Eur J Vasc Endovasc Surg (2019) 57, 633–638

Editor's Choice – Long-term Survival and Risk Analysis in 136 Consecutive Patients With Type B Aortic Dissection Presenting to a Single Centre Over an 11 Year Period

Rachel E. Clough [a,b], David Barillà [a], Pascal Delsart [a], Guillaume Ledieu [a], Rafaelle Spear [a], Siobhan Crichton [c], Claire Mounier Vehier [a], Janet Peacock [c], Jonathan Sobocinski [a], Stephan Haulon [a,d,*]

[a] Aortic Centre, CHRU de Lille, France
[b] Division of Imaging Sciences and Biomedical Engineering, King's College London, UK
[c] Division of Health and Social Care Research, King's College London, UK
[d] Aortic Centre, Hôpital Marie Lannelongue, Le Plessis-Robinson, Université Paris Sud, France

WHAT THIS PAPER ADDS

The management of patients with type B aortic dissection is complex. A number of cases series have been published to try to better understand these patients but they are limited by their small sample size. The International Registry on Aortic Dissection (IRAD) is a highly valuable resource but pools data from a number of centres across the world and is therefore limited by data heterogeneity. This study describes the findings of the largest single centre series published to date and provides new insights into management of these complex patients.

Objectives: To evaluate, in patients with acute type B aortic dissection, the results of medical and endovascular treatment in a large single centre experience and to investigate the clinical and imaging features on presentation that relate to poor outcome.

Methods: This was a retrospective analysis of prospectively collected clinical and CT imaging data. Consecutive patients (136) with acute type B aortic dissection were included in the study over an 11 year period. The characteristics of patients receiving endovascular (complicated) or medical treatment (uncomplicated) were compared. Kaplan–Meier estimators were used to estimate cumulative overall survival and survival free of aortic events. Factors associated with overall and aortic event free survival were also explored using Cox proportional hazards models.

Results: The mean follow up was 51 months (1–132), during which time 33 deaths and 48 aortic events occurred. At one and five years, overall survival was 94.0% and 74.8%, respectively, and freedom from aortic events was 75.6% and 58.7%. There was no difference in all cause survival and aortic event free survival at one and five years between the patients treated endovascularly and those receiving medical treatment alone. Risk analysis for aortic events demonstrated the maximum size of the proximal entry tear, the maximum thoracic aortic diameter, and the thoracic aortic false lumen maximum diameter to have a significant effect on the incidence of aortic events.

Conclusions: Active management of patients with type B aortic dissection results in good long-term survival even in the presence of features traditionally associated with adverse outcomes. All patients require close lifetime surveillance as aortic events continue to occur during follow up even after endografting.

Keywords: Aortic dissection, Risk analysis, Survival
Article history: Received 7 December 2017, Accepted 23 August 2018, Available online 4 October 2018

INTRODUCTION

Type B aortic dissection is a complex clinical entity. In the presence of complications such as rupture and end organ ischaemia, urgent endovascular intervention is required.[1] Uncomplicated cases are currently treated medically, with active management of blood pressure, to try to prevent complications during follow up such as extension of the dissection, aneurysm formation, and rupture.

Previous series have however, demonstrated that even in the presence of good blood pressure control survival is poor, with only 50–70% of patients alive at five years, with a delayed expansion of the false lumen in 20–50% of

* Corresponding author. Aortic Centre, Department of Aortic and Vascular Surgery, Hôpital Marie Lannelongue, Le Plessis-Robinson, INSERM UMR_S 999, Université Paris Sud, France.
E-mail address: s.haulon@hml.fr (Stephan Haulon).

https://doi.org/10.1016/j.ejvs.2018.08.042

patients at four years.[2–4] Many of the deaths that occur during follow up are aortic related.

A number of groups, largely in Asian patient populations, have tried to identify clinical and anatomical features that could be measured on presentation and be used to predict outcome, and therefore identify patients at high risk of aortic events and death during follow up.[5] The majority of these studies have been performed in small series (<40 patients), with results from some studies contradicting the results of others and so currently there is no reliable method to identify these high risk patients.

