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ORIGINAL ARTICLE

Thoracic endovascular aortic repair for the treatment of ruptured acute type B aortic dissection

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Abstract

Purpose To evaluate the efficacy of thoracic endovascular aortic repair (TEVAR) for ruptured acute type B aortic dissection (r-ATBAD).

Materials and methods The study included 18 patients (15 men and 3 women) who underwent TEVAR for r-ATBAD in two institutions between 1997 and 2017. The mean patient age was 74 ± 10 years. The false lumen was patent in 13 patients (72%) and was mostly thrombosed in 5 patients (28%). Three patients had malperfusion of aortic branches. Eight patients (44%) were in circulatory shock.

Results Eleven patients (61%) died during or following TEVAR during admission. The causes of death were aortic rupture ($n = 6$), sepsis ($n = 2$), cerebral hypoxia ($n = 1$), pneumonia ($n = 1$), and renal failure ($n = 1$). Statistical analysis showed that dissection extending to the infrarenal level was significantly related to death from aortic rupture ($P = 0.013$). Early adverse events were observed in 12 patients (67%). One patient died from a non-aorta-related cause (sepsis) after discharge. The overall survival rate at 1 year was 39%. After discharge, an aorta-related adverse event (intimal injury) was observed in one patient. The adverse event-free survival rate at 1 year was 17%.

Conclusions Our results indicate that TEVAR for r-ATBAD is associated with high mortality and morbidity. More advanced strategies may be required to improve the outcome.

Keywords Aortic dissection · Complication · Rupture · Endovascular repair · Stent graft

Introduction

Ruptured acute type B aortic dissection (r-ATBAD) is one of the most catastrophic events affecting the aorta. It accounts for approximately 10–20% of whole acute aortic dissection cases and is one of the most frequent causes of death [1–5]. With open surgical repair, the operative mortality rate has been reported to exceed 20% [6–8]. In addition, many patients died without any intervention attempt during admission because of poor general condition.

Thoracic endovascular aortic repair (TEVAR) has been recognized as a first-line treatment approach for complicated acute aortic dissection since Dake et al. reported its efficacy in the late 1990s [9]. The target complications range widely from relatively mild complications, such as refractory hypertension, to severe complications, such as malperfusion of the aortic branches and aortic rupture. Previous reports showed obvious superiority of TEVAR to open repair for the relief of dissection-related complications including aortic rupture [9–16].

The purpose of the present study is to assess our 21-year experience of TEVAR for r-ATBAD to clarify its efficacy and identify factors leading to death from aortic rupture.

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Materials and methods

Study design

This retrospective study reviewed the medical records of 25 patients with r-ATBAD who had been transported to two institutions between July 1997 and January 2017. The study was approved by the institutional review board of each participating institution. Written informed consent was obtained from the patients who were alive and who were being followed up. For patients who were dead or lost to follow-up, we posted an explanation of the study on our institutional websites and allowed them or their families to decide on opting out of the study.

Patient selection

Since 1997, when TEVAR was initiated in our institutions, the first-line treatment for r-ATBAD has been TEVAR whenever patients' anatomy was suitable.

All patients underwent emergent contrast-enhanced computed tomography (CT) immediately after being transported to the emergency room. Following confirmation of r-ATBAD on CT, the possibility of TEVAR was assessed with regard to the landing zone and the access route. Specifically, the landing zone proximal to the entry tear needed to be 2.0 cm or longer and 40 mm or smaller in diameter. The access route needed to be large enough to allow the delivery system. Patients who were judged as unfit for TEVAR or open surgical repair because of high age or poor general condition were treated medically.

Procedural details

Until 2008, non-commercial custom-made devices manufactured with Z stents (Cook Inc., Bloomington, IN, USA) covered with expanded polytetrafluoroethylene (Bard, Tempe, AZ, USA) or polyester (UBE, Tokyo, Japan) had been used. Subsequently, commercial devices became available, and these commercial devices were used. The commercial devices used were Talent (Medtronic, Minneapolis, MN), TX2 (Cook, Bloomington, IN), TAG, and CTAG (W.L. Gore & Assoc., Flagstaff, AZ). The device diameter was determined according to the aortic diameter at the proximal and distal landing zones. Measurement of diameters was performed on axial CT and angiography, being performed mainly on curved planar reconstruction CT images in the later period. As a basic rule, the device diameter was oversized by 10–20% when compared with the aortic diameter at the proximal landing zone when the proximal landing zone was not dissected. When a dissected portion (both proximally and distally) had to be selected as the landing zone (i.e., bypass to aortic branches to secure a sufficiently long and healthy landing zone could not be performed), a device diameter equal to or slightly smaller than the aortic diameter and larger than the true lumen diameter was adopted.

