Complicated Acute Aortic Syndromes Affecting the Descending Thoracic Aorta: Endovascular Treatment Compared With Open Repair

Address for correspondence:
Guering Eid-Lidt, MD
Department of Interventional
Cardiology
National Institute of Cardiology
"Ignacio Chávez"
Juan Badiano 1,
CP 14080 Mexico City,
Mexico
guering@yahoo.com

Guering Eid-Lidt, MD, FSCAI; Jorge Gaspar Hernández, MD, FSCAI, FACC;
Hector González-Pacheco, MD, FACC; Pablo Acevedo Gómez, MD; Samuel Ramírez
Marroquín, MD; Valentín Herrera Alarcon, MD; Jorge Cervantes Salazar, MD; Marco
Martínez-Ríos, MD, FSCAI, FACC

Department of Interventional Cardiology (Eid-Lidt, Gaspar Hernández, Acevedo Gómez);
Coronary Care Unit (González-Pacheco); Department of Cardiovascular Surgery (Ramírez
Marroquín, Herrera Alarcon, Cervantes Salazar), and; General Director (Martínez-Ríos), National
Institute of Cardiology "Ignacio Chávez," Mexico City, Mexico

ABSTRACT

Background: For patients with complicated acute thoracic aortic syndromes, endovascular treatment, when feasible, is preferred over open surgery. However, there are limited data on the long-term benefits of endovascular treatment in complicated acute aortic syndromes affecting the descending thoracic aorta.

Hypothesis: The endovascular treatment is expected to have more favorable long-term mortality and fewer late reintervention in complicated acute thoracic aortic syndromes.

Methods: Of 155 consecutive patients with acute aortic syndromes, 94 met the inclusion criteria of the study; 63 underwent endovascular repair (group 1) and 31 underwent open repair (group 2). Patients with a diagnosis of acute aortic syndrome localized in the descending thoracic aorta distal to the emergence of the left subclavian artery, complicated by rupture, malperfusion syndrome, and/or acute aortic expansion, were included. Indications for repair of the descending thoracic aorta included impending rupture in 70.2%, malperfusion syndrome in 29.8%, and persistence of pain with aortic expansion (aortic diameter >40 mm) in 2 patients.

Results: During the follow-up period (63.0 ± 24.6 months), the cumulative survival free from cardiovascular death rates at 5 years was 92.0% and 51.4% in group 1 and 2, respectively (log rank $P = 0.0001$). Late mortality related to the aorta was 1.6% with thoracic endovascular aortic repair and 0% with surgical treatment.

Conclusions: Endovascular treatment in patients with complicated acute thoracic aortic syndromes localized at the descending thoracic aorta is feasible and safe, with a lower rate of early complications and similar long-term benefits when compared with surgical treatment.

Introduction

Acute aortic syndrome (AAS) comprises a group of potentially life-threatening entities.¹ For patients with life-threatening complications, such as malperfusion syndrome, aortic rupture, and accelerated expansion with persistent pain, surgical treatment has traditionally been indicated.^{2,3} However, early mortality with surgical treatment can reach 30% or exceed 50% in patients with various comorbidities.³ Thoracic endovascular aortic repair (TEVAR) is currently seeing increased use in patients with complicated AAS at the level of the descending thoracic aorta as an alternative to surgery, especially in patients with high comorbidity

or hemodynamic instability.^{4–11} According to the Task Force on Practice Guidelines of the American College of Cardiology Foundation, the American Heart Association, and the European Society of Cardiology, TEVAR, when feasible, should be considered the first-line treatment mainly in complicated acute type B dissection.^{12,13} However, there is limited information on the long-term benefits of TEVAR in patients with complicated AAS as concerns the need for reintervention and late mortality.^{9–11}

Methods

Patient Characteristics

During the period from 2002 to 2014, 432 patients were admitted to our institution with the diagnosis of AAS. Of these cases, 64% were localized at the ascending thoracic

The authors have no funding, financial relationships, or conflicts of interest to disclose.