There is growing interest in early endovascular treatment of patients with type B aortic dissection to prevent aortic events during follow up and therefore improve survival. It is hoped that treating patients early will produce the best outcomes because the aorta has plasticity and is therefore likely to undergo positive aortic remodelling following stent graft insertion.[6]

The present authors have 11 years of experience in the management of patients with type B aortic dissection using best medical treatment for uncomplicated and thoracic endovascular repair (TEVAR) for complicated dissection. The aim of this study was to evaluate the results that can be achieved with medical and endovascular treatment in these patients in a large single centre experience and to investigate the features on presentation that relate to a poor outcome.

MATERIALS AND METHODS

Study design

One hundred and thirty-six consecutive patients with acute type B aortic dissection were included in the study over an 11 year period. All patients were managed in a high dependency setting with medical management implemented by medical specialists trained in the management of hypertension. Patients that presented with evidence of aortic rupture, end organ ischaemia, or ongoing pain despite good blood pressure control, were defined as complicated and treated endovascularly, those without these features were treated with best medical therapy. Patients were excluded if the aortic dissection was secondary to trauma or iatrogenic injury. Patients were categorised into groups based on their treatment. All patients were followed up by both a cardiologist with a special interest in aortic dissection and hypertension and a vascular surgeon.

Endovascular repair

Thoracic endovascular repair was performed in a standard operating room with mobile C-arm or in a hybrid operating room using percutaneous vascular access whenever possible. Left common carotid to left subclavian artery (LSCA) bypass or transposition was considered if the stent graft covered the origin of the LSCA. Absolute indications for LSCA revascularisation were: left internal mammary artery bypass graft; diminutive, atretic, or absent right vertebral artery; left arm arterio-venous fistula for haemodialysis; patent left axillo-femoral bypass graft; and dominant left vertebral artery. These procedures were performed prior to, or at the time of the TEVAR procedure, depending on the level of urgency of the case. The stent graft was sized to the proximal non-dissected aorta with 10—15% oversizing. In cases of rupture the stent grafts were positioned to cover the thoracic aorta from proximal to the primary entry tear to the level of the coeliac trunk.

Best medical therapy

Best medical therapy was implemented using a combination of antihypertensive medications. The preferred stepwise approach was as follows: β blocker, ACE inhibitor, calcium channel blocker, then diuretics with a final option of α receptor blocker and/or a centrally acting antihypertensive agent. Blood pressure control was based on the European Society of Cardiology guidelines with a target blood pressure of below 135/80 mmHg.

Image analysis

Computed tomography (CT) angiographic images of the aorta were acquired on presentation and during follow up using an iodinated contrast agent. All images were analysed on a dedicated Aquarius iNtuition workstation (TeraRecon, Foster City, CA, USA) by two vascular surgeons experienced in endograft planning for aortic dissection. The arterial phase images, reconstructed to $\leq$1 mm, were evaluated and the number of entry tears, the size of the primary entry tear, the length of the dissection, the amount of thrombosis in the false lumen, and the dimensions of the aorta and the true and false lumens were recorded. The true lumen was identified as the lumen continuous with the proximal non-dissected aorta. The maximum diameter of the primary entry tear was measured using multi-planar reformatted (MPR) images, false lumen thrombosis was assumed to be present when there was absence of contrast enhancement, and volumes were calculated by segmentation and summation over contiguous slices. The following were recorded: (i) the maximum aortic diameter; (ii) the true and false lumen diameters in the thoracic aorta at the level of the inferior pulmonary vein; and (iii) the true and false lumen diameters in the abdominal section at the level of the IMA (or at the mid-point of the third lumbar vertebra if the IMA could not be seen). All diameter measurements were made using MPR images to ensure precision.

Statistical analysis

The characteristics of patients receiving endovascular or medical treatment alone were summarised within each group and compared using Mann—Whitney, chi-square, or Fisher's exact test, as appropriate. Kaplan—Meier estimators were used to estimate cumulative overall survival and survival free of aortic events with 95% CI one and five years after admission. Aortic events were rupture, extension of the dissection, and further intervention. Kaplan—Meier estimators were also calculated within subgroups of patients. Factors associated with overall and aortic event free survival were also explored using Cox proportional hazards models. Because of the small sample sizes, for categorical data the estimation of one and five year survival rates and the application of Cox models were