When non-commercial custom-made devices were used, the procedural endpoint was only entry closure due to the difficulty of use of long devices. Since commercial devices were available, they were placed from the aortic arch or the proximal descending aorta proximally to mid- or distal descending aorta distally to cover the assumed rupture site sufficiently in addition to the entry.

When antihypertensive therapy was judged as necessary, it was initiated immediately after diagnosis and was continued after TEVAR. When respiratory failure caused by a hematoma in the chest was identified, a drainage tube was placed or the hematoma was removed under thoracoscopy to relieve the respiratory failure.

Follow-up

Antihypertensives were administrated in all patients who survived. Follow-up CT was performed at 1 week, 1 month, 3 months, 6 months, and 1 year after TEVAR and yearly thereafter. For patients who could not continue visiting the hospital, their clinical courses were determined by making phone calls to their family doctors or their own homes.

Statistical analysis

Data are shown as frequencies, percentages, or medians and interquartile ranges (IQR). Missing data were not defaulted to negative, and denominators reflected only reported cases. We defined survival from aortic rupture as the primary endpoint of the treatment strategy. To detect the contributing factors to survival from aortic rupture, nominal variables were compared using Fisher's exact test and continuous variables were compared using Student's *t* test between the group who survived aortic rupture and that who died of aortic rupture. Survival analysis was performed according to the Kaplan–Meier method. All analyses were performed using a statistical software package (SPSS 25, IBM Corporation, Armonk, NY, USA). A P value < 0.05 was considered significant.

Results

Patient characteristics

Among 25 patients with r-ATBAD, 3 patients underwent open surgical intervention because of anatomical

limitations. Four patients did not undergo any invasive intervention because of high age or poor general condition. The remaining 18 patients underwent TEVAR. There were 15 men and 3 women. The patient characteristics are shown in Table 1. The mean patient age was 74 ± 10 years. Six patients (33%) were older than 80 years. Approximately 80% of the patients had hypertension. Eight patients (44%) were in circulatory shock, and TEVAR was performed under percutaneous cardiopulmonary support (PCPS) in one of these patients. With regard to dissection-related comorbidities other than aortic rupture, visceral ischemia was observed in two patients and renal ischemia was observed in one patient.

No variable showed a significant difference between the patients who died of aortic rupture and the other patients.

Preoperative CT findings

Table 2 shows the preoperative CT findings. The false lumen of the descending thoracic aorta was completely patent in 13 patients (72%) and more than 90% of the lumen was thrombosed, except for the entry tear site, in the remaining 5 patients (28%). Three patients had coexisting true thoracic aortic aneurysms.

The entry tear was located at Zone 3 in 12 patients and at Zone 4 in 6 patients. The dissection reached Zone 2 in 3 patients, Zone 3 in 14, and Zone 4 in 1 proximally. Additionally, the dissection was limited to the suprarenal part of the descending aorta in 8 patients, whereas it reached the infrarenal part in the remaining 10 patients distally. The dissection reached below the renal arteries in significantly more patients who died of aortic rupture than patients who survived aortic rupture (60% vs. 0%, $P = 0.013$).

Table 1 Demographics and comorbidities

Variable	Overall	Survived rupture	Died of rupture	*P* value
n (%)	18 (100%)	12 (67%)	6 (33%)	
Age, mean (SD), years	74 (± 10)	75(± 8)	73 (± 13)	0.664
Age ≥ 80 years	6 (33%)	4 (33%)	2 (33%)	1.000
Male sex	15 (83%)	10 (83%)	5 (83%)	1.000
Smoking	7 (64%)	5 (63%)	2 (67%)	1.000
Comorbidities				
Hypertension	10 (77%)	10 (83%)	0 (0%)	0.231
Dyslipidemia	1 (7%)	1 (10%)	0 (0%)	1.000
Diabetes mellitus	1 (6%)	1 (8%)	0 (0%)	1.000
Coronary artery disease	1 (11%)	1 (11%)	[a]	
Cerebrovascular disease	4 (36%)	4 (36%)	[a]	
Chronic kidney disease	5 (31%)	4 (36%)	1 (20%)	1.000
Pulmonary disease	1 (11%)	1 (11%)	[a]	
History of thoracotomy	1 (10%)	0 (0%)	1 (100%)	0.100
Dissection-related comorbidities				
Circulatory shock	8 (44%)	5 (42%)	3 (50%)	1.000
Visceral ischemia	2 (11%)	1 (8%)	1 (17%)	1.000
Renal ischemia	1 (6%)	1 (8%)	0 (0%)	1.000

