

EVAR in Hostile Aortic Neck; indications outside the IFU's

Fouad S. Fouad¹ and Abdelrahman Mohamed²

¹Division of vascular Surgery, Department of Surgery, Cairo University

²Vascular Surgery Department, Ain-Shams University

ABSTRACT

Aim: The aim of this study was to evaluate the outcome of unselected, real-world patients with "off-label" proximal necks treated with endovascular repair (EVAR). **Patients and Methods:** This is a retrospective study of 9 patients with AAA who underwent endovascular repair over a period of 18 months from January 2013 and were followed up for one year. All patients had an aortic neck that had challenging anatomy either short (less than 10 mm) (n=4) or severely angulated (more than 60°) (n=5). None of them had more than 50% of the neck circumference lined by mural thrombus. None of the patients had a conical neck. The follow-up protocol included physical examination, duplex-ultrasound scan (DUS), and CT at 30 days.

Results: During the study period, 9 patients underwent standard EVAR. They were 7 men and 2 women with an age range of 56 to 74 with a mean of 65 years. All of them had one or more comorbidities such as: Diabetes (n=3), hypertension (n=5), coronary artery disease (CAD)(n=4), COPD (n=2) and previous laparotomy (n=1). The device that was used is the Endurant II® (Medtronic, Santa Rosa, CA, USA). Immediate technical success was achieved in all cases. One patient had acute myocardial infarction and one had temporary renal dysfunction within 30 days. At 1-year follow-up, 1 patient suffered a type Ia endoleak which required a proximal aortic extension, and 1 had an acute iliac limb occlusion, treated by surgical thromboembolectomy. **Conclusion:** This study presents some limitations; it is a non-randomized retrospective study with a small number of patients. Also, a longer follow-up would be needed to confirm the durability of EVAR in patients with hostile aortic necks. We do confirm the notion that this minimally invasive procedure can be performed safely and effectively in patients with challenging neck anatomy.

Keywords: EVAR, AAA, endoleak

INTRODUCTION

The link between aortic neck anatomy and the development of complications, such as type Ia endoleak and endograft migration, has been evident since the inception of EVAR in the early 1990s.²⁻⁴

A short infrarenal neck length and excessive aortic neck angulation can compromise the proximal fixation and sealing of the EVAR device, both in the immediate and the long term.

Neck diameter is also important whether absolute (in cylindrical necks) or relative (i.e. conical necks). The presence of mural thrombus in the proximal neck and the amount of calcium in its wall also contribute to unfavorable neck anatomy.⁵

A neck length of 15 mm is generally considered to be the minimum requirement for reliably achieving adequate infrarenal graft fixation especially for new generation devices. Neck lengths shorter than 15 mm are associated with higher rates of early and late type Ia

endoleaks, with approximately 10% to 40% requiring intraoperative proximal aortic cuff deployment. Equally, severe aortic neck angulation affects proximal fixation. Conventional wisdom is that neck angulation greater than 45 to 60 degrees constitutes a relative contraindication for use of AAA endografts.⁶

PATIENTS AND METHODS

This is a retrospective study of 9 patients with AAA who underwent endovascular repair over a period of 18 months from January 2013 and were followed up for one year.

All patients had an aortic neck that had challenging anatomy either short (less than 10 mm)(n=4) or severely angulated (more than 60°) (n=5). None of them had more than 50% of the neck circumference lined by mural thrombus. None of the patients had a conical neck.

Preoperative Planning

CT angiography was performed using a multidetector CT scan with and without contrast

medium during the arterial and venous phases, at a thickness of 1 mm. All measurements (diameter, length, and angles) were performed using a workstation with dedicated reconstruction software and center lumen line (CLL) reconstruction. Post-analysis included three-dimensional (3D) volume rendering, preoperative simulated angiographic projections, and multiplanar reconstruction. In particular, AAA neck length was defined as the longitudinal distance between the first transverse computed tomography (CT) section directly distal to the lowermost renal artery and the first transverse CT section that showed at least a 15% larger outer aortic wall diameter, whereas infrarenal AAA neck angulation was defined as the true angle between the longitudinal axis of the proximal AAA neck and the longitudinal axis of the AAA lumen as analyzed on three-dimensional CT reconstructions.