Table 1. Baseline characteristics of 136 consecutive patients presenting with type B aortic dissection

	All (n = 136)	Endovascular treatment (n = 64)	Medical treatment only (n = 72)	p value
Mean age (± SD), years	61.7 (13.2)	58.5 (12.7)	64.5 (13.1)	.008
Male sex	105 (77.2)	51 (79.7)	54 (75.0)	.515
Mean height (± SD), cm	173.5 (9.7)	174.6 (8.9)	172.6 (10.3)	.264
Hypertension	97 (71.3)	48 (75.0)	49 (68.1)	.371
Diabetes	13 (9.6)	7 (9.9)	6 (9.4)	.924
Dyslipidaemia	34 (25.0)	11 (17.2)	23 (31.9)	.047
Median BMI (IQR), kg/m^2	27 (24.2–31.0)	27.0 (24.0–31.4)	26.9 (24.4–29.7)	.945
Coronary artery disease	8 (5.9)	3 (4.7)	5 (7.0)	.721
Co-existing AAA	13 (9.6)	7 (10.9)	6 (8.5)	.625
Previously treated AAA	6 (4.4)	0	6 (8.5)	.029
Renal insufficiency	0	0	0	
Connective tissue disease	8 (5.9)	6 (9.4)	2 (2.8)	.149
Family history of Marfan	5 (3.7)	3 (4.7)	2 (2.8)	.668

Data are presented as n (%) unless stated otherwise. BMI = body mass index; SD = standard deviation; IQR = inter quartile range; AAA = abdominal aortic aneurysm.

restricted to variables with at least 20% of patients in each subgroup. Where Cox models indicated a statistically significant association, Kaplan–Meier curves were plotted. For continuous variables cut points were selected that divided the data into three approximately equal sized groups and Kaplan–Meier curves estimates for each of these groups. Analysis was conducted using STATA 13MP and p values $< .05$ were considered to be statistically significant.

RESULTS

Baseline characteristics

The baseline characteristics for the 136 patients are summarised in Table 1. The average age at admission was 61.7 years and the majority of the cohort were male (77.2%). Seventy-one per cent of the cohort had hypertension, 9.6% had diabetes, and 5.9% had known connective tissue disease. Sixty-four patients presented with complications and were treated endovascularly. The most common complication was end organ ischaemia (n = 45); aortic rupture was present in 17 cases and five patients were treated for ongoing pain despite good blood pressure control. Three patients had both rupture and end organ ischaemia. The patients that were treated endovascularly were significantly younger than the patients treated with medical treatment alone (mean age 58.5 vs. 64.5 years). β Blockers were the most commonly used antihypertensive agent to treat aortic dissection in both treatment groups (Table S1).

The incidence of in hospital events were: acute coronary syndrome 3.7% (endovascular n = 3, medical treatment alone n = 2), neurological complications 7.4% (endovascular n = 7, medical treatment alone n = 3), dialysis 2.9% (endovascular n = 3, medical treatment alone n = 1), and pulmonary infection 11.8% (endovascular n = 11, medical treatment alone n = 5).

Anatomical features

The anatomical features of the cohort are shown in Table 2 and Table S2. At presentation the maximum aortic diameter was greater in the endovascularly treated group compared with the group treated with medical treatment alone. In both groups the dissection tended to involve both the thoracic and abdominal aorta (endovascular group 93%, medical treatment only group 81.7%). In the endovascularly treated group the true lumen tended to be smaller than the false lumen, whereas in the group that received medical treatment only the true lumen was larger than the false lumen. In both treatment groups a large proportion of the patients had a patent false lumen and there was no difference in the number of entry tears. The size of the largest entry tear was significantly greater in the endovascular treatment group.

At two years' follow up there was an average increase in the true lumen diameter of the thoracic aorta of 8 mm in the endovascular treatment group with no corresponding increase in the true lumen diameter of the abdominal segment in this group. The majority of the patients in the medically treated group had partial (51.7%) or complete (34.5%) thoracic aortic false lumen thrombosis, whereas in the endovascularly treated group approximately one third of patients had patency of the false lumen. At five years the average size of the false lumen in the endovascularly treated group had increased compared with the 2 year data and only 27.3% of patients had complete thrombosis of the thoracic aortic false lumen following endovascular treatment.