aorta level ($n = 277$) and 36% at the descending thoracic aorta level ($n = 155$). Of the patients with AAS affecting the descending aorta, 94 met the inclusion criteria of the study; 63 underwent endovascular repair (group 1) and 31 underwent open surgery (group 2). Patients age > 18 years with a diagnosis of AAS located in the descending thoracic aorta distal to the emergence of the left subclavian artery, complicated by rupture, malperfusion syndrome, and/or acute aortic expansion, were included. A diagnosis of AAS and complications was established prior to open surgical or endovascular intervention by computed tomography or magnetic resonance imaging. Patient selection for open repair or endovascular treatment was performed on an individual basis according to the anatomical characteristics of the descending thoracic aorta, comorbidity, and technical feasibility. In most cases we used a multidisciplinary approach (medical, endovascular, and surgical specialties) to choose the most appropriate strategy for the individual patient. Informed consent was obtained from each patient or his or her relatives.

Study Endpoints

The aim of this study was to compare the rate of major adverse cardiovascular events during hospitalization and to analyze the long-term mortality and the need for late reintervention in patients undergoing TEVAR or open surgery. Major adverse cardiovascular events were defined as one of the following: overall and cardiovascular death, myocardial infarction, stroke, paraplegia, conversion to open surgery (TEVAR group), reintervention, and aortic rupture.

Statistical Analysis

Categorical variables are presented as frequencies and percentages and were compared using the Fisher exact test. Continuous variables were described as mean $\pm$ SD, and comparisons between groups were made using the t test. Propensity score matching was performed to avoid potential clinical and demographic confounding factors. The Cox proportional hazards model was used to identify predictors of major adverse events during the study period with univariate and stepwise multivariate analysis. Survival analysis was performed using the Kaplan-Meier method, and survival curves were compared using a log-rank test. Statistically significant differences were considered at $P < 0.05$. Data analysis was performed using SPSS version 20.0 (IBM Corp., Armonk, NY).

Results

Demographics and Clinical Characteristics

The mean age of the patients was 50.9 ± 12.5 years, and the most common risk factor was hypertension (84.0% of the total population). Table 1 shows the baseline characteristics of the patients included in the study. The most common clinical presentation of AAS was aortic dissection (82.9%), and the most common complication was impending aortic rupture at 70.2%. The groups were comparable in demographics and clinical characteristics (tables 1 and 2).

Table 1. Demographic Characteristics

	Endovascular Treatment, $n = 63$	Surgical Treatment, $n = 31$	P Value
Age, y	51.0 ± 13.1	50.8 ± 11.6	0.81
Arterial hypertension	55 (87.3)	24 (77.4)	0.17
DM	4 (6.3)	3 (9.6)	0.42
Hyperlipidemia	17 (26.9)	5 (16.1)	0.18
Smoking	25 (39.6)	13 (41.9)	0.50
Marfan syndrome	11 (17.4)	4 (12.9)	0.40
Prior cardiac surgery	10 (15.8)	2 (6.4)	0.17
Prior aortic surgery	4 (6.3)	0 (0)	0.57
Prior cerebrovascular disease	3 (4.7)	1 (3.2)	0.61
Obesity	2 (19.0)	4 (12.9)	0.48
Abbreviations: DM, diabetes mellitus; SD, standard deviation. Data are presented as n (%) or mean $\pm$ SD.			

Table 2. Clinical Characteristics

	Endovascular Treatment, $n = 63$	Surgical Treatment, $n = 31$	P Value
Aortic pathology			
Penetrating aortic ulcer and intramural hematoma	6 (9.5)	0 (0)	0.08
Intramural hematoma	7 (11.1)	7 (22.5)	0.12
Complicated acute type B dissection	55 (87.3)	23 (74.1)	0.09
Traumatic transection	3 (4.7)	0 (0)	0.38
Complications			
Hemothorax	20 (31.7)	7 (22.5)	0.25
Impending rupture	49 (77.7)	17 (54.8)	0.02
Distal organ malperfusion	21 (33.3)	7 (22.5)	0.20
Aortic diameter > 40 mm	50 (79.3)	31 (100)	0.003
Data are presented as n (%).			

Open Thoracic Aortic Repair

Surgical repair in all patients involved resection of the affected segment with Dacron graft placement. Open repair was performed in all patients with extracorporeal perfusion support with a mean aortic clamp time of 90.5 ± 13.2 minutes. Distal aortic perfusion with partial left-heart bypass was utilized to prevent visceral and spinal-cord injuries. In 3 patients an extended procedure was performed with partial arch replacement. In this setting a hypothermic circulatory arrest with retrograde cerebral perfusion was used. Aortic diameter was 57.2 ± 22.5 mm, and a diameter > 40 mm was recorded in all patients.