End Points

The end points were 30-day and 1-year technical and clinical success. Primary technical success was defined as successful passage of the delivery system through the iliac vessels, the correct deployment of the device, the appropriate positioning of the contralateral limb, and the complete withdrawal of the delivery system in the absence of surgical conversion, mortality, type Ia, Ib, or III endoleaks, and migration (> 5 mm displacement) or stent-graft limb occlusion in the first 24 hours after surgery. The post-operative patency of renal arteries was assessed by duplex scan upon discharge.

Clinical success was defined as the absence of intraoperative, 30-day, or in-hospital mortality or any significant morbidity such as aneurysm rupture, major adverse event (MAE), minor adverse event. Acute myocardial infarction (AMI), respiratory complications requiring invasive mechanical ventilation, and renal dysfunction (RD) were considered as MAE. All other medical conditions were registered as minor. AMI was suggested by electrocardiographic changes and confirmed by the elevation of cardiac enzymes, regardless of symptoms. RD was defined as a rise in serum creatinine exceeding the baseline value by 30% and surpassing an absolute level of 2.0 mg/dL.

The follow-up protocol included physical examination, duplex-ultrasound scan (DUS), and CT at 30 days.

RESULTS

During the study period, 9 patients underwent standard EVAR. They were 7 men and 2 women with an age range of 56 to 74 with a mean of 65 years. All of them had one or more comorbidities that made the endovascular option more appealing than open surgical repair such as: Diabetes (n=3), hypertension (n=5), coronary artery disease (CAD)(n=4), COPD (n=2) and previous laparotomy (n=1).

An epidural anesthetic was used in 3 (33.3%) of patients, while all other cases were performed under general anesthesia. Bilateral surgical cutdown to both groins were performed in all patients.

The stent-graft device that was used is the Endurant II® (Medtronic, Santa Rosa, CA, USA). Immediate technical success was achieved in all cases.

At 30-day follow-up, no endoleaks, reintervention, stent-graft migration, or AAA-related mortality were observed. One patient had AMI and one had temporary RD. All patients completed the 1-year follow-up. No AAA-related death or AAA rupture was reported. At 1-year follow-up, 1 patient suffered a type Ia endoleak due to downward stent graft migration which required a proximal aortic extension, and 1 had an acute iliac limb occlusion, treated by surgical thromboembolectomy.

DISCUSSION

Some authors who evaluated first-generation devices concluded that application of endografts outside anatomically specific IFU variables had an incremental negative effect on late results, indicating that adherence to the IFU guidelines was appropriate to clinical practice when using such devices.⁷

The outcomes of EVAR with the newer generations of devices, which have different profiles and more active fixation mechanisms, are still unknown beyond the IFU.⁸

In our series the outcomes of the patients compare favorably with those from other series internationally.

In a German series of 177 consecutive patients with AAA's who were treated by the Endurant stent-graft the 30-day rate of type I endoleak was higher amongst the 56 patients with off-label use

compared with no type I endoleak amongst the 121 patients within the IFU (2 patients ,3.6% vs. 0 in IFU). Nevertheless, after a follow-up of 1 year, this finding did not affect the results in terms of survival or freedom from any device-related reintervention.⁹

Similarly, AbuRahma and colleagues concluded that late reinterventions were no more frequent in patients with a very short proximal aortic neck, despite a higher rate of early and late type I endoleak.¹⁰

In a study from the Netherlands severe neck angulation had no effect on the midterm outcomes as long as there was adequate length of the aortic neck.¹¹

In studies mentioned above, the presence of a ‘‘hostile proximal aortic neck,’’ as defined by the manufacturer’s IFU, did not significantly affect short- and mid- term clinical success.

The prevalence of severe comorbidities amongst our population would call for more demand for EVAR procedures regardless of their compatibility to devices' IFU.

The use of branched or fenestrated endografts is clearly a suitable alternative to standard EVAR in patients with challenging necks. However, we should take into account an obvious increase in costs compared with standard grafts, and a non-negligible risk of reoperation because of branch-related complications.

CONCLUSION

This study presents some limitations; it is a non-randomized retrospective study with a small number of patients.

Also, a longer follow-up would be needed to confirm the durability of EVAR in patients with hostile aortic necks. We do confirm the notion that this minimally invasive procedure can be performed safely and effectively in patients with challenging neck anatomy.

