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1 **Malperfusions in acute type B aortic dissection – predictors of outcomes**

2

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18

19

20 **Abstract**

21 *Context:* Acute type B aortic dissection (AoD) with malperfusion is a rare and complex  
22 disease. In this specific setting, invasive treatment is generally required to improve aortic  
23 branch vessels flow. Thoracic stentgrafting (TEVAR) of the proximal descending thoracic  
24 aorta to cover/exclude the proximal intimal entry tear would promote resolution of the  
25 dynamic component (the most prevalent) of the malperfusion conflict by redirecting the  
26 flow within the true lumen.

27

28 *Aim:* studying outcomes of patients undergoing TEVAR for acute (<14 days) type B AoD  
29 complicated with malperfusion; and to depict any pre- and intraoperative predictors that  
30 could affect the prognosis of those patients.

31

32 *Method:* from March 2005 to January 2016, all patients treated with TEVAR for acute type B  
33 AoD with malperfusion in 2 European high volume aortic centres were retrospectively  
34 studied. Pre-, intra and postoperative details were collected. Preoperative computed  
35 tomography angiogram was re-analysed on a dedicated 3D workstation (Aquarius  
36 inTuitionViewer, Terarecon). 30-day malperfusion-related complication and mortality rates  
37 were determined. Logistic regression model was used to assess pre- and intraoperative  
38 factors affecting postoperative outcomes; in particular, details on the mechanisms of  
39 malperfusion and organ(s) involved were entered into the model and tested.

40

41 *Results:* Total of 41 patients (60 [IQR 51-68.5] years old; 78% male) have been included in  
42 the analysis. Patients were mostly (68.3%) affected by only 1 malperfusion syndrome, with  
43 renal ischemia being the most frequent (53.6%). The median length of aortic coverage was  
44 197mm [IQR 157-209]. Additional visceral/renal/iliac stentings were performed after stent  
45 graft implantation for 25 branches in 17 patients (41%).

46 The 30-day mortality rate was 17.1%. All but 2 early deaths were related to malperfusion.

47 The number of malperfusion syndromes was the only independent factor associated to  
48 increasing 30-day malperfusion-related complications or death (3 vs 1; HR=30.3 (p=.001) and  
49 3 vs 2; HR=9.9 (p=.004)).

50

51 Conclusion: Prognosis of patients with acute type B AoD complicated initially with  
52 malperfusion syndrome(s) is severe, especially if several territories are ischemic. Early  
53 identification of those complications can be life-saving but still lacks hard criteria.  
54

55 **Introduction**

56 Acute type B aortic dissection might expose patient to rapid death when at the onset  
57 malperfusion(s) and/or aortic rupture occur. Endovascular repair is now admitted as the  
58 first-line treatment for aortic thoracic diseases lowering postoperative morbi-mortality  
59 rates. Critical rise of pressure in the false aortic lumen is the starting point of those 2  
60 complications and the equipoise for excluding the proximal tear with stentgraft. Situations at  
61 risks of complications in the acute setting of aortic dissection need to be properly depicted  
62 to select patients who can benefit of immediate appropriate invasive treatments. 4 main  
63 situations are generally admitted to conduct toward rapid invasive treatment: rupture,  
64 and/or malperfusion, and/or refractory hypertension and/or intractable pain.  
65 Malperfusion syndrome at onset is difficult to be established with certainty, as patients are  
66 painful. In this setting, malperfusion syndrome might be over or in the opposite  
67 underestimated that would result in inadequate potentially treatment decision making. Its  
68 diagnosis is usually based on a beam of arguments such as clinical symptoms, imaging study  
69 and biological assessment.  
70 This work aims at analysing outcomes and determining whether any finite morphological  
71 characteristics of the dissected aorta and/or clinical elements and/or any elements of the  
72 endovascular procedure would predict outcomes in patients with acute type B presenting  
73 with malperfusion syndrome(s) at the onset.

74

## 75 **Material & Methods**

76 We retrospectively included consecutive patients that received thoracic stentgraft (TEVAR)  
77 for acute (<14d) type B aortic dissection complicated by malperfusion between March 2005  
78 and December 2015 in Lille (CHU Lille, France) and Malmö (Skåne University Hospital,  
79 Sweden). Patients with retrograde arch extension of the dissection, complete aortic rupture  
80 or with sub-acute or chronic aortic dissection were excluded. Moreover, only patients with  
81 available complete CTA imaging at symptoms onset have been included.