Figure 1. Complicated acute thoracic aortic syndrome: aortic rupture.

Endovascular Thoracic Aortic Repair

Endovascular repair was performed in a catheterization laboratory under general anesthesia and with surgical dissection of the common femoral artery. The average diameter of the aorta in this group was 56.9 ± 22.3 mm. The length of the stent was 184.2 ± 38.4 mm (120–300 mm); its mean diameter was 36.9 ± 4.9 mm. In total, 77.7% of the patients received a single stent, 14.2% received 2 stents, and 7.9% had 3 stents. A total of 82 stents were implanted in 63 patients (1.3 stents per patient). Forty-three stents (52.4%) were Zenith (William Cook Europe, Bjaeverskov, Denmark); 6 (7.3%) were Valiant (Medtronic Vascular, Inc., Santa Rosa, CA); 2 (2.4%) were Relay (Bolton Medical, Barcelona, Spain); and 31 (37.8%) were Talent (Medtronic; Figures 1 and 2).

Early Outcomes

The average hospital stay in group 1 was 7.2 ± 4.3 days and in group 2 it was 19.5 ± 5.6 days. In the total population, 5.3% of patients exhibited clinical evidence of paraplegia: 1.5% in the endovascular group and 12.9% in the surgical group ($P = 0.039$). Early cardiovascular mortality in the total population was 20.2%, being lower in the group undergoing endovascular repair (6.3% vs 48.3%; $P = 0.0001$). Four patients died in the endovascular group: 1 patient with persistent pain and refractory hypertension after endovascular repair died on the fifth day during surgical repair (surgical conversion), 2 other patients died from malperfusion syndrome with mesenteric ischemia, and another died from aortic rupture when removing the affixed pleural seal to drain the hemothorax, 4 days after TEVAR.

Fifteen patients died early in the surgical group: 5 in the operating room and 10 in the intensive care unit. Of the patients who died in the operating room, 2 were due to retrograde dissection and 3 to aortic rupture. Of the patients who died in intensive care, 2 deaths were caused

Figure 2. Thoracic endovascular aortic repair.

by an ischemic cerebral vascular event, 3 by cardiogenic shock, and 5 by multiple organ failure. Eighty percent of these deaths in the open-repair group occurred during the first years of the study (from 2002 to 2008). We observed a remarkable reduction in early mortality in our patients in this group during the last period of the study (18.7%, post-2008).

Malperfusion syndrome (odds ratio: 4.6, 95% confidence interval: 1.1–19.2, $P = 0.037$) was one independent predictor of early cardiovascular mortality.

Late Outcomes

Long-term follow-up was 63.0 ± 47.5 months, with aorta-related mortality in the total population at 21.2%. In the surgical group, no additional deaths occurred during the follow-up period. In group 1, 2 additional deaths were documented. One patient with Marfan syndrome died from aortic rupture before reintervention for proximal type I endoleak, and another patient died from laryngeal cancer. Late mortality related to the aorta was 1.6% with TEVAR and 0% with surgical treatment. The rate of late reintervention in the endovascular group was 11.8% ($n = 7/59$). In 4 patients, proximal type I endoleak was documented, which was corrected with proximal stent implantation; in another patient with type II endoleak from retrograde filling through the subclavian artery, vascular closure with an occluder system was performed (Amplatzer Vascular Plug II; AGA Medical Corp., Plymouth, MN); and in 2 additional patients, a distal stent-graft was

Figure 3. Long-term survival free of death related to the aorta (log rank $P = 0.0001$). Abbreviations: TEVAR, thoracic endovascular aortic repair.

implanted for extension of the dissection in one case and malperfusion syndrome in the other. In the surgical group, 3 patients underwent reintervention ($n = 3/16$) by spinal cord ischemia, pleuropulmonary fistula, and tamponade.

