

1 **Long-term survival and risk analysis in 136 consecutive patients with type**
2 **B aortic dissection presenting to a single centre over an 11-year period**

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24

25 **What does this study add to the existing literature and how will it influence**
26 **future clinical practice?**

27 The management of patients with type B aortic dissection is complex. A number
28 of cases series have been published to try to better understand these patients but
29 they are limited by their small size. IRAD is a highly valuable resource but pools
30 data from a number of centres across the world and is therefore limited by the
31 heterogeneity of the data. This manuscript describes the findings of the largest
32 single centre series published to date and provides new insights into the
33 management of these complex patients.

34

35 **Abstract**

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

40

41 *Design:* Retrospective analysis of prospectively collected clinical and CT imaging
42 data.

43

44 *Materials:* 136 consecutive patients with acute type B aortic dissection were
45 included in the study over an 11-year period.

46

47 *Methods:* Characteristics of patients receiving endovascular (complicated) or
48 medical treatment (uncomplicated) were compared. Kaplan Meier estimators
49 were used to estimate cumulative overall survival and survival free of aortic
50 events. Factors associated with overall and aortic event free survival were also
51 explored using Cox-proportional hazards models.

52

53 *Results:* The mean follow-up was 51 months (1-132), during which time 33
54 deaths and 48 aortic events occurred. At one year and five years overall survival
55 was 94.0% and 74.8% respectively and freedom from aortic events was 75.6%
56 and 58.7%. There was no difference in all cause survival and aortic event-free
57 survival at one and five years between the patients treated endovascularly and
58 those receiving medical treatment alone. Risk analysis for aortic events
59 demonstrated the maximum size of the proximal entry tear, the maximum

60 thoracic aortic diameter and the thoracic aortic false lumen maximum diameter
61 to have a significant effect on the incidence of aortic events.

62

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

67

68 *Keywords:* Survival, risk analysis, aortic dissection

69

70 **Introduction**

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

77

78 Previous series have however demonstrated that even in the presence of good
79 blood pressure control survival is poor, with only 50-70% of patients alive at 5
80 years, with a delayed expansion of the false lumen in 20-50% of patients at 4-
81 years.(2-4) Many of the deaths that occur during follow up are aortic related.

82

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

90

91 There is growing interest in early endovascular treatment of patients with type B
92 aortic dissection to prevent aortic events during follow up and therefore improve
93 survival. It is hoped that treating patients early will produce the best outcomes

94 because the aorta has plasticity and is therefore likely to undergo positive aortic
95 remodelling following stent graft insertion.(6)

96

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

103

104

105 **Materials and methods**

106 *Study design*

107 136 consecutive patients with acute type B aortic dissection were included in the
108 study over an eleven-year period. All patients were managed in a high
109 dependency setting with medical management implemented by medical
110 specialists trained in the management of hypertension. Patients that presented
111 with evidence of aortic rupture, end-organ ischaemia or on-going pain despite
112 good blood pressure control were defined as complicated and treated
113 endovascularly, those without these features were treated with best medical
114 therapy. Patients were excluded if the aortic dissection was secondary to trauma
115 or iatrogenic injury. Patients were categorised into groups based upon their
116 treatment. All patients were followed up by both a cardiologist with a special
117 interest in aortic dissection and hypertension and a vascular surgeon.

118

119 *Endovascular repair*

120 Thoracic endovascular repair was performed in a standard operating room with
121 mobile C-arm or in a hybrid operating room using percutaneous vascular access
122 whenever possible. Left common carotid to left subclavian artery (LSCA) bypass
123 or transposition was considered if the stent graft covered the origin of the LSCA.
124 Absolute indications for LSCA revascularisation were: left internal mammary
125 artery bypass graft; diminutive, atretic or absent right vertebral artery; left arm
126 arterio-venous fistula for haemodialysis; patent left axillo-femoral bypass graft;
127 and dominant left vertebral artery. These procedures were performed prior to,
128 or at the time of the TEVAR procedure, depending upon the level of urgency of
129 the case. The stent graft was sized to the proximal un-dissected aorta with 10-

130 15% oversizing. In cases of rupture the stent grafts were positioned to cover the
131 thoracic aorta from proximal to the primary entry tear to the level of the coeliac
132 trunk.