82 The preoperative evaluation of patients was made by an interdisciplinary medical team  
83 (vascular surgeons, angiologists or cardiologists and anesthesiologists) before TEVAR.

84 Patients underwent computed tomography angiogram (CTA) with thin axial reconstructions  
85 on arterial phase ( $\leq 1$  mm) preoperatively. Postoperatively CTA was done at least yearly.  
86 Radiological evidence of malperfusion (dynamic, static or both) was assumed whenever  
87 compromised perfusion to the aortic side branches was seen. The distinction was made  
88 between isolated radiological signs and/or clinical malperfusion symptoms (pain,  
89 paraparesis/paraplegia, oliguria/anuria, intestinal bleeding) and/or laboratory evidence of  
90 organ hypoperfusion were present (significant rise in creatine and/or lactic acid serum level).

91 For the purpose of the analysis, the each of the considered malperfused territories was  
92 assumed as a separate malperfusion syndrome.

93 Demographics, medical history and initial clinical presentation including the number of  
94 organ affected by malperfusion syndromes were retrospectively reviewed.

95 CTA morphological details of the aortic dissection at initial presentation (diameters, lengths,  
96 characteristics of the entry tear(s), aortic lumens status, mechanisms of aortic branch  
97 perfusion as described by Williams et al. [1]) were analysed on a 3D-workstation (Aquarius,  
98 Terarecon) from an arterial- and a late-phases CTA.

99 Our strategy of treatment enclosed a first stage of proximal entry tear exclusion with aortic  
100 stentgraft; then, an aortogram is performed to evaluate the perfusion of visceral / renal  
101 arteries. In case of unsatisfying flows within one or several aortic collaterals an additional  
102 distal stentgraft was then deployed. A second aortogram is then completed and the decision  
103 of selective stenting might be taken into consideration if a doubt remained on the quality of  
104 the organ perfusion.

105 Procedural details and postoperative elements according to the SVS reporting standards [2]  
106 were analysed and included early and late complications, freedom-from-reintervention and  
107 overall survival according to the Kaplan-Meier method. Complications were considered  
108 related to malperfusion syndrome when consequences of organ failure were in the context  
109 of the presence of conflict(s) of perfusion visualized on the pre- or post-operative imaging  
110 exam and/or in the presence of clinical symptoms.

111 The present study was approved by the French national data protection commission  
112 (Commission Nationale de l'Informatique et des Libertés; reference: DEC2015-19).

113

#### 114 **Statistical Analysis**

115 All data were analysed using SAS statistical software version 9.4 (SAS Institute Inc, Cary, NC).  
116 Graphs representation has been achieved using R 3.0.1 software (the R foundation, Vienna,  
117 Austria) and Graphpad Prism v7.0 (Austria). Quantitative variables are expressed as mean  $\pm$   
118 SD in case of normal distribution or median (interquartile range) otherwise, and categorical  
119 variables are expressed as number (percentage). Normality of distributions was checked  
120 using the Shapiro-Wilk test.

121 The Cox proportional hazard model was used to depict any correlation between  
122 preoperative (demographics / anatomic) and procedural variables of patients and a

123 composite criterion that includes 30-day malperfusion-related complications and 30-day  
124 death. Variables having a significant test in univariate analysis ( $<0.20$ ) were introduced in the  
125 model and tested.  $P < 0.05$  was considered significant.

126

127



128 **Results**

129 *Population / clinical presentation at onset*

130 From march 2005 to december 2015, 54 patients underwent TEVAR in Malmö and Lille for  
131 acute type B aortic dissection including one or several malperfusion syndromes.

132 Over the same period of time 357 patients in Lille and 334 patients in Malmö received  
133 thoracic stentgrafts for thoracic aortic disease. Preoperative CTA was missing for 6 patients.

134 In 7 more patients, selective stenting was performed prior to TEVAR within first hours of  
135 aortic dissection occurrence and thus not included in this present study. In total, 41 patients  
136 were included in our analysis.