REFERENCES

1. Dillavou ED, Muluk SC, Rhee RY, Tzeng E, Woody JD, Gupta N, et al. Does hostile neck anatomy preclude successful endovascular aortic aneurysm repair? *J VascSurg*2003;38:657-63.
2. Fox AD, Whiteley MS, Murphy P, Budd JS, Horrocks M. Comparison of magnetic resonance imaging measurements of abdominal aortic aneurysms with measurements obtained by other imaging techniques and intraoperative measurements: possible implications for endovascular grafting. *J VascSurg*1996;24:632-8.
3. Moore WS, Vescera CL. Repair of abdominal aortic aneurysm by transfemoral endovascular graft placement. *Ann Surg*1994;220:331-9; discussion: 339-41.
4. Parodi JC. Endovascular repair of abdominal aortic aneurysms and other arterial lesions. *J VascSurg*1995;21:549-55.
5. Jordan WD, Ouriel K, Mehta M, Varnagy D, Moore WM, Arko FR, Joye J, de Vries JP. Outcome-based anatomic criteria for defining the hostile aortic neck. *Journal of vascular surgery*. 2015 Jun 30;61(6):1383-90.
6. O’Brien GC, Raffi Q and White GH. Endovascular Treatment of NonrupturedInfrarenal Aortic and Aortoiliac Aneurysms in Stanley JC, Veith F, Wakefield TW(eds). Current therapy in vascular and endovascular surgery. Elsevier Health Sciences; 2014 Mar 28.
7. Abbruzzese TA, Kwolek CJ, Brewster DC, et al. Outcomes following endovascular abdominal aortic aneurysm repair (EVAR): an anatomic and device-specific analysis. *J VascSurg*2008;48:19-28.
8. Verhagen HJ, Torsello G, De Vries JP, et al. Endurant stent-graft system: preliminary report on an innovative treatment for challenging abdominal aortic aneurysm. *J CardiovascSurg (Torino)* 2009;50:153-8.
9. Torsello G, Troisi N, Donas KP, et al. Evaluation of the Endurant stent graft under instructions for use vs off-label conditions for endovascular aortic aneurysm repair. *J VascSurg*2011; 54:300-6.
10. AbuRahma AF, Campbell J, Stone PA, et al. The correlation of aortic neck length to early and late outcomes in endovascular aneurysm repair patients. *J VascSurg*2009;50:738-48.
11. Oliveira NF, Gonçalves FB, de Vries JP, Ultee KH, Werson DA, Hoeks SE, Moll F, van Herwaarden JA, Verhagen HJ. Mid-term results of EVAR in severe proximal aneurysm neck angulation. *European Journal of Vascular and Endovascular Surgery*. 2015 Jan 31;49(1):19-27.

TEVAR in Uncomplicated Acute Type B Aortic Dissection

¹Fouad S Fouad and ²Abdelrahman Mohamed

¹Division of Vascular Surgery, Department of Surgery, Cairo University

²Department of Vascular Surgery, Ain-Shams University

ABSTRACT

Aim : TEVAR in uncomplicated Acute Type B Aortic Dissection can be performed safely and may reduce late false lumen expansion and mortality compared to best medical therapy (BMT) alone. **Methods:** This is a retrospective study in which six patients had endovascular treatment (TEVAR) for uncomplicated Acute Type B Aortic Dissection over a period of 12 months starting from January 2015 with a mean follow-up six months (range from 4 to 15 months). The initial goals for BMT were to reduce SBP to 100 - 120 mm Hg and pain relief. All of the patients had an initial CT angiogram on admission to confirm the diagnosis and to exclude rupture and malperfusion and to plan the endovascular intervention. Follow up CT scans were obtained to exclude rapid progression of the size of the false lumen. The devices used were Zeinith[®] TX2[®] (Cook Medical, Indiana, USA) in 4 patients and Relay Plus[®] (Bolton Medical, Werfern Group, USA) in 2 patients. **Results:** By the time of intervention all our patients were treated at their subacute phase (i.e. between 14 and 90 days). The left subclavian artery had to be covered in four out of six patients. None of them developed ischemic symptoms in their arms. A proximal Type I endoleak was evident on the completion angiogram in two patients which necessitated ballooning in one patient and a proximal aortic cuff in the other. The postoperative course was uneventful with no endograft related complications and no clinical evidence of spinal cord ischemia. There were no in-hospital or 30-day mortality rates. One patient (16.6%) died after six months from the intervention. Follow up CT scans done 1month, 3months and 6months after discharge revealed no increase in the maximum aortic diameter and total complete thrombosis of the false lumen in all patients. **Conclusion:** This study has many limitations. First being a single center retrospective study. Second, limited number of patients enrolled and also a short duration of follow-up, yet it represents an addition to case collection of proven efficacy and safety to manage those cases with type B aortic dissection.