Survival analysis

The mean follow up in the cohort was 51 (1–132) months; 33 deaths, eight aortic related, 10 cardiac, 10 cancer related, and five other, occurred, and 48 aortic events were recorded. The 30 day all cause survival and aortic event free survival were 98.5% (94.3–99.6%) and 94.8% (89.4–97.5%), respectively. Cumulative survival was 94.0% (95% CI 88.4–97.0%) at one year and 74.8% (64.5–82.5%) at five years (Fig. 1). The aortic event free survival at one year and at five years was 75.6% (67.3–82.1%) and 58.7% (48.1–67.8%), respectively (Fig. 2).

Table 2. Anatomical features of 136 consecutive patients with type B aortic dissection, at first presentation and during follow-up.

Timepoint	Anatomical feature of dissection	All (n = 136)	Endovascular treatment (n = 64)	Medical treatment only (n = 72)	p value
At presentation					
	Median max aortic diameter (IQR), cm	38 (35–43)	41 (36–45)	37 (34–40)	.003
	Thoracic and abdominal aorta involved	103 (87.2)	53 (93.0)	50 (81.7)	.096
	Median total thoracic aortic diameter (IQR), cm	37 (34–42)	39 (34–42)	36 (32.5–38)	.006
	Median true lumen diameter (IQR), cm	18 (14–23)	18 (15–24)	19.5 (12.5–23)	.900
	Median false lumen diameter (IQR), cm	18 (12–26)	20 (14–28)	17 (10–23)	.060
	False lumen status, %				
	Patent	61 (52.6)	34 (59.7)	27 (45.8)	.105
	Partially thrombosed	45 (38.8)	21 (36.8)	24 (40.7)	
	Completely thrombosed	10 (8.6)	2 (3.5)	8 (13.6)	
2 year follow-up					
	Median max aortic diameter (IQR), cm	40.5 (36–44.5)	42 (38–47)	39 (35–42)	.088
	Median total thoracic aortic diameter (IQR), cm	40 (36–43)	41 (36–44)	38 (35–41)	.067
	Median true lumen diameter (IQR), cm	25 (15–30)	25.5 (16–30)	21 (13–28)	.180
	Median false lumen diameter (IQR), cm	18 (8–27)	18.5 (8–28)	18 (8–26)	.665
	False lumen status, %				
	Patent	17 (24.3)	13 (31.7)	4 (13.8)	.184
	Partially thrombosed	29 (41.4)	14 (34.2)	15 (51.7)	
	Completely thrombosed	24 (34.3)	14 (34.2)	10 (34.5)	
5 year follow-up					
	Median max aortic diameter (IQR), cm	40 (37–47)	42 (38–48)	37.5 (33–41.5)	.045
	Median total thoracic aortic diameter (IQR), cm	40.5 (36–44.5)	42 (38–47)	39 (35–42)	.088
	Median true lumen diameter (IQR), cm	23 (16–31)	20 (16–32)	28 (18–30)	.600
	Median false lumen diameter (IQR), cm	21 (8.5–28)	23 (12–28)	14 (4–34)	.316
	False lumen status, %				
	Patent	14 (28.6)	13 (39.4)	1 (6.3)	.049
	Partially thrombosed	19 (38.8)	11 (33.3)	8 (50.0)	
	Completely thrombosed	16 (32.7)	9 (27.3)	7 (43.8)	

Data are presented as n (%) unless stated otherwise. BMI = body mass index; IQR = inter quartile range.

There was no difference in all cause survival at one and five years between the patients treated endovascularly and those receiving medical treatment alone (HR 0.99 (0.49–2.02), p = .996). There was no difference in aortic event free survival at one and five years between the patients treated endovascularly and those receiving medical treatment alone (HR 1.33 (0.78–2.34), p = .329).