137 The Table 1 presents the demographic features of our population at onset. Median age was  
138 60 [IQR 51-68.5] with 78% being males. Most of the patients presented with symptoms only  
139 from one of the territories (68.3%) with renal ischemia being the most frequent (in 53.6%).  
140 Isolated spinal cord malperfusion was present in 5 patients (12.2%) (Table 2). The median  
141 interval between the onset of symptoms and TEVAR was 1 day [IQR; 0-3].

142

143 *Morphological data at initial onset*

144 Median of maximal transaortic diameter within the descending thoracic aorta was 37mm

145 [IQR 37-40] with a median true lumen diameter of 17mm [IQR 11-19]. Diameters of the

146 aorta at different levels along the descending thoracic and abdominal aorta are depicted in

147 Table 3. In all patients, the dissection extended beyond the diaphragm and further down to

148 the abdominal aorta in 95.2% of patients. The dissection originated in the proximity of the

149 left subclavian artery origin in all patients with a main primary proximal entry tear median

150 length of 9mm [IQR 5-30].

151 None of patients got a fully thrombosed false lumen at the thoracic level at onset (Table 4).

152 Compromised branch vessel perfusion on the preoperative CTA was not systematically  
153 associated with clinical malperfusion syndrome. There were radiological signs of  
154 malperfusion in all but 4 patients. The mechanisms of the conflict of perfusion for visceral,  
155 renal and iliac arteries are depicted on the Figure 1 (a & b).

156

#### 157 *TEVAR and adjuncts at the initial procedure (Table 5)*

158 All patients received at least one thoracic stent graft to exclude the proximal entry tear  
159 within the true lumen. All stentgrafts but one were manufactured by CookMedical  
160 (Bloomington, IND., USA); there were 5 Zenith Alpha, 36 ZTEG-2 (P or PT, GianTurco design)  
161 and 1 Tag (WLGore, Flagstaff, CA., USA). For 15 out of 41 (37%) the proximal component got  
162 degressive diameters. The median length of aortic coverage was 197mm [IQR 157-209] and  
163 10 patients (24.4%) received 2 components. In 78.2% of patients, the LSA was intentionally  
164 covered with 12.5% undergoing LSA revascularization at the same time.

165 Additional selective stents were deployed after stent graft implantation in 25 aortic  
166 branches for 17 patients. One patient had intimal flap fenestration after TEVAR and selective  
167 stenting for a persistent conflict of perfusion on the left renal artery.

168

#### 169 *Early outcomes*

170 The 30-day mortality rate was 17.1% (7/41). The causes of death were 3 multiple organ  
171 failures, 2 bowel infarctions, 1 haemorrhagic stroke and 1 aortic rupture. Among the  
172 patients who survived, 3 of them underwent bowel resection without any additional visceral  
173 revascularisation, 2 required permanent dialysis, 1 developed a massive stroke with residual  
174 hemiplegia and 1 with paraparesis. The median length of ICU stay was 2 [IQR 2-4] days.

175 Partial recovery of spinal cord ischemia symptoms occurred in 2 patients, whilst for the 3  
176 others a complete resolution was noted. 1 patient presenting initially with left limb ischemia  
177 developed lower limbs paraparesis postoperatively.

178

#### 179 *Follow up*

180 During a median follow-up of 48 [IQR 33 – 73] months there were no CTA signs of progress  
181 of stenosis in the aortic branches. Two patients reported lower limb claudication after  
182 discharge that remained stable during follow up. 5 other patients presented with clinical  
183 symptom attributable to chronic malperfusion (3 renal arteries and 2 SMA).

184 8 patients underwent secondary interventions during follow-up: 5 for the above-mentioned  
185 reasons, 2 for abdominal aortic dilatation, and 1 for a proximal type 1 endoleak.

186 According to Kaplan-Meier method, the survival rate was 80.0% [95%CI; 70.5-96.0] at 6 and  
187 12 months and 77.4% [95%CI; 67.3-93.8] at 2 years (Figure 2).

188

#### 189 *Predictive model*

190 From this analysis, only the number of malperfusion syndromes at onset seems to affect  
191 early outcomes of our patients; patients presented with 3 malperfusion syndromes at onset  
192 appeared to be 30.3 [3.8-242.6; p=.001] times more at risk for any 30-day major  
193 complication and/or death compared to patients with only 1 or 2 malperfusion syndromes.

194

195 **Discussion**

196 This multicentric study demonstrates that the number of malperfusion syndromes at onset  
197 in the setting of acute type B aortic dissection directly influenced clinical outcome.

