Perioperative and mid-term results of endovascular management of complicated type B aortic dissection using a proximal thoracic endoprosthesis and selective distal bare stenting

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OBJECTIVES: To present results of endovascular treatment of complicated type B aortic dissection.

METHODS: Patients with acutely complicated type B aortic dissection extending from the left sub-clavian artery to the abdominal aorta were treated. The strategy involved the placement of a covered endoprosthesis to seal the primary entry tear and, in cases where malperfusion persisted, distal extension with uncovered stents, to enhance true lumen (TL) expansion and reperfusion of the ischaemic arterial branches originating from the TL.

RESULTS: Thirty-five patients were included. Mean age was 63.1 (37–79) years and malperfusion syndrome occurred in 71.4%. Average aortic coverage with endoprostheses was 220.6 mm (136–355 mm). In 17 cases (48.5%), distal extension with uncovered stents was necessary. Thirty-day major morbidity was 28.5%, and mortality 2.8%. At follow-up (25.6 ± 19.5 months; 4–73 months), overall mortality was 15.2%, and aortic mortality 12.1%. Additional aortic intervention was necessary in 18.2%. Although patients undergoing distal extension with bare metal stents suffered most often from preoperative malperfusion, no significant differences were noted in the 30-day and follow-up clinical results. Follow-up angio-computed tomography showed complete thrombosis of the false lumen in the proximal half of the thoracic aorta in 76.4%. Patients treated with bare stents had significantly larger aortic TL sizes, at the different abdominal aorta levels.

CONCLUSIONS: In patients with complicated type B aortic dissection, tailored elongation with uncovered stents is a safe treatment of persistent malperfusion. Although the results presented are encouraging, randomized data and a longer follow-up are required to confirm benefits and complications of this strategy.

Keywords: Aortic dissection type B • Endovascular stent • Bare metal

INTRODUCTION

We present our mid-term clinical and radiographic results in the treatment of complicated type B aortic dissection using a tailored strategy of proximal entry tear coverage by means of endoprosthesis implantation followed, whenever required, by bare stenting of the distal thoracic aorta, to achieve homogeneous aortic true lumen (TL) expansion, correct persistent distal malperfusion and facilitate false lumen (FL) thrombosis.

MATERIALS AND METHODS

TREATMENT INDICATIONS

Endovascular treatment was performed in a series of patients with Stanford type B aortic dissection complicated with at least one of the following conditions: malperfusion syndrome (clinical and imaging diagnosis), acute expansion of the FL (more than 25% maximal diameter from the last imaging evaluation), aortic rupture (haemorrhage outside of the aortic boundaries), thoraco-abdominal pain unresponsive to maximal medical treatment and arterial
hypertension unresponsive to maximal treatment (including intravenous treatment).

Patients were treated at different time points from the initial diagnosis of aortic dissection (acute and subacute) and timing was based mainly on clinical evolution and responsiveness to medical treatment.

Preoperative evaluation

All patients were submitted to preprocedural thoraco-abdominal contrasted computed tomography (CT). In particular, after multiplanar and 3D imaging reconstruction, the aortic dissection anatomy was defined and the size and perfusion of the FL and TL at various anatomical levels were noted together with the position and dimensions of the most proximal entry tear and eventual distal re-entries. A proximal landing zone of at least 1.5 cm in length was identified and aortic dimensions at this level were calculated after multiplanar image reconstruction. Proximal landing zone diameter guided our endoprosthesis selection and we adopted an oversizing no greater than 10% of the proximal landing zone diameter. As a general rule, we primarily aimed at covering all distal re-entries, to minimize the risk of continuous retrograde perfusion of the FL. In patients where, in order to achieve an adequate proximal landing zone, intentional closure of the left subclavian artery (LSA) ostium was expected, complete evaluation of the supra-aortic vessels and intra-cranial circulation was performed by means of either CT or magnetic resonance imaging. Surgical revascularization of the LSA was performed before the endovascular procedure, whenever a hypoplastic non-compensatory right vertebral artery was noticed and/or anomalies of the Willis’ circle, which could have increased the risk of hypoperfusion of the basilar artery, were present.