SD standard deviation

[a]No record

Table 2 Preoperative computed tomography findings

Variable	Overall	Survived rupture	Died of rupture	*P* value
Patent false lumen	13 (72%)	8 (67%)	5 (83%)	0.615
Coexistence of TAA*	3 (17%)	1 (6%)	2 (33%)	0.245
Location of the entry, Z3	12 (67%)	8 (67%)	4 (67%)	1.000
Top end of dissection, Z2	3 (17%)	3 (25%)	0 (0%)	0.515
Bottom end of dissection, infrarenal	10 (56%)	4 (%)	6 (100%)	0.013*

*Significant at 5% level of probability

TAA thoracic aortic aneurysm

Table 3 Treatment details

Variable	Overall	Survived rupture	Died of rupture	*P* value
Device, non-commercial	6 (33%)	5 (42%)	1 (17%)	0.600
Top end of device location, Z1–Z2	5 (28%)	4 (33%)	1 (17%)	0.615
Bottom end of device location, above T12	16 (89%)	11 (92%)	5 (83%)	1.000
Treatment length, mean (SD), mm	183 (±70)	166 (±68)	217 (±67)	0.153
Device/aorta ratio at the proximal neck, mean (SD)	1.00 (±0.15)	0.95 (±0.58)	1.10 (±0.09)	0.049*
Device/aorta ratio at the distal neck, mean (SD)	0.99 (±0.15)	0.99 (±0.15)	0.99 (±0.17)	0.933

*Significant at 5% level of probability

SD standard deviation

Treatment details

Treatment details are shown in Table 3. Non-commercial custom-made devices were used in 6 patients (33%) until 2008, whereas commercial devices were used in 12 patients. The following commercial devices were used: Talent ($n=2$; Medtronic, San Jose, CA, USA), TX2 ($n=6$; Cook Inc., Bloomington, IN, USA), and TAG/CTAG ($n=4$; W. L. Gore & Associates, Flagstaff, AZ, USA). The mean treatment length was 182 ± 70 mm. The mean treatment length was 103 ± 27 mm in the initial period with non-commercial devices. In contrast, the mean treatment length was 233 ± 45 mm in the later period when commercial devices became available, which was significantly longer than in the initial period ($P=0.000$).

Among the 18 patients, the bottom end of the device was above T12 in 16 patients (89%) and was at or below T12 in 2 patients (11%). The mean ratio of the diameter of the top end of the device to the aortic diameter was 1.00 ± 0.15, and the mean ratio of the diameter of the bottom end of the device to the aortic diameter was 0.99 ± 0.15. At the proximal neck, the oversizing was slightly larger in patient with repeated rupture than those without repeated rupture in our series ($P=0.049$).

Debranching of cervical vessels was required in three patients. All three vessels were debranched in one patient, and the left common carotid artery and left subclavian artery were debranched in two patients.

No variable showed a significant difference between the patients who died of aortic rupture and the other patients.

Early outcomes

Following TEVAR, 11 patients (61%) died during admission. The causes of death were aortic rupture in six patients, sepsis in two, cerebral hypoxia in one, pneumonia in one, and renal failure in one (Table 4). Among the six patients who died of aortic rupture, one died of aortic rupture 4 days after TEVAR, whereas the other five died of aortic rupture during or immediately after TEVAR (Fig. 1). One patient who underwent TEVAR under PCPS died of cerebral hypoxia 13 days after TEVAR.

Table 4 Causes of in-hospital death

Cause	*N*
Aortic rupture	6
Sepsis	2
Cerebral hypoxia	1
Pneumonia	1
Renal failure	1

Early aorta- or TEVAR-related adverse events (including the causes of death) were observed in 12 patients (67%) (Table 5). In one patient, aortic rupture developed 2 days after TEVAR, and it was repaired with additional TEVAR. Except for this patient, 6 of 7 patients who suffered aortic rupture during hospitalization died as mentioned above. Type Ia endoleak was observed in two patients, and both of these patients died of aortic rupture during TEVAR before adding further intervention. Type IIIb endoleak was observed on CT obtained immediately after TEVAR in one patient. This patient developed an aorto-esophageal fistula during hospitalization and eventually died of sepsis. Access route injury developed in two patients. Graft interposition to the external iliac artery was additionally performed in one patient, whereas a stent graft was placed in the external iliac artery in another patient. Minor dissection of the right common carotid artery near the anastomotic site developed in one patient, which was observed without any intervention.

