

Endovascular Repair in Acute Complicated Type B Aortic Dissection: 3-Year Results from the Valiant US Investigational Device Exemption Study

Chang Young Lim, M.D.

Department of Thoracic and Cardiovascular Surgery, Andong General Hospital

Acute complicated type B aortic dissection (TBAD) is a potentially catastrophic, life-threatening condition. If left untreated, there is a high risk of aortic rupture, irreversible organ or limb damage, or death. Several risk factors have been associated with acute complicated TBAD, including age and refractory hypertension. In the acute phase, even uncomplicated patients are more prone to develop complications if hypertension and pain are left medically untreated. Innovations in stent graft technologies have incrementally improved outcomes since their first use for this condition in 1999, though improvement is needed in mitigating periprocedural complications, adverse events, and mortality. In the past decade, endovascular repair has become the preferred treatment because of its superior outcomes to open repair and medical therapy. The Valiant Captivia Thoracic Stent Graft System is a third-generation endovascular stent graft with advancements in minimally invasive delivery, conformability to the anatomy, and the minimization of adverse sequelae. Herein, this stent graft is briefly reviewed and its 3-year outcomes are presented. Freedom from all-cause and dissection-related mortality was 79.1% and 90.0%, respectively. The Valiant Captivia Stent Graft represents a safe, effective intervention for acute complicated TBAD. Continued surveillance is needed to verify its longer-term durability.

Key words: 1. Self expandable metallic stents
2. Aortic aneurysm, thoracic
3. Aortic dissection
4. Endovascular procedures
5. Valiant captivia

Introduction

1) *The challenge of acute complicated type B aortic dissections*

Acute aortic dissection is the most common type of potentially catastrophic aortic condition, and, if untreated, poses a high risk of early mortality. The widely adopted Stanford classification distinguishes type B aortic dissections (TBADs), which involve the

descending aorta distal from the left subclavian artery (LSA), from type A dissections, which involve the aortic arch. TBADs may further be classified as complicated or uncomplicated based on their distinctive patterns of clinical presentation, and the prognosis may differ significantly. In-hospital mortality has been reported to be roughly 50% for complicated TBAD patients and 10% for uncomplicated TBAD patients [1-5]. Roughly one-quarter of all acute TBADs

Received: April 20, 2017, Revised: May 5, 2017, Accepted: May 11, 2017, Published online: June 5, 2017

*Corresponding author: Chang Young Lim, Department of Thoracic and Cardiovascular Surgery, Andong General Hospital, 11 Angsil-ro, Andong 36743, Korea
(Tel) 82-54-840-0248 (Fax) 82-54-840-1515 (E-mail) cylimmd@gmail.com*

© The Korean Society for Thoracic and Cardiovascular Surgery. 2017. All right reserved.

© This is an open access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/4.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

may be considered complicated if there are signs of imminent rupture, any sign of organ or limb malperfusion, the presence of refractory hypertension, hypotension (<90 mm Hg systolic), or cardiogenic shock.

Acute complicated TBADs are challenging because of the formidable risk of organ or limb malperfusion, and/or the rapid expansion of the thoracic aorta, which may lead to rupture (a diameter of >4.5 cm or 5 cm). A timely intervention is critical for survival. Malperfusion is caused by the static or dynamic compression of the true lumen by the false lumen, which must be successfully managed to restore end-organ perfusion and avoid irreversible end-organ or limb damage [6]. Partial false lumen thrombosis independently predicts aortic growth and follow-up mortality in acute TBAD [7,8]. In 2007, Song et al. [9] reported that upper false lumen diameters ≥ 22 mm predicted late mortality (up to 9 years) versus diameters <22 mm ($p < 0.001$).

Compared to patients with chronic expanding TBADs, patients with acute complicated TBADs are known to have poorer outcomes because of their presentation and aggressive course. In 2006, Bockler et al. [10] reported a 30-day mortality rate of 19% in patients with acute complicated TBADs versus 0% in patients with chronic TBADs. Systemic hypertension has been reported as one of the most important risk factors for acute TBAD, present in about 80% of patients [1], as well as increasing age, atherosclerosis, and taller stature [11-14].

