Goldstone AR, et al. EuroSCORE II. *Eur J Cardiothorac Surg* 2012;**41**(4):734–44.

27 Brunkwall J, Kasprzak P, Verhoeven E, Heijmen R, Taylor P. ADSORB Trialists. Endovascular repair of acute uncomplicated aortic type B dissection promotes aortic remodelling: 1 year results of the ADSORB trial. *Eur J Vasc Endovasc Surg* 2014;**48**(3):285–91.