Keywords: Type B Aortic Dissection, TEVAR, False Lume

INTRODUCTION

An intimal tear is the inciting pathology of an aortic dissection, with a cleavage plane affecting the intima and media of the arterial wall and propagating to some degree, either antegrade or retrograde.^{1,2}

The typical type B dissection has an intimal tear that originates within few centimeters of the takeoff of the left subclavian artery, attributable to the large pressure fluctuations per unit time notoriously occurring in this area.³⁻⁵

Several imaging modalities are used in the diagnostic evaluation of aortic dissection, with computed tomographic angiography(CTA) and magnetic resonance imaging (MRI/MRA) having the highest sensitivity and specificity for diagnosis.

In spite of its higher sensitivity and specificity, MRI is more expensive with more

time consumption in such acute pathology⁸⁻¹¹. Current consensus holds that patients with complicated Acute Type B Aortic Dissection (cATBAD) could be treated with thoracic endovascular aortic repair (TEVAR)¹², leading to better in-hospital survival than open surgery.¹³

In an interdisciplinary expert consensus document on management of Type B Aortic Dissection the following suggestions were made to define complicated dissection as having one or more of the following:

- Malperfusion is indicative of impending organ failure (spinal, iliac, or visceral arteries) and must be recognized early. Diagnosis of static or dynamic organ malperfusion is corroborated by laboratory markers (bilirubin, amylases, lactate dehydrogenase, creatine phosphokinase and serum creatinine) and imaging data.

- Refractory Hypertension which is defined as failure of control despite full medical therapy
- Increases in perioaortic hematoma and hemorrhagic pleural effusion in 2 subsequent CT examinations during medical expectant management of acute type B aortic dissection are findings suggestive of impending rupture.¹⁴

Approximately 25% of patients presenting with acute type B aortic dissection are complicated at admission by malperfusion syndrome or hemodynamic instability, resulting in a high risk of early death if untreated.^{12,15,16}

Patients with uncomplicated Acute Type B Aortic Dissection (uATBAD) are commonly treated with conservative therapy (best medical treatment [BMT]). However, the long-term outcome of medical therapy alone is suboptimal¹⁷ with a reported 30% to 50% mortality rate at 5 years and a delayed expansion of the false lumen in 20% to 50% of patients at 4 years.¹⁸

Subgroup analysis showed that a thrombosed false lumen predicts lower event rates with ATBAD¹⁹ and favorable false lumen remodeling after TEVAR.²⁰⁻²²

METHODS

This is a retrospective study in which six patients had endovascular treatment (TEVAR) for uncomplicated Acute Type B Aortic Dissection over a period of 12 months starting from January 2015 with a mean follow-up six months (range from 4 to 15 months).

We used the following classification to define types of dissection according to the duration:²³ Acute dissection: <15 days and Subacute dissection: 15-92 days while Chronic dissection: > 92 days.

There were four men and two women with an age range of 56-72 years and a mean of 64. The only presenting symptom was acute onset of chest pain with negative screening for acute coronary syndromes using the three consecutive sets of cardiac enzymes and frequent ECG tracings. They all had hypertension (> 140/90 mmHg) on admission but only three of them were known to be hypertensive prior to admission.

Their high blood pressure was controlled in the critical care unit. Antihypertensive medications (calcium-channel blockers, nitroglycerine, b-blockers, or a combination) were

administered to all patients, and were able to control pressure to the goal value within the initial 3 days. The initial goals for BMT were to reduce SBP to 100-120 mm Hg and pain relief. For persistent chest pain, after blood pressure control, a narcotic analgesic (morphine hydrochloride) was prescribed.