133

134 *Best medical therapy*

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

141

142 *Image analysis*

143 Computed tomography (CT) angiographic images of the aorta were acquired on
144 presentation and during follow-up using an iodinated contrast agent. All images
145 were analysed on a dedicated Aquarius iNtuition workstation (TeraRecon, Foster
146 City, Calif) by two vascular surgeons experienced in the endograft planning for
147 aortic dissection. The arterial phase images, reconstructed to ≤ 1 mm, were
148 evaluated and the number of entry tears, the size of the primary entry tear, the
149 length of the dissection, the amount of thrombosis in the false lumen and the
150 dimensions of the aorta and the true and false lumen were recorded. The true
151 lumen was identified as the lumen continuous with the proximal undissected
152 aorta. The maximum diameter of the primary entry tear was measured using
153 multi-planar reformatted (MPR) images, false lumen thrombosis was assumed to
154 be present when there was absence of contrast enhancement and volumes were

155 calculated by segmentation and summation over contiguous slices. The
156 following were recorded: i. the maximum aortic diameter; ii. the true and false
157 lumen diameters in the thoracic aorta at the level of the inferior pulmonary vein;
158 and iii. the true and false lumen diameters in the abdominal section at the level of
159 the IMA (or at the mid-point of the third lumbar vertebra if the IMA could not be
160 seen). All diameter measurements were made using MPR images to ensure
161 precision.

162

163 *Statistical analysis*

164 Characteristics of patients receiving endovascular or medical treatment alone
165 were summarised within each group and compared using Mann-Whitney, chi-
166 squared or fisher's exact test, as appropriate. Kaplan Meier estimators were used
167 to estimate cumulative overall survival and survival free of aortic events with
168 95% confidence intervals at one and 5 years after admission. Aortic events were
169 rupture, extension of the dissection and further intervention. Kaplan Meier
170 estimators were also calculated within subgroups of patients. Factors associated
171 with overall and aortic event free survival were also explored using Cox-
172 proportional hazards models. Due to small sample sizes, for categorical data the
173 estimation of one and five year survival rates and the application of Cox models
174 were restricted to variables with at least 20% of patients in each subgroup.
175 Where Cox models indicated a statistically significant association Kaplan Meier
176 curves were plotted. For continuous variables cut points were selected that
177 divided the data into three approximately equal sized groups and Kaplan Meier
178 curves estimates for each of these groups. Analysis was conducted using STATA
179 13MP and p-values <0.05 considered statistically significant.

180 **Results**

181 *Baseline characteristics*

182 The baseline characteristics for the 136 patients are summarised in Table 1. The
183 average age at admission was 61.7 years and the majority of the cohort were
184 male (77.2%). Seventy-one per cent of the cohort had hypertension, 9.6% had
185 diabetes and 5.9% had known connective tissue disease. Sixty-four patients
186 presented with complications and were treated endovascularly. The most
187 common complication was end-organ ischaemia (n=45); aortic rupture was
188 present in 17 cases and 5 patients were treated for on-going pain despite good
189 blood pressure control. Three patients had both rupture and end-organ
190 ischaemia. The patients that were treated endovascularly were significantly
191 younger than the patients treated with medical treatment alone (mean age 58.5
192 versus 64.5 years). β -blocker was the most commonly used antihypertensive
193 agent to treat aortic dissection in both treatment groups [Suppl. table 1].
194 The incidence of in-hospital events were: acute coronary syndrome 3.7%
195 (endovascular n=3, medical treatment alone n=2), neurological complications
196 7.4% (endovascular n=7, medical treatment alone n=3), dialysis 2.9%
197 (endovascular n=3, medical treatment alone n=1), pulmonary infection 11.8%
198 (endovascular n=11, medical treatment alone n=5).

199

200 *Anatomical features*

201 The anatomical features of the cohort are shown in Table 2 and Suppl. Table 2. At
202 presentation the maximum aortic diameter was greater in the endovascularly
203 treated group compared with the group treated with medical treatment alone. In
204 both groups the dissection tended to involve both the thoracic and abdominal

205 aorta (endovascular group 93%, medical treatment only group 81.7%). In the
206 endovascularly treated group the true lumen tended to be smaller than the false
207 lumen, whereas in the group that received medical treatment only the true
208 lumen was greater than the false lumen. In both treatment groups a large
209 proportion of the patients had a patent false lumen and there was no difference
210 in the number of entry tears. The size of the largest entry tear was significantly
211 greater in the endovascular treatment group.