Operative phases

All procedures were performed in a hybrid operating room, under general anaesthesia, with endotracheal intubation and mechanical ventilation. Cerebrospinal fluid pressure monitoring was not routinely adopted. Intra procedural guidance was achieved with fluoroscopy and transoesophageal echocardiography.

Surgical exposure of the femoral artery was performed to facilitate the endovascular procedure. Endoprosthesis and stent deployment was routinely achieved under rapid right ventricular pacing. Once complete deployment of the endoprosthesis was achieved, a control aortography confirmed adequate re-expansion of the aortic TL and reduced perfusion of the FL along the entire extension of the thoraco-abdominal aorta.

Distal extension of the endoprosthesis was achieved by means of uncovered stents in selected cases of incomplete expansion of the distal TL, continuous retrograde perfusion of the FL with high flow rates and evidence of malperfusion of arterial branches originating from the TL. Those findings were routinely evaluated by means of intraoperative aortography and transoesophageal echocardiography.

The uncovered stents had the function to ‘scaffold’ the dissection lamella even across the ostia of the major abdominal arterial branches preventing, in this way, aortic TL collapse and peripheral malperfusion. To guarantee adequate proximal anchoring, stent diameter was selected on the basis of the endoprosthesis’ size (at least the same diameter or less than 25% oversizing) and an imbrication length of at least 3–5 cm was considered acceptable.

Data collection and statistical analysis

Data were collected and analysed prospectively. The study was approved by an institutional review committee, an IRB approval was obtained and subjects gave informed consent. Follow-up duration was 25.6 ± 19.5 months (4–73 months).

At follow-up, aortic events and aortic mortality were noted. Pre- and post-procedural (latest follow-up) aortic CT findings were compared. Clinical and imaging findings were also compared between patients undergoing treatment with or without bare metal stent extension. Differences were tested using the paired and unpaired Student’s t-test and the Wilcoxon signed-rank test. A P < 0.05 was considered statistically significant. Thrombosis of the FL at four different thoraco-abdominal aortic levels was evaluated: I: thoracic aorta from LSA ostium to the level of the left atrium; II: thoracic aorta from the level of the left atrium to the diaphragm; III: abdominal aorta from the diaphragm to the renal arteries; IV: abdominal aorta distal to the renal arteries. Kaplan-Meier survival analysis, including aortic event-free survival, was performed. The statistical calculations were run using the SPSS 11.0 software.

RESULTS

Preoperative findings

The present series includes 35 patients treated as a result of complicated type B aortic dissection. The patients included were treated during a period of 6 years and represent a sample of the total population of patients treated with thoracic endovascular aortic repair (TEVAR) at our institution. Moreover, during the same time frame, we were referred a total of 360 patients with type B aortic dissection. Demographic and comorbidity data are summarized in Table 1.

Malperfusion syndrome (71.4%), aortic rupture (14.3%), rapid FL enlargement (8.6%), uncontrollable thoracic pain (2.8%) and unresponsive systemic arterial hypertension (2.8%) were all indications for endovascular treatment. Table 2 summarizes the arterial involvement and mechanism for distal malperfusion, as documented by preprocedural CT angiography. The median time interval from diagnosis to endovascular therapy was 18.7 ± 41.7 days in the overall cohort and 25 (71.4%) patients were treated within the first 14 days after diagnosis of type B aortic dissection.

Planned neck vessels revascularization

Proximal landing zone was distal to the LSA ostium (IIIA) in 8 (22.8%) patients. In the majority of patients (n = 22, 62.8%), a coverage of the LSA ostium was preplanned (landing zone IIC). In 4 (11.4%) patients, the proximal landing zone was distal to the origin of the brachiocephalic trunk (BCT) (IIIB) and coverage of the left common carotid artery (LCCA) and LSA was preplanned. In 1 case (2.8%), proximal landing was scheduled at the level of the distal ascending aorta (IIIA) with intentional coverage of BCT, RCCA and LSA. A total of 14 (40%) patients underwent some sort of neck vessel surgical revascularization before the endovascular treatment. Eight patients (22.8%) had sole LSA revascularization, 3 (8.5%) had simultaneous LCCA and LSA revascularization, 2 (5.6%) had sole LCCA revascularization and 1 (2.8%) had simultaneous revascularization of the BCT-LCCA-LSA.
Perioperative findings