Factors associated with overall and aortic event free survival

Patient characteristics, cardiovascular risk factors and imaging features, and their association with all cause survival, are summarised in Tables S3–S5, respectively. Age was significantly associated with all cause survival with an

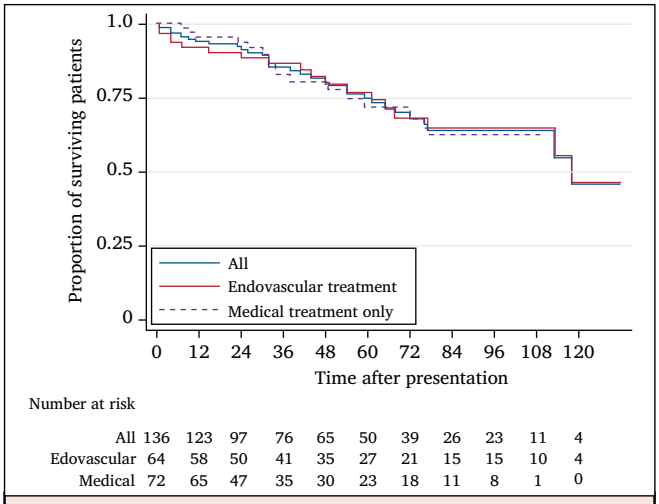

Figure 1. Kaplan–Meier estimate demonstrating 94.4% and 75.5% cumulative overall survival after type B aortic dissection at one and five years, respectively.

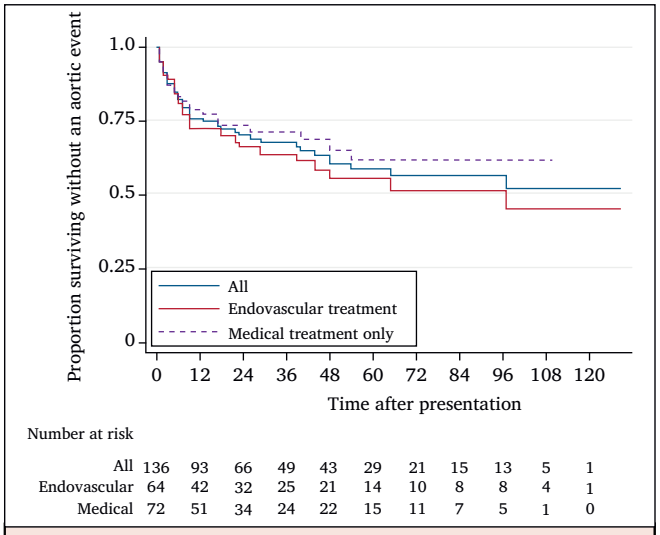

Figure 2. Kaplan Meier estimates of cumulative aortic event free survival after type B aortic dissection. The rates at one and five years were 76% and 58%, respectively.

increase in hazard of death of 26% for every five years' increase in age (HR 1.26 (1.08—1.46), $p = .003$) (Fig. S1). There was no association between cardiovascular parameters, CRP, eGFR, and the amount of hypertensive medication on admission and all cause mortality. There was an inverse relationship between the amount of antihypertensive medication on discharge and survival. An increase of one drug in the number of antihypertensive medications prescribed resulted in a decrease in hazard of death of 32% (HR 0.68 (0.53—0.88), $p = .013$) (Fig. S2). Aortic event free survival data are presented in Tables S6—S8. Taller patients were more likely to experience an aortic event during follow up; with a 1 cm increase in height associated with an increase in the hazard of experiencing an aortic event of 5% (HR 1.04 (1.01—1.05), $p = .005$) (Fig. S3). An increase in the diameter of the largest entry tear (Fig. S4), the diameter of the descending thoracic aorta (Fig. S5), and the diameter of the descending thoracic aorta false lumen (Fig. S6) were all significantly associated with an increased risk of aortic events during follow up. A 1 mm increase in both the size of the primary tear and the descending thoracic aorta was associated with a 7% increase in the hazard of an aortic event (HR 1.07 (1.02—1.11), $p = .003$) and ((HR 1.07 (1.02—1.11), $p = .002$), respectively) and a 5% increase for a 1 mm increase in the descending thoracic aorta false lumen (HR 1.05 (1.01—1.09), $p = .008$).