212 At 2-years follow-up there was an average increase in the true lumen diameter of
213 the thoracic aorta of 8mm in the endovascular treatment group with no
214 corresponding increase in the true lumen diameter of the abdominal segment in
215 this group. The majority of the patients in the medically treated group had
216 partial (51.7%) or complete (34.5%) thoracic aortic false lumen thrombosis
217 whereas in the endovascularly treated group approximately one third of patients
218 had patency of the false lumen. At 5-years the average size of the false lumen in
219 the endovascularly treated group had increased compared to the 2-year data and
220 only 27.3% of patients had complete thrombosis of the thoracic aortic false
221 lumen following endovascular treatment.

222

223 *Survival analysis*

224 The mean follow up in the cohort was 51 (1-132) months; thirty-three deaths, 8
225 aortic-related, 10 cardiac, 10 cancer-related and 5 other occurred, and 48 aortic
226 events were recorded. The 30-day all cause survival and aortic event-free
227 survival were 98.5% (94.3-99.6%) and 94.8% (89.4-97.5%) respectively.
228 Cumulative survival was 94.0% (95% CI 88.4 - 97.0%) at one year and 74.8%
229 (64.5-82.5%) at five years [Figure 1]. The aortic event-free survival at one year

230 and at five years was 75.6% (67.3-82.1%) and 58.7% (48.1-67.8%) respectively
231 [Figure 2].

232 There was no difference in all cause survival at one and five years between the
233 patients treated endovascularly and those receiving medical treatment alone
234 (HR=0.99 (0.49-2.02), p=0.996). There was no difference in aortic event-free
235 survival at one and five years between the patients treated endovascularly and
236 those receiving medical treatment alone (HR=1.33 (0.78-2.34), p=0.329).

237

238 *Factors associated with overall and aortic event free survival*

239 Patient characteristics, cardiovascular risk factors and imaging features, and
240 their association with all-cause survival, are summarised in supplementary
241 tables 3, 4 and 5 respectively. Age was significantly associated with all cause
242 survival with an increase in hazard of death of 26% for every 5 years increase in
243 age (HR=1.26 (1.08-1.46), p=0.003) [Suppl. Figure 1]. There was no association
244 between cardiovascular parameters, CRP, eGFR and the amount of hypertensive
245 medication on admission and all cause mortality. There was an inverse
246 relationship between the amount of anti-hypertensive medication on discharge
247 and survival. An increase of one drug in the number of antihypertensive
248 medications prescribed resulted in a decrease in hazard of death of 32%
249 (HR=0.68 (0.53-0.88), p=0.013) [Suppl. Figure 2]. Aortic event-free survival data
250 is presented in supplementary tables 6, 7 and 8. Taller patients were more likely
251 to experience an aortic event during follow up; with a one centimetre increase in
252 height associated with an increase in the hazard of experiencing an aortic event
253 of 5% (HR=1.04 (1.01-1.05), p=0.005) [Suppl. Figure 3]. An increase in the
254 diameter of the largest entry tear [Suppl. Figure 4], the diameter of the

255 descending thoracic aorta [Suppl. Figure 5] and the diameter of the descending
256 thoracic aorta false lumen [Suppl. Figure 6] were all significantly associated with
257 an increased hazard of aortic events during follow up. A one mm increase in both
258 the size of the primary tear and the descending thoracic aorta was associated
259 with a 7% increase in the hazard of an aortic event ((HR=1.07 (1.02-1.11),
260 p=0.003) and (HR=1.07 (1.02-1.11), p=0.002), respectively) and a 5% increase
261 for a 1mm increase in the descending thoracic aorta false lumen (HR=1.05 (1.01-
262 1.09), p=0.008).