Table 3 summarizes operative findings, including numbers and types of implanted endoprostheses and uncovered stents. In 17

Table 1: Baseline clinical characteristics of the patient population

<table>
<thead>
<tr>
<th>Demographic data</th>
<th>No. of patients</th>
<th>Age (years)</th>
<th>Male gender</th>
<th>Body mass index (kg/m²)</th>
<th>Interval between diagnosis and treatment (days)</th>
<th>ASA class 3</th>
<th>ASA class 4</th>
<th>Follow-up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number</td>
<td>35</td>
<td>63 ± 11.9</td>
<td>27 (77.1%)</td>
<td>26.4 ± 4.8 (19.2–40.4)</td>
<td>18.7 ± 41.7 (0–238)</td>
<td>19 (54.2%)</td>
<td>16 (45.7%)</td>
<td>25.6 ± 19.5 (0.4–72.8)</td>
</tr>
</tbody>
</table>

Previous aortoarterial interventions

- Supracoronary ascending aorta/partial replacement of aortic arch in type A dissection
- Valve conduit in type A dissection
- TAAA in abdominal aortic replacement and off-pump CABG
- Interventional fenestration of the dissection lamella
- Renal artery stenting
- Bare metal stenting of aorta

Relevant comorbidities

- Arterial hypertension
- Dyslipidaemia
- Chronic lung disease
- CAD
- CKD
- Type 2 diabetes mellitus
- Cancer
- Curatively treated
- In treatment

Dissection-related morbidity and clinical symptoms

- Chest pain
- AKI
- Claudication
- Abdominal pain
- Oliguria/anuria
- Acute dialysis dependency
- Preinterventional mechanical ventilation dependency
- Preinterventional catecholamine dependency

Data are expressed as rates and mean ± SD with relative ranges.


In all patients with preoperative malperfusion syndrome (n = 25; 71.4%), intraprocedural recovery of good organ perfusion was documented at the end of the intervention. One patient (2.8%) required implantation of a renal artery stent for persistent left renal artery malperfusion. In a second patient (2.8%), we observed complete recanalization of a hepatic artery that presented with static occlusion at the time of diagnosis.

In 2 patients (5.7%), inadvertent proximal and distal misplacement of the endoprosthesis was observed during deployment. In one patient, this resulted in iatrogenic occlusion of the LCCA that was managed with an emergent surgical revascularization. In a second patient, the distal misplacement of the endoprosthesis resulted in incomplete coverage of the primary entry tear that required placement of a second more proximal endoprosthesis.

Postoperative findings

Tables 4 and 5 summarize outcomes in the two groups and in the overall cohort. During the hospital stay, 2 patients died (5.7%). One patient died as a result of retrograde aortic dissection 13 days after complete debranching of the neck vessels and stenting of the aortic arch and thoracic aorta. A second patient died 43 days after the endovascular procedure as a result of protracted sepsis with multiorgan failure.

The 30-day morbidity rate was 28.5%. Neurological complications were observed in 2 patients (5.7%). One patient suffered from multiple brain embolisms, resulting in transient paraparesis of the lower limbs. Another patient suffered from a brain stem infarction, few days after the procedure, resulting in transient balance loss. Furthermore, 2 patients (5.7%) suffered from transient paraparesis of the lower limbs that was fully resolved at the time of discharge. In one case, cerebrospinal fluid drainage was placed and surgical revascularization of the covered LSA was performed. A second patient was handled conservatively.

Within 30 days after endovascular treatment, 1 patient (2.8%) developed recurrent abdominal malperfusion that was solved after coeliac trunk and superior mesenteric artery stenting. No differences were noticed between patients undergoing standard endovascular treatment and elongation with bare metal stents (Table 4).
Follow-up mortality

After hospital discharge, 5 patients died during the clinical observation interval of 25.6 ± 19.5 months. Four patients died as a result of aortic complications. One patient developed, 99 days after standard endovascular treatment, and four after bare metal stent extension (P = ns).