DISCUSSION

This study evaluates the outcomes of patients with type B aortic dissection treated endovascularly in the presence of complications and with best medical therapy in uncomplicated cases. The data at one and five years of follow up demonstrate that there is no difference in all cause survival and aortic event free survival between these two groups. Survival has traditionally been worse in patients with complicated compared with uncomplicated type B dissection, with survival figures in the region of 56.3—87% and 70.2—89%, respectively, at five years.[7] Better outcomes can be achieved in complicated patients by early identification and active management of complications, a low rate of procedural mortality and morbidity, and active management of patients during follow up using blood pressure control, surveillance imaging, and timely re-intervention when required.[1,3,8]

The aim of thoracic endovascular treatment is to cover the proximal entry tear to direct aortic blood flow towards the true lumen, to induce false lumen thrombosis and positive aortic remodelling, with the intended benefit of improving survival. It is thought that early endovascular treatment is likely to result in the maximum amount of aortic remodelling, because the aorta still has plasticity, and therefore result in the best long-term outcomes. Thoracic endovascular repair in the acute setting is associated with a relatively high risk of retrograde type A aortic dissection.[9] In the context of life threatening conditions such as rupture and visceral malperfusion, this risk is considered acceptable. However, in the absence of non-life threatening complications or when considering prophylactic treatment of type B aortic dissection, this risk must be carefully evaluated.

The data in this manuscript suggest that thoracic endovascular aortic repair is not able to prevent all aortic events during follow up, which is one of the primary aims of this treatment. Data from the INSTEAD trial also show a continued incidence of aortic events[4] at up to 52 months, the mean follow up in this series, and the consensus document has similar findings.[10] Techniques other than thoracic endovascular repair are available and should be considered for the management of patients with chronic type B aortic dissection to try to augment the effect of endovascular repair. These include extension of the aortic endografting into the abdominal segment using branched and fenestrated devices, placement of endovascular coils and plugs (candy plug) in the false lumen, occlusion of the false lumen by ballooning a stent graft in the true lumen to prevent retrograde flow (knickerbocker technique), and the STABILISE technique.[11—13]

A more in depth evaluation of the anatomy in these patients demonstrates that patients in the group with complications treated endovascularly tended to have a larger primary entry tear, a larger starting aortic diameter, and a narrower true lumen compared with the group treated with medical treatment alone. Patients in the medically treated group tended to have a larger true compared with false lumen. These features suggest that the pressure in the false lumen of patients with complications on presentation may be greater than in the group treated medically. Following TEVAR, thoracic but not abdominal aortic remodelling was seen, which is consistent with the results of other series.[14] Approximately one third of patients in the endovascular group did not have false lumen thrombosis in the thoracic aorta at two years, and this led to a progressive increase in thoracic aortic diameter during follow up. This residual flow in the false lumen in these patients may have influenced the incidence of aortic events during follow up.[15] The long follow up in this series allows a particular evaluation of false lumen thrombosis in type B aortic dissection over time.

Uncomplicated patients with type B aortic dissection typically follow a varied course following presentation. Some centres now advocate high frequency serial imaging (~3 CT angiograms) in the first 10 days following presentation to try to identify patients early who are likely to undergo rapid development of aortic complications. This series has shown that anatomical features such as a large proximal entry tear, a large descending thoracic aortic diameter, and a large diameter of the descending thoracic aortic false lumen on presentation are related to an increased risk of experiencing an aortic event during follow up. Taller patients were also more likely to experience an aortic event during follow up and may represent a group with undiagnosed connective tissue disease.[16] The effect of height on outcomes was significant, with a 10 cm increase in height resulting in a 48% increase in the risk of

experiencing an aortic event during follow up. Blood pressure control was shown to have a significant effect on all cause survival, with more active management related to better outcomes.

The International Registry of Aortic Dissection (IRAD) contains data collected from centres across the world and represents a unique resource to study the diagnosis and management of patients with aortic dissection.[17] One of the limitations of the registry, however, is the heterogeneity of the data, which reflects the local clinical management of patients and local CT image interpretation in each of the centres. One of the strengths of the series described in the current study is that patients were treated in a single centre, with a standardised procedure for clinical management and image interpretation. During the course of the study there was an increase in the level of clinical and surgical experience and this is one of the limitations of this study, also data on re-intervention and stent graft type were not collected. The image analysis was limited in that inter- and intra-observer reproducibility was not specifically performed and thrombosis was assumed to be present when contrast in the false lumen was absent; standard clinical acquisition protocols were used but these can over represent the amount of thrombosis if low flow states are present.