263

264

265

266 **Discussion**

267 This study evaluates the outcomes of patients with type B aortic dissection
268 treated endovascularly in the presence of complications and with best medical
269 therapy in uncomplicated cases. The data at 1- and 5-years of follow-up
270 demonstrate that there is no difference in all cause survival and aortic event-free
271 survival between these two groups. Survival has traditionally been worse in
272 patients with complicated compared with uncomplicated type B dissection, with
273 survival figures in the region of 56.3-87% and 70.2-89% respectively at 5
274 years.(7) Better outcomes can be achieved in complicated patients by early
275 identification and active management of complications, a low rate of procedural
276 mortality and morbidity and active management of patients during follow using
277 blood pressure control, surveillance imaging and timely re-intervention when
278 required.(1, 3, 8)

279

280 The aim of thoracic endovascular treatment is to cover the proximal entry tear to
281 direct aortic blood flow towards the true lumen, to induce false lumen
282 thrombosis and positive aortic remodelling, with the intended benefit of
283 improving survival. It is thought that early endovascular treatment is likely to
284 result in the maximum amount of aortic remodelling, because the aorta still has
285 plasticity, and therefore result in the best long-term outcomes. Thoracic
286 endovascular repair in the acute setting is associated with a relatively high risk
287 of retrograde type A aortic dissection.(9) In the context of life-threatening
288 conditions such as rupture and visceral malperfusion this risk is considered
289 acceptable. However in the absence of non-life threatening complications or

290 when considering prophylactic treatment of type B aortic dissection this risk
291 must be carefully evaluated.

292

293 The data in this manuscript suggest that thoracic endovascular aortic repair is
294 not able to prevent all aortic events during follow up, which is one of the primary
295 aims of this treatment. Data from the INSTEAD trial also shows at up to 52
296 months, the mean follow-up in this series, a continued incidence of aortic events
297 (4) and the consensus document also has similar findings.(4, 10, Nienaber, 2013
298 #8090) Techniques other than thoracic endovascular repair are available and
299 should be considered in the management of patients with chronic type B aortic
300 dissection to try to augment the effect of endovascular repair. These include
301 extension of the aortic endografting into the abdominal segment using branched
302 and fenestrated devices, placement of endovascular coils and plugs (candy-plug)
303 in the false lumen, occlusion of the false lumen by ballooning a stent graft in the
304 true lumen to prevent retrograde flow (knickerbocker technique) and the
305 STABILISE technique.(11-13)

306

307 A more in depth evaluation of the anatomy in these patients demonstrates that
308 patients in the group with complications treated endovascularly tended to have a
309 larger primary entry tear, a larger starting aortic diameter, and a narrower true
310 lumen compared with the group treated with medical treatment alone. Patients
311 in the medically treated group tended to have a larger true compared with false
312 lumen. These features suggest that the pressure in the false lumen of patients
313 with complications on presentation may be greater than in the group treated
314 medically. Following TEVAR thoracic but not abdominal aortic remodelling was

315 seen, which is consistent with the results of other series.(14) Approximately one
316 third of patients in the endovascular group did not have false lumen thrombosis
317 in the thoracic aorta at 2 years, and this led to a progressive increase in thoracic
318 aortic diameter during follow-up. This residual flow in the false lumen in these
319 patients may have influenced the incidence of aortic events during follow-
320 up.(15) The long follow-up in this series allows a particular evaluation of false
321 lumen thrombosis in type B aortic dissection the over time.

322

323 Uncomplicated patients with type B aortic dissection typically follow a varied
324 course following presentation. Some centres now advocate high frequency serial
325 imaging (~3 CT angiograms) in the first ten days following presentation to try to
326 identify patients early that are likely to undergo rapid development of aortic
327 complications. In this series we have shown that anatomic features such as a
328 large proximal entry tear, a large descending thoracic aortic diameter and a large
329 diameter of the descending thoracic aortic false lumen on presentation are
330 related to an increased hazard of experiencing an aortic event during follow-up.
331 Taller patients were also more likely to experience an aortic event during follow
332 up and may represent a group with undiagnosed connective tissue disease.(16)
333 The effect of height on outcomes was significant, with a 10cm increase in height
334 resulting in a 48% increase in the hazard of experiencing an aortic event during
335 follow up. Blood pressure control was shown to have a significant effect on all-
336 cause survival, with more active management related to better outcomes.