Late outcomes

Seven patients were alive and were discharged. During a median follow-up period of 30 months (interquartile range 19–118 months), one patient died of sepsis. Five among the remaining six patients are being followed up without any adverse event. Intimal injury at the bottom end of the device was observed in one patient 3 months after TEVAR. An additional stent graft was placed in this patient.

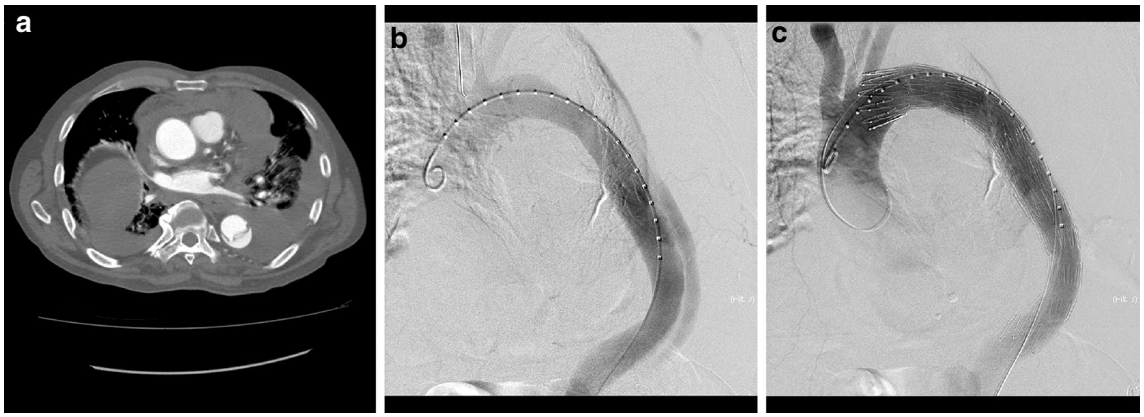

Fig. 1 Contrast-enhanced CT shows r-ATBAD with massive mediastinal and pleural hematoma in a man in his 80s (**a**). Pre-TEVAR DSA shows the entry tear in the mid-descending aorta (**b**). Post-TEVAR DSA shows complete obliteration of the entry tear with stent grafts placed from the proximal descending aorta to T11 level (**c**). However, he died of repeated rupture immediately after TEVAR

Table 5 In-hospital morbidities related to the aorta or TEVAR

Morbidities	*N*
Aortic rupture	7 (38%)
Type Ia endoleak	2 (11%)
Type IIIb endoleak	1 (6%)
Access route injury	2 (11%)
Cerebral infarction	2 (11%)
Cerebral hypoxia	1 (6%)
Paraplegia	1 (6%)
Dissection of the right common carotid artery	1 (6%)
Aorto-esophageal fistula	1 (4%)

According to the Kaplan–Meier method, the overall survival rate was 39% at 1 year, and the aorta- or TEVAR-related adverse event-free survival rate was 17% at 1 year.

Discussion

The first report on TEVAR for acute aortic dissection by Dake et al. included both uncomplicated and complicated acute aortic dissection cases [9]. Among the complicated cases, three had aortic rupture and two survived following TEVAR. Following this report, the use of TEVAR for acute aortic dissection has rapidly spread. Its most important value has been its superiority to open surgery for the treatment of aortic dissection accompanied with serious complications, that is, malperfusion of aortic branches and aortic rupture. The rationale of TEVAR for malperfusion is clear, as closure of the entry tear with a stent graft immediately decreases the false lumen pressure and resolves the true lumen compression. This was proven by an experimental model by Chung et al. and was confirmed in clinical practice by Dake et al. [9, 17]. Since then, the efficacy of TEVAR for aortic dissection in the treatment of malperfusion has been well recognized, and it is currently considered as a class I treatment option [18].

Many investigators have claimed that TEVAR is a safe and effective method for the treatment of aortic dissection complicated with aortic rupture [9–16]. In the past, operative mortality was extremely high when open surgical intervention was the only treatment option [6–8]. On the other hand, the mortality rate associated with TEVAR for r-ATBAD did not exceed 20% in most previous studies. This is because TEVAR does not require thoracotomy, hypothermia, full heparinization, or use of an artificial cardiopulmonary pump.