There were no symptoms or signs of branch vessel malperfusion involving the lower extremities, the bowels or the kidneys, nor neurological deficit. There were no lab markers indicative of ischemic bowel, failing kidneys or skeletal muscle infarction (e.g. rising serum lactate, creatinine and/or CPK levels).

All of the patients had an initial CT angiogram on admission to confirm the diagnosis and to exclude rupture and malperfusion and to plan the endovascular intervention. Follow up CT scans were obtained to exclude rapid progression of the size of the false lumen which would've been indicative of impending rupture. The endografts that were used to repair these dissections were Zeinth[®] TX2[®] (Cook Medical, Indiana, USA) in 4 patients and Relay Plus[®] (Bolton Medical, Werfern Group, USA) in 2 patients. They ranged in diameter from 24-36 mm and they ranged in length from 120-200mm. They were all introduced via a single groin cutdown mostly on the left side in five patients and in one patient we went through the right groin because of a tight iliac stenosis on the left side. A right brachial artery access was used in all patients to introduce a pigtail catheter for angiographic purposes. A transesophageal echo cardiogram (TEE) was used in all patients to confirm the presence of the guide wire in the true lumen.

Our end-points were all cause and aorta specific mortality rates, the absence of maximum aortic diameter progression and shrinking of the false lumen.

RESULTS

The initial CT angiogram revealed the site of the proximal entry tear to be within 2 cm of the takeoff of the left subclavian artery in all patients. The size of the proximal entry tear was more than 1 cm in two patients. The false lumen diameter was 22 mm or more in three patients.

By the time of intervention all our patients were treated at their subacute phase (i.e. between 14 and 90 days). The left subclavian artery had to

be covered in four out of six patients to ensure adequate seal in the proximal landing zone. None of these four patients developed ischemic symptoms in their arms in their postoperative period and consequently we did not have to do a Carotid-Subclavian bypass.

A proximal Type I endoleak was evident on the completion angiogram in two patients in whom the left subclavian artery was already covered which necessitated ballooning in one patient and a proximal aortic cuff in another patient and that was sufficient to achieve a good seal at the proximal landing zone without encroaching on the origin of the left Common Carotid Artery. The postoperative course was uneventful with no endograft related complications and no clinical evidence of spinal cord ischemia. There were no in-hospital or 30-day mortality rates. One patient (16.6%) died after six months from the intervention and although we couldn't determine whether or not it was aorta related but it may be worth mentioning that this particular patient was known to have ischemic heart disease. Pre-discharge CT scans revealed complete thrombosis of the false lumen in four out of six patients (66.6%) with 2 patients having incomplete thrombosis of their false lumen. Incomplete thrombosis was defined as the presence of blood flow in any portion of the false lumen parallel to the stent graft, and complete thrombosis was defined as absence of blood flow in any portion of the false lumen parallel to the stent graft.

After discharge, all patients with hypertension were treated with calcium antagonists, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, or b-blockers (either alone or in combination) to maintain the morning SBP (140 mm Hg or less).

Follow up CT scans done 1month, 3months and 6months after discharge revealed no increase in the maximum aortic diameter and total complete thrombosis of the false lumen in all patients.

DISCUSSION

In our modest experience thoracic endovascular repair inpatients with uncomplicated acute type B aortic dissection (uATBAD) was safe and effective. However, the benefit of this type of treatment does not become apparent

before two years. This is supported by evidence from prospective randomized studies such as the INSTEAD trial which demonstrated that endovascular repair had no advantage over best medical therapy in the first two years²⁴. However, the extended follow-up of those same patients (INSTEAD-XL) revealed higher aorta related mortality rates in the BMT only study arm²⁶. Similar outcomes after five years were suggested by Fattori et al from the IRAD (international registry of aortic dissections) database analysis which reflects "real-world" scenario.²⁰

Our results compare favorably with the 1-year results of the ADSORB trial (multicenter randomized European trial) that showed more frequent false lumen thrombosis and aortic remodeling in those patients treated medically plus TEVAR compared to those managed only medically.²⁷

Although it was not intended but by the time of intervention, all our patients fell into the subacute category (14-90 days) and this may have aided in improving the outcomes as there is a growing body of evidence that prophylactic TEVAR in uATBAD cases if done in the subacute phase reduces the incidence of retrograde dissection (which could be fatal) and at the same time has no disadvantage (compared to acute cases) regarding aortic remodeling.²⁸

Regarding the coverage of the left subclavian artery (LSA); some surgeons routinely perform LSA revascularization in these patients, whereas others do so prophylactically in certain circumstances (e.g., a dominant left vertebral artery, a previous left internal mammary coronary artery bypass graft, or absent right vertebral artery) and some do it only if symptoms develop after TEVAR.²⁹ We decided to adopt the latter policy, and none of our patients had adverse outcomes from left subclavian artery coverage and therefore required no intervention.