337

338 The International Registry of Aortic Dissection (IRAD) contains data collected
339 from centres across the world and represents a unique resource to study the

340 diagnosis and management of patients with aortic dissection.(17) One of the
341 limitations of the registry however is the heterogeneity of the data, which
342 reflects the local clinical management of patients and local CT image
343 interpretation in each of the centres. One of the strengths of the series described
344 in the current manuscript is that patients were treated in a single centre, with a
345 standardised procedure for clinical management and image interpretation.
346 During the course of the study there was an increase in the level of clinical and
347 surgical experience and this is one of the limitations of this study, also data on re-
348 intervention and stent graft type was not collected. The image analysis was
349 limited in that inter- and intra-observer reproducibility was not specifically
350 performed and thrombosis was assumed to be present when contrast in the false
351 lumen was absent; standard clinical acquisition protocols were used but these
352 can over-represent the amount of thrombosis if low-flow states are present.
353
354 In conclusion, active management of patients with type B aortic dissection
355 results in good long-term survival despite presenting features that have
356 traditionally been associated with adverse outcomes. All patients require close
357 lifetime surveillance as aortic events, even after endografting, continue to occur
358 during follow-up.
359

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422 **Tables**

	All	Endovascular treatment	Medical treatment only	p- value
Total	136	64	72	
Age, mean(sd)	61.7(13.2)	58.5(12.7)	64.5(13.1)	0.008
Male sex, %	77.2	79.7	75.0	0.515
Height (cm), mean(sd)	173.5(9.7)	174.6(8.9)	172.6(10.3)	0.264
Hypertension, %	71.3	75.0	68.1	0.371
Diabetes, %	9.6	9.9	9.4	0.924
Dyslipidaemia, %	25.0	17.2	31.9	0.047
BMI, median(IQR)	27(24.2- 31.0)	27.0(24.0- 31.4)	26.9(24.4- 29.7)	0.945
Coronary artery disease, %	5.9	4.7	7.0	0.721
Co-existing AAA, %	9.6	10.9	8.5	0.625
Previously treated AAA, %	4.4	0	8.5	0.029
Renal insufficiency, %	0	0	0	
Connective tissue disease, %	5.9	9.4	2.8	0.149
Family history of Marfan, %	3.7	4.7	2.8	0.668

423

424

425

426

Table 1.
Patient characteristics

	All	Endovascular treatment	Medical treatment only	p-value
<i>Presentation</i>				
Maximum aortic diameter, median(IQR)	38(35-43)	41(36-45)	37(34-40)	0.003
Dissection involving thoracic and abdominal aorta, %	87.2	93.0	81.7	0.096
Total diameter of the thoracic aorta, median(IQR)	37(34-42)	39(34-42)	36(32.5-38)	0.006
True lumen diameter in the thoracic aorta, median(IQR)	18(14-23)	18(15-24)	19.5(12.5-23)	0.900
False lumen diameter in the thoracic aorta, median(IQR)	18(12-26)	20(14-28)	17(10-23)	0.060
False lumen status, %				
Patent	52.6	59.7	45.8	0.105
Partially thrombosed	38.8	36.8	40.7	
Completely thrombosed	8.6	3.5	13.6	
<i>2 years</i>				
Maximum aortic diameter, median(IQR)	40.5(36-44.5)	42(38-47)	39(35-42)	0.088
Total diameter of the thoracic aorta, median(IQR)	40(36-43)	41(36-44)	38(35-41)	0.067
True lumen diameter in the thoracic aorta, median(IQR)	25(15-30)	25.5(16-30)	21(13-28)	0.180
False lumen diameter in the thoracic aorta, median(IQR)	18(8-27)	18.5(8-28)	18(8-26)	0.665
False lumen status, %				
Patent	24.3	31.7	13.8	0.184
Partially thrombosed	41.4	34.2	51.7	
Completely thrombosed	34.3	34.2	34.5	
<i>5 years</i>				
Maximum aortic diameter, median(IQR)	40(37-47)	42(38-48)	37.5(33-41.5)	0.045
Total diameter of the thoracic aorta, median(IQR)	40.5(36-44.5)	42(38-47)	39(35-42)	0.088
True lumen diameter in the thoracic aorta, median(IQR)	23(16-31)	20(16-32)	28(18-30)	0.600
False lumen diameter in the thoracic aorta, median(IQR)	21(8.5-28)	23(12-28)	14(4-34)	0.316
False lumen status, %				
Patent	28.6	39.4	6.3	0.049
Partially thrombosed	38.8	33.3	50.0	
Completely thrombosed	32.7	27.3	43.8	

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Table 2.
Anatomical features of the patient cohort

432 **Figure legends**

433 *Figure 1:* Kaplan Meier estimate demonstrating 94.4% and 75.5% cumulative
434 overall survival at one and five years respectively

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436 *Figure 2:* The aortic event-free survival at one year and at five years was 75.5%
437 and 58.0% respectively

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