2) Interventions for acute complicated type B aortic dissection

The mainstay life-saving treatments for acute complicated TBAD are open surgical repair (OSR), which was first performed in the 1950s, and thoracic endovascular aortic repair (TEVAR), first undertaken in 1999 by both Dake et al. [15] (a series of 19 acute complicated TBADs with early death occurring in 16% of the patients) and Nienaber et al. [16] (a case-control study of 24 subacute or chronic TBAD patients, 12 of whom underwent OSR, with a 33% mortality rate, and 12 of whom underwent TEVAR, with no early deaths). Medical therapy is typically a less aggressive modality reserved for uncomplicated acute, subacute, and chronic TBAD cases. However, medical therapy was employed for many patients before the debut of endovascular interventions. Currently,

only a minority of patients with acute complicated TBAD are treated by medical therapy alone, if the use of either TEVAR and OSR is ruled out because of contraindicating patient comorbidities, anatomical considerations, or a lack of appropriate facilities, equipment, or expertise [17]. OSR and TEVAR each have their own rates of morbidity, treatment related complications, and mortality [15,18]. Although OSR is preferred for type A aortic dissections, endovascular repair has become the preferred first-line treatment for acute complicated TBADs and is increasingly employed for the distal arch, with OSR recommended to be reserved for patients unsuitable for endovascular repair [19].

Endovascular repair is now a standard therapy for acute complicated TBAD [20]. Fattori et al. [21,22] reported that TEVAR mortality rates when used to treat acute complicated TBADs are now similar to the mortality rates when TEVAR is used to treat uncomplicated TBADs, particularly with advancements in endovascular technologies. TEVAR offers a nearly a 4-fold increase in early survival for patients with acute complicated TBADs, which may be attributable to its myriad advantages [23]. TEVAR is less invasive than OSR. It is also a straightforward intervention to train for and perform, particularly with the availability of newer generation thoracic devices. The periprocedural advantages of TEVAR include a shorter procedure time, reduced blood loss, and faster patient recovery [24]. The technical success rates of TEVAR are high for both acute TBAD (ranging from 93.3% to 100.0%) [25-27] as well as for chronic TBAD (ranging from 77.6% to 100.0%) [28-30]. In addition, TEVAR for acute TBAD is surmised to promote aortic remodeling and have a prophylactic effect against late complications [31-33]. Although TEVAR is susceptible to periprocedural complications such as endoleak, new entry tears, and migration, technical success is commonly achieved.

3) Thoracic endovascular aortic repair versus open surgical repair versus medical therapy

Several studies have shown TEVAR survival outcomes to be superior to OSR in the short- to mid-term time horizon [34-36]. Notable studies include a 2010 comparative analysis by Zeeshan et al. [37] at the University of Pennsylvania. In-hospital mortality was significantly different between groups, with mor-

tality rates of 4% for TEVAR, 40% for OSR, and 33% for medical therapy ($p=0.006$). For TEVAR patients, survival was reported at years 1, 3, and 5 to be 82%, 79%, and 79%, whereas the survival in patients receiving OSR or medical therapy (grouped together) was 58%, 52%, and 44% ($p=0.008$) at the same times [37]. These findings were confirmed in a 2014 comparative decision model study by Hogendoorn et al. [19], which concluded that TEVAR showed greater total effectiveness than OSR for all age groups.

In 2013, Fattori et al. [22] reported a propensity-matched analysis of the International Registry of Acute Aortic Dissection database ($N=1,129$ patients). Notably, this study revealed historical differences between TEVAR and medical therapy from 1995 to 2015; that is, from before the advent of TEVAR for acute TBADs, during the rising trend of TEVAR adoption in the 2000s, all the way to contemporary practice. TEVAR plus medical therapy ($n=276$, 25.2%) was compared to medical therapy alone ($n=853$, 74.8%) for both acute complicated and acute uncomplicated TBADs. Several baseline characteristics were significantly different between groups. It is interesting to note that TEVAR-treated TBADs were significantly more likely than medically managed dissections to be considered complicated (61.7% versus 37.2%, respectively; $p<0.001$), which is understandable given the scope of the time period and the seesaw-like shift in the usage of these respective interventions. Further differences between TEVAR and medical therapy can be seen in in specific complication categories, including malperfusion (21.7% versus 8.4%, $p<0.001$), significantly worse reported pain (27.5% versus 15.7%, $p<0.001$), acute renal failure prior to treatment (21.4% versus 12.4%, $p<0.001$) and any pulse deficit on presentation (28.3% versus 13.4%, $p<0.001$), all of which were more prevalent in the interventional group. In-hospital mortality was statistically similar (10.9% versus 8.7%, $p=0.273$) despite the TEVAR group presenting with more complications. However, the cumulative probability of mortality at 5 years was significantly lower in the TEVAR group than in the group of patients who underwent medical therapy alone (29.0% versus 15.5%, $p=0.018$) [22].