Follow-up re-intervention

In 9 patients (25.7%), endoleaks were diagnosed at CT angiography, before hospital discharge. Five endoleaks occurred in patients after standard endovascular treatment, and four after bare metal stent extension (P = ns).
Follow-up organ function recovery after malperfusion

In 16 patients (45.7%), renal malperfusion due to dynamic and/or static obstruction of the renal arteries was diagnosed, with consecutive acute renal failure. Nine patients (25.4%) required emergent dialysis before endovascular treatment and 8 of them were weaned off dialysis before discharge and maintained adequate renal function at follow-up. One patient with a history of end-stage chronic renal failure remained on peritoneal dialysis at follow-up.

One patient developed occlusion of the renal artery immediately after aortic endoprosthesis release, and as result of displacement of the dissection flap. The vessel was immediately recanalized and successfully stented.

Ten patients (28.5%) initially diagnosed with intestinal ischaemia were fully recovered after endovascular treatment of the aortic dissection and remained symptom-free at follow-up. One patient (2.8%) underwent a hemicolectomy for intestinal gangrene before endovascular treatment. In 10 cases (28.5%), lower limb malperfusion was reported preoperatively. Distal perfusion recovered immediately after treatment in all patients and was confirmed at clinical follow-up.

Kaplan–Meier analysis and event-free survival

The Kaplan–Meier analysis showed a 1- and 3-year survival probability of 80.0 ± 6.8%. The 1- and 3-year survivals free from any major adverse vascular events were 77.1 ± 7.1 and 67.6 ± 9.0%, respectively. No significant differences were observed in the estimated survival and event-free survival of patients treated with and without bare metal stent elongation (P = 0.7).

Follow-up imaging

An imaging follow-up (16.6 ± 13.7 months) was performed by means of contrast CT angiography. The degree of FL thrombosis was investigated at four different thoraco-abdominal aortic levels (see Materials and Methods section) and findings are summarized in Table 6.

Table 6: False lumen thrombosis after endovascular treatment

<table>
<thead>
<tr>
<th>Aortic level</th>
<th>Baseline</th>
<th>Latest follow-up*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Segment I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No thrombosis</td>
<td>79.4 (27/34)</td>
<td>0.0 (0/34)</td>
</tr>
<tr>
<td>Partial thrombosis</td>
<td>20.5 (7/34)</td>
<td>23.5 (8/34)</td>
</tr>
<tr>
<td>Complete thrombosis</td>
<td>0.0 (0/34)</td>
<td>76.4 (26/34)</td>
</tr>
<tr>
<td>Segment II</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No thrombosis</td>
<td>88.5 (31/35)</td>
<td>5.7 (2/35)</td>
</tr>
<tr>
<td>Partial thrombosis</td>
<td>11.4 (4/35)</td>
<td>28.5 (10/35)</td>
</tr>
<tr>
<td>Complete thrombosis</td>
<td>0.0 (0/35)</td>
<td>65.7 (23/35)</td>
</tr>
<tr>
<td>Segment III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No thrombosis</td>
<td>97.0 (33/34)</td>
<td>26.4 (9/34)</td>
</tr>
<tr>
<td>Partial thrombosis</td>
<td>2.9 (1/34)</td>
<td>55.8 (19/34)</td>
</tr>
<tr>
<td>Complete thrombosis</td>
<td>0.0 (0/34)</td>
<td>17.6 (6/34)</td>
</tr>
<tr>
<td>Segment IV</td>
<td></td>
<td></td>
</tr>
<tr>
<td>No thrombosis</td>
<td>93.7 (30/32)</td>
<td>43.7 (14/32)</td>
</tr>
<tr>
<td>Partial thrombosis</td>
<td>6.2 (2/32)</td>
<td>46.8 (15/32)</td>
</tr>
<tr>
<td>Complete thrombosis</td>
<td>0.0 (0/32)</td>
<td>9.3 (3/32)</td>
</tr>
</tbody>
</table>

No thrombosis: presence of contrast agent without thrombosis in false lumen; partial thrombosis: presence of contrast agent and thrombosis in false lumen; complete thrombosis: thrombosis of false lumen without the presence of contrast agent.