Published reports show the baseline risks of adverse outcomes in patients who have TEVAR and LSA coverage are 6% arm ischemia, 4% spinal cord ischemia, 2% vertebrobasilar ischemia, 5% anterior circulation stroke, and 6% death.³⁰

We were lucky enough not to have any cases of spinal cord ischemia however should paraparesis develop it should be emergently treated by CSF drainage and blood pressure augmentation as demonstrated by a recent study

from the cardiovascular group in Emory University in Atlanta.³¹

The ideal concept is to perform stent-grafting in this subgroup of patients with uATBAD prone to developing progression of the disease and future complications. A number of studies have suggested several prognostic factors of early or late adverse events such as the patency of the false lumen in the follow-up, an initial aortic diameter ≥ 4 cm with a patent false lumen, an initial false lumen diameter ≥ 22 mm in the proximal descending aorta, recurrent pain or hypertension, partial false lumen thrombosis and a proximal entry tear size > 10 mm.³²

CONCLUSION

This study has many limitations. First being a single center retrospective study. Second, limited number of patients enrolled and also a short duration of follow-up, yet it represents an addition to case collection of proven efficacy and safety to manage those cases with type B aortic dissection.

REFERENCES

1. Khan IA, Nair CK. Clinical, diagnostic, and management perspectives of aortic dissection. *Chest* 2002;122:311-28.
2. Wilson SK, Hutchins GM. Aortic dissecting aneurysms: causative factors in 204 subjects. *Arch Pathol Lab Med* 1982;106: 175-80.
3. Crawford ES. The diagnosis and management of aortic dissection. *JAMA* 1990;264:2537-41.
4. Hirst AE, Johns VJ, Kime SW. Dissecting aneurysm of the aorta: a review of 505 cases. *Medicine (Baltimore)* 1958;37: 217-79.
5. Wheat MW. Acute dissection of the aorta. *CardiovascClin*1987;17:241-62.
6. Hartnell G, Costello P. The diagnosis of thoracic aortic dissection by noninvasive imaging procedures. *N Engl J Med* 1993;328:1637; author reply 1638.
7. Clague J, Magee P, Mills P. Diagnostic techniques in suspected thoracic aortic dissection. *Br Heart J* 1992;67:428-9.
8. LePage MA, Quint LE, Sonnad SS, Deeb GM, Williams DM. Aortic dissection: CT features that distinguish true lumen from false lumen. *AJR Am J Roentgenol* 2001;177: 207-11.
9. Hata N, Tanaka K, Imaizumi T, Ohara T, Ohba T, Shinada T, et al. Clinical significance of pleural effusion in acute aortic dissection. *Chest* 2002;121:825-30.
10. Fruehwald FX, Neuhold A, Fezoulidis J, Globits S, Mayr H, Wicke K, et al. Cine-MR in dissection of the thoracic aorta. *Eur J Radiol*1989;9:37-41.
11. Tomiguchi S, Morishita S, Nakashima R, Hara M, Oyama Y, Kojima A, et al. Usefulness of turbo-FLASH dynamic MR imaging of dissecting aneurysms of the thoracic aorta. *CardiovascInterventRadiol*1994;17:17-21.
12. 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. *J Am CollCardiolIntv*2008;1:395-402.
13. Chou HP, Chang HT, Chen CK, et al. Outcome comparison between thoracic endovascular and open repair for type B aortic dissection: a population-based longitudinal study. *J Chin Med Assoc* 2015;78:241-8.
14. Fattori R, Cao P, De Rango P, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. *J Am CollCardiol*2013;61:1661-78.
15. Tsai TT, Fattori R, Trimarchi S, et al. International Registry of Acute Aortic Dissection. Long-term survival in patients presenting with type B acute aortic dissection: insights from the International Registry of Acute Aortic Dissection. *Circulation* 2006;114:2226-31.
16. Trimarchi S, Eagle KA, Nienaber CA, et al. International Registry of Acute Aortic Dissection (IRAD) Investigators. Importance of refractory pain and hypertension in acute type B aortic dissection: insights from the International Registry of Acute Aortic Dissection (IRAD). *Circulation* 2010;122:1283-9.
17. Durham CA, Cambria RP, Wang LJ, et al. The natural history of medically managed acute type B aortic dissection. *J VascSurg*2015;61:1192-9.
18. Akin I, Kische S, Ince H, Nienaber CA. Indication, timing and results of endovascular