Longer-term outcomes for TEVAR and OSR for acute complicated TBADs, however, are sparse and remain of great interest. Current long-term survival

data suggest that no significant differences are present between TEVAR and OSR, which was recently confirmed in a 2016 meta analysis of 8 studies, in addition to a study conducted in a Chinese population of 118 inpatients ($n=45$ in the OSR group and $n=73$ in the TEVAR group) enrolled from January 2004 to January 2015. They concluded that there was no significant difference in the 10-year survival between OSR and TEVAR (56.7% and 26.1%, respectively; log-rank $p=0.953$) [38].

Only in recent years has oversizing been scrutinized and debated in terms of its relationship with severe complications over time, including stent-graft-induced new entry (SINE) tears, retrograde type A dissection, and proximal neck dilatation [17,39]. Excessive distal oversizing has been reported to be an independent predictor of SINE events, which usually require reintervention. It is believed that SINE events occur because of the fragility of the dissected intimal membrane in acute dissection as opposed to chronic dissection [39,40], although SINE events have been reported to be more common in chronic dissection [41]. However, a 2016 study of type B dissection cases reported that a distal-first rather than proximal-first deployment approach resulted in significantly fewer SINE events [42]. A better understanding of the long-term durability of TEVAR is needed to appropriately advance the technology and practice to achieve better durability and outcomes.

The US DISSECTION trial 3-year results of Valiant Captivia for acute complicated type B aortic dissection

The DISSECTION trial is an ongoing prospective, nonrandomized, multicenter evaluation of 50 patients (80% male) with acute complicated TBAD treated with the Valiant Captivia Thoracic Stent Graft System (Medtronic, Santa Rosa, CA, USA). Bavaria et al. [43] have described the design and methods of the trial and reported the 30-day and 1-year results. The study has continued with plans for a 5-year follow-up. Valiant Captivia received US Food and Drug Administration (FDA) investigational device exemption (IDE) in January 2014 and DISSECTION trial enrollment began in 16 centers in the United States.

1) The Valiant Captivia Thoracic Stent Graft System

Valiant Captivia is a third-generation stent graft system with approved indications for use in Europe and US for thoracic aortic aneurysms, transections, and dissections. As the successor to the Talent Thoracic Stent Graft System (Medtronic), Valiant has been evaluated in a number of critical research studies specifically aimed at elucidating its feasibility, safety, and effectiveness in the thoracic aorta, all with satisfactory results: VALOR II (US IDE for descending thoracic aneurysms, n=160) [44,45], VIRTUE (European registry for all types of TBADs, n=100) [46-48], the VALIANT CAPTIVIA registry (mid- to high-risk all-comer cohort, n=100) [28], the RESCUE trial (blunt thoracic aortic injury, n=50) [49,50], and the Valiant Mona LSA first-in-human feasibility trial using a modified Valiant Captivia System with branching to allow LSA patency (US FDA IDE via the new Innovation Pathway, n=9) [51]. The VIRTUE registry was a prospective cohort study comparing 3 groups of TBADs: complicated acute (n=50), subacute (n=24), and chronic (n=26). All-cause mortality at 3 years was 18%, 4%, and 24%, respectively, and dissection-related mortality was 12%, 4%, and 9%, respectively [46].

The DISSECTION trial included patients diagnosed with an acute complicated TBAD who were treated with a stent graft within 14 days from presentation. The included patients had malperfusion (visceral, renal, spinal cord, and/or lower limb ischemia) or rupture; patients were excluded if they had a history of a connective tissue disorder. The Valiant Captivia Thoracic Stent Graft System consists of a modular, self-expanding endograft preloaded into the Captivia Delivery System. The endograft size matrix was expanded in 2014, significantly broadening the size and configuration choices to address a wider range of anatomical variants. The proximal end of the endograft is composed of its bare-spring 8-Peak FreeFlo configuration, which was designed and bench-tested to successfully distribute force radially across its multiple apices. The distal end consists of either a closed web configuration devoid of bare springs or an 8-peak bare-spring configuration at the distal end.