198 Malperfusion syndromes involving lower limb and spinal cord are usually easy to detect  
199 immediately, while the clinical significance of the malperfusion of the visceral and renal  
200 arteries could be delayed. The malperfusion syndromes that affect the digestive tract  
201 however represent the 1<sup>st</sup> cause of death and/or major complication in the setting of acute  
202 aortic dissection; with early death rates up to 62% [3]. The diagnosis of malperfusion  
203 syndrome is somehow difficult in this context of patients with intractable pain, which in our  
204 series was present in all cases; which probably explains why the presence of that intractable  
205 pain itself at the onset is associated to worse outcomes independently to the presence of  
206 other concurrent complications [3].

207 This symptom is the most frequent depicted in the acute phase of aortic dissection and may  
208 improperly mask or overestimate the presence of other life-threatening complications that  
209 make considering immediate invasive treatment [4].

210 In our series, the 30-day death rate was 17.1%, which is in concordance with previous  
211 reports from 9 to 21% [5-8], even if the proportion of patients with malperfusion in most of  
212 those series was lower than in the present one. In all but 2 cases, the early deaths were in  
213 relation to visceral malperfusion (aortic rupture and stroke). This underlines the need for an  
214 early diagnosis and revascularisation when there is a visceral ischemia.

215 In this cohort, patients received primarily thoracic stent graft to exclude/cover the proximal  
216 primary entry tear and redirect the flow into the true lumen from false lumen and thereby  
217 relieving the perfusion conflicts. This strategy was first promoted by Dake M et al. in his

218 original publication in 1999 [9] and has become the first-line strategy to treat patients in the  
219 setting of acute type B aortic dissection with malperfusion according to the IRAD data [3].  
220 Several studies have shown that TEVAR requires quite frequently adjuncts to deal with  
221 persistent malperfusion conflict distally [5-8]. However, the effect of the original stentgraft is  
222 determined by several factors. The presence of any secondary intimal tears below the initial  
223 stent graft, may lead to persistent pressurisation of the distal false lumen and the continuing  
224 of the conflict of perfusion. In this case, distal stent-graft extension down to the celiac trunk  
225 must be considered if the tear is within the descending thoracic aorta, but this needs to be  
226 balanced with the risk of spinal cord ischemia associated with more extensive coverage of  
227 the aorta. Some authors recently suggested the potential benefit of routine fenestration in  
228 the distal aorta to prevent or improve spinal cord malperfusion in patients where stent graft  
229 within the descending thoracic aorta would be deployed with lots of intercostal arteries seen  
230 arising from the false lumen. Moreover, and contrary to what has been generally assumed,  
231 the authors suggest this would result in largest false lumen reduction and thus lower risk of  
232 aneurysmal degeneration of the distal aorta [10]. The understanding of the mechanisms of  
233 spinal cord malperfusion in this setting remain somehow unclear and it may feel premature  
234 to rely on single preoperative CTA analysis at this stage to decide on extensive coverage  
235 followed by fenestration to limit any spinal cord ischemia. Until further data is available, we  
236 increase the extent of aortic coverage intraoperatively in a gradual way depending on the  
237 angiographic result of the placement of the first stentgraft combined with the preoperative  
238 location of the intimal tears. On the contrary we tend to have systematic long aortic  
239 coverage when aortic rupture occurs.

240 Few authors in literature reported the median length of coverage of the descending thoracic  
241 aorta in this specific setting of aortic dissection complicated with malperfusion. Dake in his

242 original paper had a median length of coverage of 69mm whereas in our series it was  
243 197mm and in other works it was 195mm [5,9]. This intermediate length of aortic coverage  
244 could also prevent the occurrence of stent graft induced new distal entry tear (SINE) which is  
245 more common when aortic stent graft is <145mm and is associated to higher short-term  
246 mortality rate [11,12].