- treatment of type B dissection. *Eur J VascEndovascSurg* 2009; 37:289–96.
19. Erbel R, Oelert H, Meyer J, et al. Effect of medical and surgical therapy on aortic dissection evaluated by transesophageal echocardiography. Implications for prognosis and therapy. The European Cooperative Study Group on Echocardiography. *Circulation* 1993;87:1604–15.
 20. 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). *J Am CollCardiolIntv* 2013;6:876–82.
 21. Shu C, He H, Li QM, et al. Endovascular repair of complicated acute type-B aortic dissection with stentgraft: early and mid-term results. *Eur J VascEndovascSurg* 2011;42:448–53.
 22. Eriksson MO, Steuer J, Wanhainen A, et al. Morphologic outcome after endovascular treatment of complicated type B aortic dissection. *J VascIntervRadiol* 2013;24:1826–33.
 23. VIRTUE Registry Investigators. Mid-term outcomes and aortic remodelling after thoracic endovascular repair for acute, subacute, and chronic aortic dissection: the VIRTUE Registry. *European Journal of Vascular and Endovascular Surgery*. 2014 Oct 31;48(4):363-71.
 24. Qin YL, Deng G, Li TX, Wang W, Teng GJ. Treatment of acute type-B aortic dissection: thoracic endovascular aortic repair or medical management alone? *J Am CollCardiolIntv* 2013;6: 185–91.
 25. Nienaber CA, Rousseau H, Eggebrecht H, et al. Randomized comparison of strategies for type B aortic dissection: the INvestigation of STent Grafts in Aortic Dissection (INSTEAD) trial. *Circulation* 2009; 120:2519–28.
 26. Nienaber CA, Kische S, Rousseau H, Eggebrecht H, Rehders TC, Kundt G, Glass A, Scheinert D, Czerny M, Kleinfeldt T, Zipfel B. Endovascular repair of type B aortic dissection. *Circulation: Cardiovascular Interventions*. 2013 Aug 1;6(4):407-16.
 27. Brunkwall J, Lammer J, Verhoeven E, et al. ADSORB: a study on the efficacy of endovascular grafting in uncomplicated acute dissection of the descending aorta. *Eur J VascEndovascSurg* 2012;44:31-6.
 28. Nienaber CA. The Art of Stratifying Patients with Type B Aortic Dissection. *Journal of the American College of Cardiology*. 2016 Jun 21;67(24):2843.
 29. Matsumura JS, Lee WA, Mitchell RS, Farber MA, Murad MH, Lumsden AB, Greenberg RK, Safi HJ, Fairman RM. The Society for Vascular Surgery Practice Guidelines: management of the left subclavian artery with thoracic endovascular aortic repair. *Journal of vascular surgery*. 2009 Nov 30;50(5):1155-8.
 30. Rizvi AZ, Murad MH, Fairman RM, Erwin PJ, Montori VM. The effect of left subclavian artery coverage on morbidity and mortality in patients undergoing endovascular thoracic aortic interventions: a systematic review and meta-analysis. *Journal of vascular surgery*. 2009 Nov 30;50(5):1159-69.
 31. Leshower BG, Duwayri YM, Chen EP, Li C, Zehner CA, Binongo JN, Veeraswamy RK. Aortic Remodeling After Endovascular Repair of Complicated Acute Type B Aortic Dissection. *The Annals of Thoracic Surgery*. Published online in 2016
 32. Moulakakis KG, Mylonas SN, Dalainas I, Kakisis J, Kotsis T, Liapis CD. Management of complicated and uncomplicated acute type B dissection. A systematic review and meta-analysis. *Ann CardiothoracSurg* 2014; 3(3):234-246.
-