2) Baseline characteristics and index procedure

A total of 50 patients (80% males) were enrolled; 62% were Caucasian, 12% Asian, 22% black, and 4%

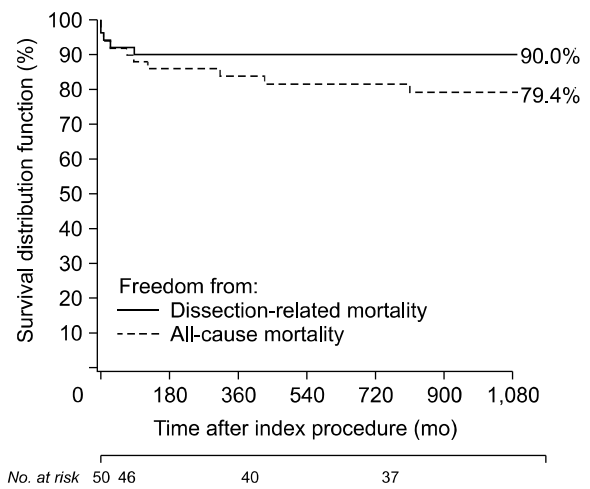


Fig. 1. All-cause mortality and dissection-related mortality through 3 years.

other. Their mean age was 57.2 ± 12.9 years. The vast majority of patients had a medical history of hypertension (90%), while relatively few had a history of myocardial infarction (6.0%) or congestive heart failure (8.0%). There were a variety of patient presentations, with 1 in 5 patients presenting with an impending rupture (20%), and fewer with paraparesis (12%). Abdominal pain at presentation was present in 36% of patients. The mean time from the onset of symptoms to the index procedure was 4.7 days.

The majority of patients complained of back or chest pain (88%). Malperfusion was diagnosed in 86% of patients, of whom visceral ischemia was present in 40%, renal ischemia in 42%, lower limb ischemia in 40%, and spinal cord ischemia in 6%. Medical therapy was administered to 84% of patients (antihypertensives), and 16% required inotropic support. At the baseline index procedure, the primary entry tear was covered in all dissections, and all cases achieved 100% delivery and deployment success with no misaligned deployments. Device oversizing was $12.0\% \pm 10.3\%$ and the length of coverage was 196.9 ± 67.1 mm. Through year 3 post-index, a total of 28 of the 32 eligible patients (87.5%) have reportedly completed their follow-up appointment. Since the previous report by Bavaria et al. [43], there have been no post-index ruptures or conversions to OSR.

3) Mortality through 3 years

All-cause mortality was 8% in the first 30 days and 14.6% (7 of 48) in the first year. Throughout

Table 1. Aortic remodeling through 3 years

Stented segment	Months			
	6	12	24	36
False lumen maximum diameter (decreased or stable) ^{a)}	75.8 (25/33)	82.4 (28/34)	78.6 (22/28)	69.2 (18/26)
True lumen maximum diameter (increased or stable) ^{a)}	93.9 (31/33)	94.1 (32/34)	85.7 (24/28)	92.3 (24/26)
False lumen thrombosis (partial or complete)	75.8 (25/33)	79.4 (27/34)	70.4 (19/27)	75.0 (18/24)

Values are presented as % (number).

^{a)}Diameter change of ≥ 5 mm from the first post-procedural imaging.

the 3 years of the study experience, of the evaluable patients, 10 have died, 8 of whom expired within the first year. Of those 10 deaths, 5 were considered dissection-related. Freedom from all-cause mortality (79.4%) and dissection-related mortality (90%) is shown in Fig. 1. No further dissection-related deaths were reported through years 2 and 3. A total of 4 dissection related deaths were reported in the first 30 days. Per protocol, any death within first 30 days must be classified as dissection-related (cardiac tamponade [day 0], mesenteric ischemia in totalis [day 1], sepsis [day 9], and pulmonary embolism [day 26]). Only 1 death was adjudicated to be dissection related within the first year (day 87); this patient's official cause of death was pneumonia, as reported in the early results [43]. Other deaths after 30 days and within the first year were cardiac arrest (days 71 and 124) and respiratory failure (day 315). Within the second year post-procedure, 1 death from natural causes was reported (day 432). In the third year, 1 additional death was reported, attributable to sepsis (day 812).