247

248 Selective stenting and/or fenestration of the flap are usually proposed when malperfusion  
249 conflict persists after the implantation of the thoracic stent grafts. Up to 41% of our patients  
250 required additional selective stenting of any of the aortic branches (visceral, renal and/or  
251 iliac arteries). However, one of the main difficulties is the evaluation of the perfusion of  
252 branch vessels itself with 2D or even with 3D angiograms. Oftentimes it is not possible to  
253 ascertain if there was complete resolution of the conflict or not, since even the contrast  
254 injection with the power injection within aorta may hide residual dynamic component of the  
255 malperfusion conflict [5]. Some authors suggested the use of IVUS or OCT technologies to  
256 assist in the assessment of the need of additional stents [5]. Nevertheless, no comparative  
257 data are available in literature to define if the availability of these techniques would modify  
258 the rates of additional selective stenting in this specific setting. IVUS is not available in our  
259 centres and this also probably meant some additional selective stenting may have been  
260 done with no strong hemodynamical arguments. Further studies are needed to clarify this  
261 issue.

262

263 The retrospective nature of the study made the possibility of having a consistent protocol for  
264 the assessment of the severity of malperfusion and time elapsed until its relief difficult to  
265 define. Current routinely available biological markers elevation is not fast enough to

266 anticipate and characterize malperfusion syndroms. Imaging techniques such as flow-MRA  
267 has great potential and could provide very interesting information in parallel to what has  
268 been show in the chronic phase to predict aortic event but cannot be applied currently in  
269 the acute phase with such long acquisition delay [13].

270 Another possible limitation of the present study is the exclusion of patients with frank aortic  
271 rupture. However, this was done to limit the effects of any malperfusion due to hypotension  
272 deriving from the rupture and hopefully was compensated by the number of patients  
273 included, which made it one of the largest studies with patients presenting with acute (<14  
274 days) type B complicated only with malperfusion at onset [5-8].

275

## 276 **Conclusion**

277 Prognosis of patients with acute type B Aortic Dissection complicated initially with  
278 malperfusion syndrome is severe, especially if the ischemia has become clinical apparent on  
279 several territories. Early depiction of this life-threatening complication lacks hard criteria,  
280 which hopefully could improve the results. Endovascular strategy based on TEVAR as first-  
281 stand associated or not with selective stenting of aortic branch vessels and/or fenestration is  
282 thought to be admitted as current standard. Rapid and more reliable flow assessment within  
283 the aortic collaterals may be helpful at the end of the procedure to ensure better chance of  
284 survival for patients.