The cumulative cardiovascular death-free survival rates at 5 years were 92.0% with TEVAR and 51.4% with surgery (log rank $P = 0.0001$). The cumulative aorta-related death-free survival rates at 5 years for group 1 and 2 were 93.7% and 57.5% (log rank $P = 0.0001$), respectively (Figure 3). Long-term survival freedom from reintervention was comparable with TEVAR and surgery (88.1% vs 81.2%; log rank $P = 0.44$) in our study.

Discussion

Our results indicate that TEVAR in patients with complicated AAS that involves the descending thoracic aorta is effective and safe compared with surgical treatment. The main findings of this study are that cardiovascular mortality was lower with endovascular treatment during early outcomes, and survival free from aorta-related death during the follow-up period (63 months) was comparable with endovascular treatment and with surgery.

Initial experiences with endovascular repair in patients with AAS, mainly patients with type B acute aortic dissection, were published in 1999.^{4,5} According to the guidelines for the diagnosis and treatment of aortic diseases, and interdisciplinary expert consensus, TEVAR is recommendation class I.^{12–14} In patients with complicated type B acute aortic dissection, early mortality with

endovascular treatment up to 17% has been reported.^{15–20} A recently published meta-analysis reported a mortality of 7.3% at 30 days with TEVAR and 19.0% with surgery.²¹ In comparative studies, mortality has been reported between 8% and 11.4% with endovascular repair and between 26.5% and 29% with surgical treatment.^{8,9} In our study, early mortality with TEVAR was 6.3%, and the main cause of in-hospital mortality was malperfusion syndrome (mesenteric ischemia), increasing the risk of early death by 4.6 $\times$. Ryan et al reported a 30-day mortality of 21.3% in patients with malperfusion syndrome, and ischemic territory was not independently associated with mortality.²² Other authors have reported 10.8% mortality.²⁰ Early mortality in our patients with malperfusion syndrome undergoing endovascular repair was 9.5%. We observed a significant improvement in the early mortality in our group undergoing open surgery after the year 2008. This is probably because the learning curve had already been reached.

The rate of neurologic events (spinal cord ischemia and cerebrovascular events) in our population (3%) is comparable with the rates reported in other studies (7.3%–9.4%).²¹

There is very limited information on the long-term benefits of endovascular repair in patients with acute thoracic aortic syndromes. Late mortality related to the aorta and the need for reintervention are 2 important factors for assessing the long-term benefits of TEVAR. Late mortality (at 5 years) related to the aorta has been reported at 8.5% to 10% with TEVAR, with untreated endoleak being the leading cause of death.^{7,8,10,11}

Wilkinson et al reported reintervention and aortic rupture death-free survival rates at 5 years of 82.8% with surgery and 78.0% with endovascular treatment repair for complicated type B acute aortic dissection.¹⁸ A recent study documented 84% overall survival and 100% aorta-related death-free survival rate at 5 years with TEVAR.²³

In our study, cumulative survival free from cardiovascular death was 92.0% with TEVAR, compared with 51.4% in the open-repair group. However, after excluding in-hospital deaths in the long-term analysis, the cardiovascular death-free survival rates at 5 years were 94.3% and 100% with TEVAR and surgery, respectively. The cumulative aorta-related death-free long-term survival rate was 57.5% in the open-surgery group and 93.7% in the endovascular group.

A rate of late reintervention has been reported from 8.9% to 26% with TEVAR, where type 1 endoleak is the main cause of reintervention.^{7,8,10,11} In the surgical group, a reintervention rate of 11.7% has been reported, due to graft infection and disease progression (aneurysm).⁹ In our study, type 1 endoleak was the most common cause for reintervention ($n = 5/7$) in patients undergoing TEVAR, with a late reintervention rate of 11.4% at 5 years. Survival freedom from late reintervention was 88.1% in the TEVAR group compared with 81.2% in the open-surgery group ($P = 0.44$), similar to the results reported by Hanna et al.²³

Conclusion

Thoracic endovascular aortic repair in patients with complicated AAS at the descending thoracic aorta is feasible and safe, with a lower rate of early complication rates compared with surgery. Long-term benefits regarding cardiovascular mortality were comparable with both treatments. The main cause of reintervention in the long term in patients who underwent TEVAR is the presence of endoleak.