In contrast, our results were quite disappointing, and the in-hospital mortality rate was over 50%, which is worse than the rate reported for open surgery in the treatment of r-ATBAD in recent studies [5, 19–21]. Surprisingly, aortic rupture was the cause of death in 55% (6/11) of patients in our series. There are several possible reasons for the poor outcome. The false lumen with the ruptured adventitia usually has many communications with the aortic true lumen and aortic branches. These communications include the primary entry tear, fenestrations between the true lumen and false lumen made by pullout of aortic branches during propagation of the dissection process, and aortic branches themselves pulled out from the true lumen and supplied by the false lumen. Theoretically, complete block of blood flow from all these communications is required to treat r-ATBAD. In addition, Bozinovski et al. commented from the view of experienced surgeons that rupture tends to occur around the aorta crossing the diaphragm [7]. Therefore, coverage of the entire thoracic aorta would be preferable for the treatment of r-ATBAD. Indeed, mortality rate does not exceed 20% in the recent large series in which the entire thoracic aorta was covered, while the frequency of spinal cord ischemia was

less than 10% [12, 15]. However, there have been a couple of studies in which acceptable outcomes were obtained with much shorter devices [11, 16]. Taking into consideration these reports claiming different treatment strategies with similar outcomes, another important factor other than treatment length may be affecting the results.

Oversizing should be considered a crucial point when aortic dissection is treated with TEVAR. Considering that the aorta should be shrunk or collapsed when compared with the usual size in patients with aortic rupture, especially in a circulatory shock state, the device diameter could have been larger than the calculated one on emergent CT. In addition, the device that has a bottom end diameter large enough to press the intimal flap against the adventitia and eliminate the false lumen might have been more effective, although the risk of retrograde type A aortic dissection or stent graft-induced new entry might increase, instead [22–25]. In our series, the mean oversizing rate was almost 0% at both proximal and distal necks, which might have contributed to poor results. At the proximal neck, the oversizing was slightly larger in patient with repeated rupture than those without repeated rupture in our series. This would be explained by the fact that there were more patients without repeated rupture in whom the top of the device was placed at the dissected portion.

For possibly improving outcomes, there are some options. One is adopting the strategy of covering the entire thoracic aorta as described above. However, the approach involving coverage of the entire thoracic aorta might not be sufficient, because a type II endoleak can persist even after the entire thoracic aorta is covered. Furthermore, upward flow in the false lumen coming from the abdominal aorta can potentially threaten control of the aortic rupture. In our series, propagation of the dissection process to the infrarenal level was a significant factor for death from rupture. This may be explained by the fact that there are more communications between the true and false lumens created by pullout of aortic branches in patients with dissecting process propagating to the infrarenal level, which would contribute to bulky upward blood flow in the false lumen. With regard to the avoidance of these risks, more oversizing to compress the intimal flap to completely eliminate the false lumen may show good results. Embolization of the false lumen could be another option. In case of endovascular aneurysm repair of a ruptured abdominal aortic aneurysm, embolization using various materials could be added to repair the ruptured adventitial site [26]. With respect to aortic dissection, Hashimoto et al. reported a case with ruptured chronic aortic dissection, in which they combined coil embolization of the false lumen and TEVAR and achieved rupture control [27].

The present study has several limitations. First, as this was a retrospective study, it was difficult to obtain a complete data set. In particular, among patients who died immediately after TEVAR, data on coexisting problems were not satisfactory because of the emergent nature of the procedure. Second, the study period was more than 20 years, and the devices and treatment strategies changed drastically over this period. In the initial period when only non-commercial devices were available, the treatment length was quite short, which led to the strategy of entry closure alone. With the introduction of commercial devices, the treatment length significantly increased, although there was no significant difference in the frequency of death from aortic rupture. Third, the number of patients was extremely limited; thus, potentially significant factors contributing to death from rupture might not have been detected. Therefore, it would be difficult to draw any definitive conclusions from our results. A prospective randomized trial would be ideal; however, it is unrealistic because of ethical issues. Therefore, accumulation of more cases with further effort to improve the outcome is necessary to evaluate the efficacy of TEVAR for r-ATBAD.

Conclusions

Our results indicate that TEVAR for r-ATBAD is associated with high mortality and morbidity. To improve the outcome, it might be necessary to cover the entire descending thoracic aorta, ensure further oversizing of the device, and introduce adjunctive procedures including embolization of the false lumen.

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Compliance with ethical standards

Conflict of interest The authors declare that they have no conflict of interest.

Ethical statement All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent Informed consent was obtained from all individual participants included in this study.

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