285

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- 326

327 Figure legends

328 Table 1 – Demographic details and comorbidities of population included in the study

329 Table 2 – Complications at onset with details of malperfusion syndromes

330 Table 3 – Aortic diameters at onset collected from the pre-TEVAR CTA

331 Table 4 – Extension of the dissection and hemodynamical status of the false lumen at onset

332 Table 5 – Procedural details with adjuncts during the index intervention

333 Figure 1 – Presentation of the mechanisms (a) involved in the malperfusion and the lumen

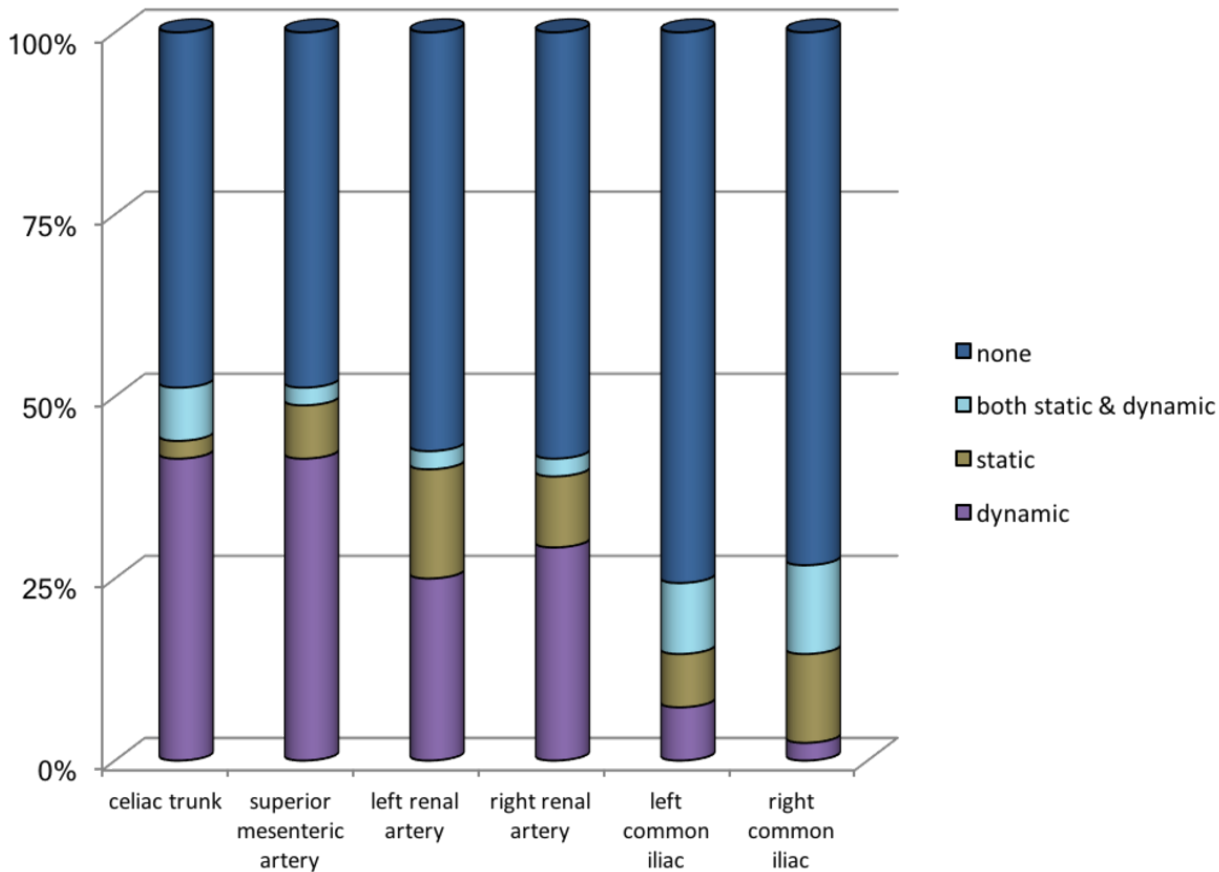
334 feeding the collateral of the aorta (b). CT: celiac Trunk; SMA: superior mesenteric artery;

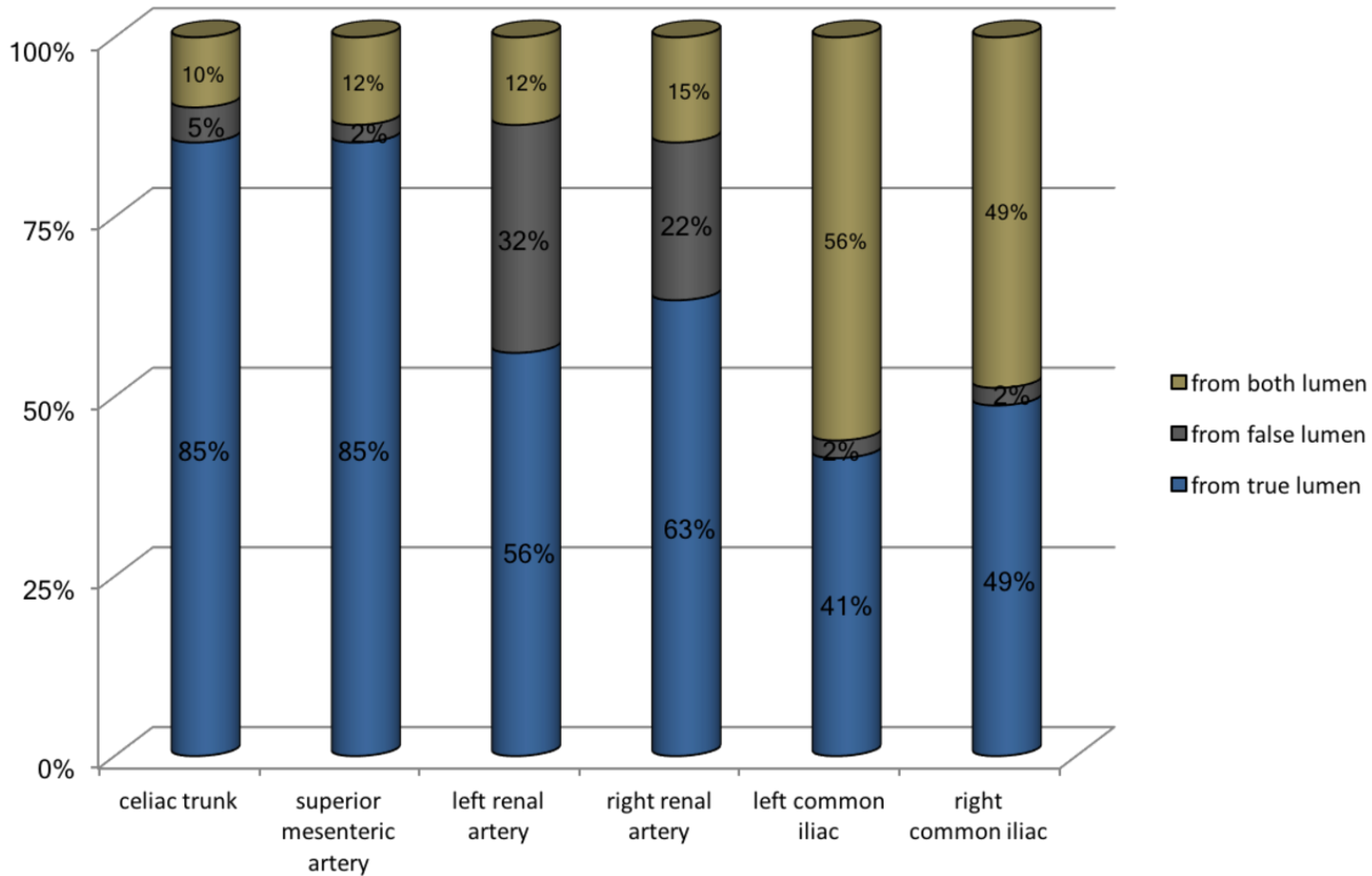
335 RRA: Right Renal Artery; LRA: Left Renal Artery; RCI: Right Common Iliac; LCI: Left Common