4) Adverse events, reinterventions, and aortic remodeling through 3 years

Within the first 30 days, there were 3 cerebrovascular accidents (6%), 1 report of chronic venous insufficiency (2% [1 of 50]), and 1 retrograde type A dissection (2.2% [1 of 46]). After 30 days and within the first year, 1 additional retrograde dissection was reported, as well as 1 case of acute renal failure. In years 2 and 3, however, there were no further serious adverse events. Notably, there were no post-operative ruptures throughout the entire study period. Four reinterventions were performed after the first year, one of which was an LSA plug and not deemed dissection-related; the other 3 reinterventions were extension endografts. There were no conversions to

OSR. Favorable remodeling was observed over stented segments at 12 months and through 3 years (Table 1).

For patients with acute complicated TBAD, the Valiant Captivia Thoracic Stent Graft System demonstrated acceptable rates of adverse events and positive outcomes through 3 years. There were no ruptures or conversions to OSR. Freedom from all-cause mortality was 79.4%, and freedom from dissection-related mortality was 90.0%. Only 1 dissection-related death occurred more than 30 days after the index procedure. There was a very low incidence of endovascular reinterventions, and favorable aortic remodeling was observed over the stented segment.

Conclusion

Acute complicated TBADs are the most common emergency aortic syndrome and pose a formidable treatment challenge. The 3-year midterm results of the Medtronic DISSECTION trial continue to demonstrate the safety and effectiveness of the Valiant Captivia Thoracic Stent Graft System for patients with acute complicated TBAD. The follow-up through 3 years demonstrates acceptable mortality given the serious risks that accompany acute complicated TBADs and the treatment thereof. Annual follow-up is planned for up to 5 years. Longer-term results are anticipated to be forthcoming to determine if this trend of durability can be sustained.

Conflict of interest

No potential conflicts of interest relevant to this article are reported.

Acknowledgments

The author would like to thank Dr Alessandro Marucchini for his assistance with data collection. On behalf of the Valor II Dissection Investigators and author are welcome to acknowledge Medtronic for medical editing.