In conclusion, active management of patients with type B aortic dissection results in good long-term survival despite presenting features that have traditionally been associated with adverse outcomes. All patients require close lifetime surveillance as aortic events, even after endografting, continue to occur during follow up.

CONFLICT OF INTEREST

Stephan Haulon is a Consultant for Cook Medical and GE Healthcare.

FUNDING

None.

APPENDIX A. SUPPLEMENTARY DATA

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ejvs.2018.08.042.

REFERENCES

1 Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. 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. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). *Eur Heart J* 2014;**35**:2873–926.

2 Acosta S, Blomstrand D, Gottsater A. Epidemiology and long-term prognostic factors in acute type B aortic dissection. *Ann Vasc Surg* 2007;**21**:415–22.

3 Coady MA, Ikonomidis JS, Cheung AT, Matsumoto AH, Dake MD, Chaikof EL, et al. Surgical management of descending thoracic aortic disease: open and endovascular approaches: a scientific statement from the American Heart Association. *Circulation* 2010;**121**:2780–804.

4 Nienaber CA, Kische S, Rousseau H, Eggebrecht H, Rehders TC, Kundt G, et al. Endovascular repair of type B aortic dissection: long-term results of the randomized investigation of stent grafts in aortic dissection trial. *Circ Cardiovasc Interv* 2013;**6**:407–16.

5 van Bogerijen GH, Tolenaar JL, Rampoldi V, Moll FL, van Herwaarden JA, Jonker FH, et al. Predictors of aortic growth in uncomplicated type B aortic dissection. *J Vasc Surg* 2014;**59**:1134–43.

6 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.

7 Nienaber CA, Clough RE. Management of acute aortic dissection. *Lancet* 2015;**385**:800–11.

8 Riambau V, Bockler D, Brunkwall J, Cao P, Chiesa R, Coppi G, et al. Editor's choice – management of descending thoracic aorta diseases: clinical practice guidelines of the European Society for vascular Surgery (ESVS). *Eur J Vasc Endovasc Surg* 2017;**53**:4–52.

9 Canaud L, Ozdemir BA, Patterson BO, Holt PJ, Loftus IM, Thompson MM. Retrograde aortic dissection after thoracic endovascular aortic repair. *Ann Surg* 2014;**260**:389–95.

10 Fattori R, Cao P, De Rango P, Czerny M, Evangelista A, Nienaber C, et al. Interdisciplinary expert consensus document on management of type B aortic dissection. *J Am Coll Cardiol* 2013;**61**:1661–78.

11 Kolbel T, Carpenter SW, Lohrenz C, Tsilimparis N, Larena-Avellaneda A, Debus ES. Addressing persistent false lumen flow in chronic aortic dissection: the knickerbocker technique. *J Endovasc Ther* 2014;**21**:117–22.

12 Kolbel T, Lohrenz C, Kieback A, Diener H, Debus ES, Larena-Avellaneda A. Distal false lumen occlusion in aortic dissection with a homemade extra-large vascular plug: the candy-plug technique. *J Endovasc Ther* 2013;**20**:484–9.

13 Hofferberth SC, Nixon IK, Boston RC, McLachlan CS, Mossop PJ. Stent-assisted balloon-induced intimal disruption and relamination in aortic dissection repair: the STABILISE concept. *J Thorac Cardiovasc Surg* 2014;**147**:1240–5.

14 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.

15 Tsai TT, Evangelista A, Nienaber CA, Myrmel T, Meinhardt G, Cooper JV, et al. Partial thrombosis of the false lumen in patients with acute type B aortic dissection. *N Engl J Med* 2007;**357**:349–59.

16 Weinsaft JW, Devereux RB, Preiss LR, Feher A, Roman MJ, Basson CT, et al. Aortic dissection in patients with genetically mediated aneurysms: incidence and predictors in the GenTAC Registry. *J Am Coll Cardiol* 2016;**67**:2744–54.

17 Pape LA, Awais M, Woznicki EM, Suzuki T, Trimarchi S, Evangelista A, et al. Presentation, diagnosis, and outcomes of acute aortic dissection: 17-year trends from the International Registry of Acute Aortic Dissection. *J Am Coll Cardiol* 2015;**66**:350–8.