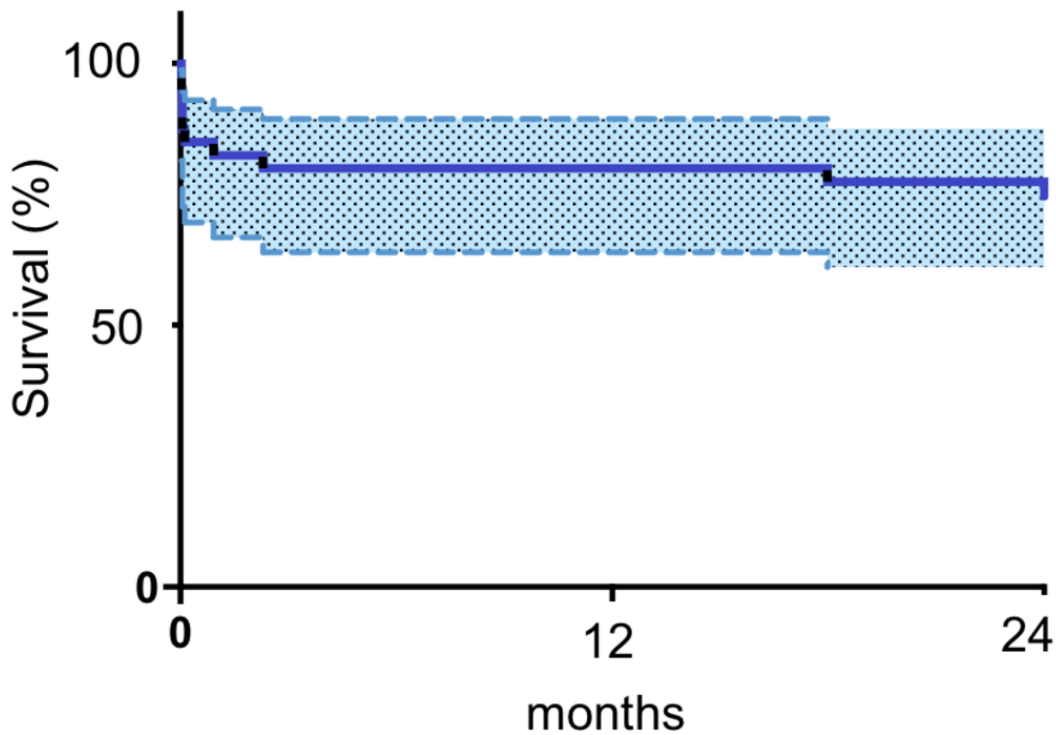
336 Iliac

337 Figure 2 – Overall survival over the 2 first years after TEVAR according to Kaplan Meier

338 method







	6 mo	12 mo	24 mo
Survival (%)	80.0 (70.5-96.0)	80.0 (71.5-96.0)	77.4 (67.3-93.8)
no at risk	33	33	31