References

- Hagan PG, Nienaber CA, Isselbacher EM, et al. *The International Registry of Acute Aortic Dissection (IRAD): new insights into an old disease*. JAMA 2000;283:897-903.
- Sakakura K, Kubo N, Ako J, et al. *Determinants of in-hospital death and rupture in patients with a Stanford B aortic dissection*. Circ J 2007;71:1521-4.
- Trimarchi S, Tolenaar JL, Tsai TT, et al. *Influence of clinical presentation on the outcome of acute B aortic dissection: evidences from IRAD*. J Cardiovasc Surg (Torino) 2012;53:161-8.
- Trimarchi S, Nienaber CA, Rampoldi V, et al. *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.
- Estrera AL, Miller CC, Goodrick J, et al. *Update on outcomes of acute type B aortic dissection*. Ann Thorac Surg 2007;83:S842-5.
- Uchida N, Shibamura H, Katayama A, Aishin K, Sutoh M, Kuraoka M. *Surgical strategies for organ malperfusions in acute type B aortic dissection*. Interact Cardiovasc Thorac Surg 2009;8:75-8.
- Trimarchi S, Tolenaar JL, Jonker FH, et al. *Importance of false lumen thrombosis in type B aortic dissection prognosis*. J Thorac Cardiovasc Surg 2013;145(3 Suppl):S208-12.
- Tsai TT, Evangelista A, Nienaber CA, et al. *Partial thrombosis of the false lumen in patients with acute type B aortic dissection*. N Engl J Med 2007;357:349-59.
- Song JM, Kim SD, Kim JH, et al. *Long-term predictors of descending aorta aneurysmal change in patients with aortic dissection*. J Am Coll Cardiol 2007;50:799-804.
- Bockler D, Schumacher H, Ganten M, et al. *Complications after endovascular repair of acute symptomatic and chronic expanding Stanford type B aortic dissections*. J Thorac Cardiovasc Surg 2006;132:361-8.
- Jonker FH, Trimarchi S, Muhs BE, et al. *The role of age in complicated acute type B aortic dissection*. Ann Thorac Surg 2013;96:2129-34.
- Harris KM, Braverman AC, Eagle KA, et al. *Acute aortic intramural hematoma: an analysis from the International Registry of Acute Aortic Dissection*. Circulation 2012;126(11 Suppl 1):S91-6.
- Collins JS, Evangelista A, Nienaber CA, et al. *Differences in clinical presentation, management, and outcomes of acute type a aortic dissection in patients with and without previous cardiac surgery*. Circulation 2004;110(11 Suppl 1):II237-42.
- Charilaou P, Ziganshin BA, Peterss S, et al. *Current experience with acute type B aortic dissection: validity of the complication-specific approach in the present era*. Ann Thorac Surg 2016;101:936-43.
- Dake MD, Kato N, Mitchell RS, et al. *Endovascular stent-graft placement for the treatment of acute aortic dissection*. N Engl J Med 1999;340:1546-52.
- Nienaber CA, Fattori R, Lund G, et al. *Nonsurgical reconstruction of thoracic aortic dissection by stent-graft placement*. N Engl J Med 1999;340:1539-45.
- 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;61:1661-78.
- Miller DC, Mitchell RS, Oyer PE, Stinson EB, Jamieson SW, Shumway NE. *Independent determinants of operative mortality for patients with aortic dissections*. Circulation 1984;70(3 Pt 2):I153-64.
- Hogendoorn W, Hunink MG, Schlosser FJ, Moll FL, Sumpio BE, Muhs BE. *Endovascular vs. open repair of complicated acute type B aortic dissections*. J Endovasc Ther 2014;21:503-14.
- Ehrlich MP, 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;145:159-65.
- Fattori R, Tsai TT, Myrmel 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.
- Fattori R, Montgomery D, Lovato L, et al. *Survival after endovascular therapy in patients with type B aortic dissection: a report from the International Registry of Acute Aortic Dissection (IRAD)*. JACC Cardiovasc Interv 2013;6:876-82.
- Nauta FJ, Trimarchi S, Kamman AV, et al. *Update in the management of type B aortic dissection*. Vasc Med 2016;21:251-63.
- 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;89:97-102.
- Shu C, He H, Li QM, Li M, Jiang XH, Luo MY. *Endovascular repair of complicated acute type-B aortic dissection with stentgraft: early and mid-term results*. Eur J Vasc Endovasc Surg 2011;42:448-53.
- Pearce BJ, Passman MA, Patterson MA, et al. *Early outcomes of thoracic endovascular stent-graft repair for acute complicated type B dissection using the Gore TAG endoprosthesis*. Ann Vasc Surg 2008;22:742-9.
- Chemelli-Steingruber I, Chemelli A, Strasak A, et al. *Endovascular repair or medical treatment of acute type B*