References

1. Vilacosta I, San Román JA. Acute aortic syndrome. *Heart*. 2001;85:365–368.
2. Nienaber CA, Powell JT. Management of acute aortic syndromes. *Eur Heart J*. 2012;33:26b–35b.
3. Trimarchi S, Nienaber CA, Rampoldi V, 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–I364.
4. Nienaber CA, Fattori R, Lund G, et al. Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement. *N Engl J Med*. 1999;340:1539–1545.
5. Dake MD, Kato N, Mitchell RS, et al. Endovascular stent-graft placement for the treatment of acute aortic dissection. *N Engl J Med*. 1999;340:1546–1552.
6. Cambria R, Crawford RS, Cho JS, et al. A multicenter clinical trial of endovascular stent graft repair of acute catastrophes of the descending thoracic aorta. *J Vasc Surg*. 2009;50:1255–1264.
7. Kaya A, Heijmen R, Rousseau H, et al. Emergency treatment of the thoracic aorta: results in 113 consecutive acute patients (the Talent Thoracic Retrospective Registry). *Eur J Cardiothorac Surg*. 2009;35:276–281.
8. Patel HJ, Williams DM, Upchurch GR Jr, et al. A comparative analysis of open and endovascular repair for the ruptured descending thoracic aorta. *J Vasc Surg*. 2009;50:1265–1270.
9. Naughton PA, Park MS, Morasch MD, et al. Emergent repair of acute thoracic aortic catastrophes: a comparative analysis. *Arch Surg*. 2012;147:243–249.
10. Caronno R, Piffaretti G, Tozzi M. Emergency endovascular stent-graft treatment for acute thoracic aortic syndromes. *Surgery*. 2006;140:58–65.
11. Pitton MB, Herber S, Schmiedt W, et al. Long-term follow-up after endovascular treatment of acute aortic emergencies. *Cardiovasc Intervent Radiol*. 2008;31:23–35.
12. Hiratzka LF, Bakris GL, Beckman JA, et al. 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with thoracic aortic disease: executive summary. *Circulation*. 2010;121:1544–1579.
13. Erbel R, Aboyans V, Boileau C, et al; The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). 2014 ESC Guidelines on the diagnosis and treatment of aortic diseases: document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. *Eur Heart J*. 2014;35:2873–2926.
14. Fattori R, Cao P, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. *J Am Coll Cardiol*. 2013;61:1661–1678.
15. Song JK. Update in acute aortic syndrome: intramural hematoma and incomplete dissection as new disease entities. *J Cardiol*. 2014;64:153–161.
16. White RA, Miller DC, Criado FJ, et al. Report on the results of thoracic endovascular aortic repair for acute, complicated, type B aortic dissection at 30 days and 1 year from a multidisciplinary subcommittee of the Society for Vascular Surgery Outcomes Committee. *J Vasc Surg*. 2011;53:1082–1090.
17. Nienaber CA, Divchev D, Palisch H, et al. Early and late management of type B aortic dissection. *Heart*. 2014;100:1491–1497.
18. Wilkinson DA, Patel HJ, Williams DM, et al. Early open and endovascular thoracic aortic repair for complicated type B aortic dissection. *Ann Thorac Surg*. 2013;96:23–30.
19. Ehrlich M, Rousseau H, Heijmen R, et al. Midterm results after endovascular treatment of acute, complicated type B aortic dissection: the Talent Thoracic Registry. *J Thorac Cardiovasc Surg*. 2013;145:159–165.
20. Fattori R, Tsai TT, Myrmet T, et al. Complicated acute type B dissection: Is surgery still the best option? A report from the International Registry of Acute Aortic Dissection. *JACC Cardiovasc Interv*. 2008;1:395–402.
21. Moulakakis KG, Mylonas SN, Dalainas I, et al. Management of complicated and uncomplicated acute type B dissection. A systematic review and meta-analysis. *Ann Cardiothorac Surg*. 2014;3:234–246.
22. Ryan C, Vargas L, Mastracci T, et al. Progress in management of malperfusion syndrome from type B dissections. *J Vasc Surg*. 2013;57:1283–1290.
23. Hanna JM, Andersen ND, Ganapathi AM, et al. Five-year results for endovascular repair of acute complicated type B aortic dissection. *J Vasc Surg*. 2014;59:96–106.