Table 1

	<b>n=41</b>
Age at onset [in years; median; IQR]	60 [51-68.5]
Male	78%(32/41)
Body mass Index [Kg/m <sup>2</sup> ; median; IQR]	28 [21-35]
Active smoker	32%(11/34)
Diabetes mellitus	5%(2/41)
Dyslipidemia	39%(16/41)
Hypertension	58%(24/41)
COPD	10%(4/41)
Myocardial infarction	5%(2/41)
Heart fibrillation	10%(4/41)
Previous cardiac surgery	7%(3/41)
Personal aortic disease history	15%(6/41)
Familial aortic disease history	7%(3/41)
Personal neoplasia history	10%(4/41)

COPD: Chronic obstructive pulmonary disease

Table 2

Organ(s) affected by malperfusion syndrome		<b>n=41</b>
renal		53.7%(22)
visceral		41.5%(17)
spinal cord		12.2%(5)
lower limb		34.1%(14)
<b>Cumulated malperfusion(s) syndrome(s) at onset per patient</b>		
	1	68.3%(28)
	2	24.4%(10)
	3	7.3%(3)
	4	0
<b>Others complications described at onset</b>		
transaortic diameter >55mm		2.4%(1)
persistent hypertension		46.3%(19)
intractable pain		78.0%(32)

Table 3

<b>aortic diameters (mean, SD)</b>	<b>true lumen (mm)</b>	<b>total diameter (mm)</b>
Aorta above LSCA	17 (8)	37 (8)
max within DTA	16 (6)	37 (8)
Aorta at CT level	11 (9)	29 (5)
Aorta at SMA level	11 (10)	28 (5)
Aorta at LRA level	11 (10)	26 (6)
Aorta at RRA level	12 (10)	26 (6)
max within Infrarenal aorta	12 (8)	25 (6)

LSCA: left subclavian artery; DTA: descending thoracic aorta; CT: celiac trunk; SMA: superior mesenteric artery;  
 LRA: left renal artery; RRA: right renal artery



Table 4

<b>Location of the proximal aspect of dissection</b>	<b>% (n)</b>	
aorta above LSCA origin	4.9 (2)	
aorta at LSCA origin	36.5 (15)	
Descending thoracic aorta	31.7 (13)	
in LSCA	26.8 (11)	
<b>Location of the distal aspect of dissection</b>	<b>% (n)</b>	
aorta above diaphragm	4.8 (2)	
infrarenal aorta	21.9 (9)	
beyond infrarenal aorta	73 (30)	
<b>Total length of dissection (mm, median IQR)</b>	439 (255-739)	
<b>Length of the proximal entry tear (mm, median IQR)</b>	9 (5-30)	
<b>False lumen status</b>	Abdominal (% , n)	Thoracic (% , n)
complete thrombosis	14.6 (6)	0
partial thrombosis	14.6 (6)	12.2 (5)
patent	70.7 (29)	87.8 (36)

LSCA: left subclavian artery

Table 5

	<b>Median</b>	<b>IQR</b>
Estimated Aortic coverage (mm)	197	157-209
Proximal stent graft diameter (mm)	36	36-40
Number of stent graft implanted	1	1-1
LSA origin coverage (% , n)	78%(32)	
	Ishimaru zone 1	5.0%(2)
	Ishimaru zone 2	73.2%(30)
		%(n)
Selective stenting and/or fenestration	43.9%(18)	
	visceral (CT or SMA)	9.8%(4)
	renal (1 or both)	24.4%(10)
	iliac (1 or both)	14.6%(6)
LSA revascularisation (bypass or transposition)	14.6%(6)	
LCC Chimney	5.0%(2)	
Preoperative CSF drainage	17%(7)	
Procedural time (min)	150	120-227

IQR:Interquartile Range; LSA:Left Subclavian artery; CT:Celiac trunk; SMA: Superior Mesenteric Artery; LCC:Left Common Carotid; CSF:cerebrospinal fluid