- aortic dissection?: a comparison. *Eur J Radiol* 2010;73:175-80.
28. Heijmen RH, Thompson MM, Fattori R, Goktay Y, Teebken OE, Orend KH. *Valiant thoracic stent-graft deployed with the new captivia delivery system: procedural and 30-day results of the Valiant Captivia Registry*. *J Endovasc Ther* 2012;19:213-25.
 29. Ruan ZB, Zhu L, Yin YG, Chen GC. *Risk factors of early and late mortality after thoracic endovascular aortic repair for complicated Stanford B acute aortic dissection*. *J Card Surg* 2014;29:501-6.
 30. Sayer D, Bratby M, Brooks M, Loftus I, Morgan R, Thompson M. *Aortic morphology following endovascular repair of acute and chronic type B aortic dissection: implications for management*. *Eur J Vasc Endovasc Surg* 2008;36:522-9.
 31. Nienaber CA, Clough RE. *Management of acute aortic dissection*. *Lancet* 2015;385:800-11.
 32. Conrad MF, Carvalho S, Ergul E, et al. *Late aortic remodeling persists in the stented segment after endovascular repair of acute complicated type B aortic dissection*. *J Vasc Surg* 2015;62:600-5.
 33. Singh M, Hager E, Avgerinos E, Genovese E, Mapara K, Makaroun M. *Choosing the correct treatment for acute aortic type B dissection*. *J Cardiovasc Surg (Torino)* 2015;56:217-29.
 34. Minami T, Imoto K, Uchida K, et al. *Clinical outcomes of emergency surgery for acute type B aortic dissection with rupture*. *Eur J Cardiothorac Surg* 2013;44:360-4.
 35. Kokotsakis J, Kaskarelis I, Misthos P, et al. *Endovascular versus open repair for blunt thoracic aortic injury: short-term results*. *Ann Thorac Surg* 2007;84:1965-70.
 36. Buz S, Zipfel B, Mulahasanovic S, Pasic M, Weng Y, Hetzer R. *Conventional surgical repair and endovascular treatment of acute traumatic aortic rupture*. *Eur J Cardiothorac Surg* 2008;33:143-9.
 37. Zeeshan A, Woo EY, Bavaria JE, et al. *Thoracic endovascular aortic repair for acute complicated type B aortic dissection: superiority relative to conventional open surgical and medical therapy*. *J Thorac Cardiovasc Surg* 2010;140(6 Suppl):S109-15.
 38. Zhu Y, Wang B, Meng Q, Liu J, Zhai S, He J. *Long-term efficacy of endovascular vs open surgical repair for complicated type-B aortic dissection: a single-center retrospective study and meta-analysis*. *Braz J Med Biol Res* 2016;49:e5194.
 39. Dong Z, Fu W, Wang Y, et al. *Stent graft-induced new entry after endovascular repair for Stanford type B aortic dissection*. *J Vasc Surg* 2010;52:1450-7.
 40. Weng SH, Weng CF, Chen WY, et al. *Reintervention for distal stent graft-induced new entry after endovascular repair with a stainless steel-based device in aortic dissection*. *J Vasc Surg* 2013;57:64-71.
 41. Jang H, Kim MD, Kim GM, et al. *Risk factors for stent graft-induced new entry after thoracic endovascular aortic repair for Stanford type B aortic dissection*. *J Vasc Surg* 2017;65:676-85.
 42. Chen IM, Huang CY, Weng SH, et al. *Implantation sequence modification averts distal stent graft-induced new entry after endovascular repair of Stanford type B aortic dissection*. *J Vasc Surg* 2016;64:281-8.
 43. Bavaria JE, Brinkman WT, Hughes GC, et al. *Outcomes of thoracic endovascular aortic repair in acute type B aortic dissection: results from the Valiant United States Investigational Device Exemption Study*. *Ann Thorac Surg* 2015;100:802-8.
 44. Fairman RM, Tucheck JM, Lee WA, et al. *Pivotal results for the Medtronic Valiant Thoracic Stent Graft System in the VALOR II trial*. *J Vasc Surg* 2012;56:1222-31.
 45. Matsumoto AH, Angle JF, Secic M, Carlson GA, Fisher L, Fairman RM. *Secondary procedures following thoracic aortic stent grafting in the first 3 years of the VALOR test and VALOR II trials*. *J Vasc Interv Radiol* 2014;25:685-92.
 46. VIRTUE Registry Investigators. *Mid-term outcomes and aortic remodelling after thoracic endovascular repair for acute, subacute, and chronic aortic dissection: the VIRTUE Registry*. *Eur J Vasc Endovasc Surg* 2014;48:363-71.
 47. Patterson BO, Vidal-Diez A, Karthikesalingam A, Holt PJ, Loftus IM, Thompson MM. *Comparison of aortic diameter and area after endovascular treatment of aortic dissection*. *Ann Thorac Surg* 2015;99:95-102.
 48. Brooks M, Loftus I, Morgan R, Thompson M. *The Valiant thoracic endograft*. *J Cardiovasc Surg (Torino)* 2006;47:269-78.
 49. Khojnejzhad A, Donayre CE, Azizzadeh A, White R; RESCUE investigators. *One-year results of thoracic endovascular aortic repair for blunt thoracic aortic injury (RESCUE trial)*. *J Thorac Cardiovasc Surg* 2015;149:155-61.
 50. Khojnejzhad A, Azizzadeh A, Donayre CE, et al. *Results of a multicenter, prospective trial of thoracic endovascular aortic repair for blunt thoracic aortic injury (RESCUE trial)*. *J Vasc Surg* 2013;57:899-905.
 51. Roselli EE, Arko FR 3rd, Thompson MM; Valiant Mona LSA Trial Investigators. *Results of the Valiant Mona LSA early feasibility study for descending thoracic aneurysms*. *J Vasc Surg* 2015;62:1465-71.