*16.6 ± 13.7 months.

Table 7: Aortic diameter after endovascular treatment

<table>
<thead>
<tr>
<th>Measuring point</th>
<th>Lumen</th>
<th>Baseline (n = 35)</th>
<th>1-year follow-up (n = 30)</th>
<th>Latest follow-up* (n = 35)</th>
<th>P-value*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left common carotid artery (mm)</td>
<td>TL</td>
<td>30.0 ± 4.7</td>
<td>31.5 ± 4.1</td>
<td>31.5 ± 4.2</td>
<td>0.1</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>3.2 ± 5.8</td>
<td>1.6 ± 3.3</td>
<td>1.3 ± 3.0</td>
<td>0.06</td>
</tr>
<tr>
<td>Left sub-clavian artery (mm)</td>
<td>TL</td>
<td>21.9 ± 7.5</td>
<td>29.6 ± 4.3</td>
<td>29.5 ± 4.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>14.0 ± 7.6</td>
<td>6.7 ± 13.6</td>
<td>7.0 ± 12.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Coeliac trunk (mm)</td>
<td>TL</td>
<td>9.0 ± 7.6</td>
<td>18.1 ± 6.5</td>
<td>19.2 ± 6.5</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>22.8 ± 6.8</td>
<td>143 ± 9.0</td>
<td>135 ± 9.6</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Superior mesenteric artery (mm)</td>
<td>TL</td>
<td>9.3 ± 7.5</td>
<td>17.2 ± 6.1</td>
<td>17.8 ± 5.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>20.9 ± 6.4</td>
<td>121 ± 7.9</td>
<td>124 ± 9.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Left renal artery (mm)</td>
<td>TL</td>
<td>9.45 ± 7.1</td>
<td>15.5 ± 5.4</td>
<td>15.8 ± 5.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>17.1 ± 5.7</td>
<td>11.7 ± 6.5</td>
<td>11.8 ± 5.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Right renal artery (mm)</td>
<td>TL</td>
<td>9.29 ± 7.14</td>
<td>15.3 ± 5.4</td>
<td>15.9 ± 5.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>17.5 ± 6.0</td>
<td>11.7 ± 6.5</td>
<td>11.7 ± 7.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Right common iliac artery (mm)</td>
<td>TL</td>
<td>6.9 ± 4.4</td>
<td>9.5 ± 3.2</td>
<td>9.9 ± 3.3</td>
<td>0.001</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>7.0 ± 6.3</td>
<td>5.5 ± 5.6</td>
<td>5.0 ± 5.4</td>
<td>0.04</td>
</tr>
<tr>
<td>Left common iliac artery (mm)</td>
<td>TL</td>
<td>7.5 ± 6.2</td>
<td>9.7 ± 4.7</td>
<td>9.7 ± 4.4</td>
<td>0.009</td>
</tr>
<tr>
<td></td>
<td>FL</td>
<td>7.0 ± 5.4</td>
<td>6.1 ± 4.3</td>
<td>5.7 ± 4.3</td>
<td>0.07</td>
</tr>
<tr>
<td>Maximum thoracic diameter (mm)</td>
<td>TL+FL</td>
<td>43.2 ± 8.8</td>
<td>426 ± 13.6</td>
<td>43.0 ± 13.1</td>
<td>0.9</td>
</tr>
<tr>
<td>Maximum abdominal diameter (mm)</td>
<td>TL+FL</td>
<td>34.0 ± 6.8</td>
<td>35.1 ± 8.1</td>
<td>36.1 ± 8.7</td>
<td>0.1</td>
</tr>
<tr>
<td>Minimum thoracic diameter (mm)</td>
<td>TL</td>
<td>11.0 ± 7.9</td>
<td>21.5 ± 6.7</td>
<td>20.7 ± 7.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Minimum abdominal diameter (mm)</td>
<td>TL</td>
<td>7.8 ± 7.2</td>
<td>15.2 ± 5.8</td>
<td>15.4 ± 6.8</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

*16.6 ± 13.7 months.
*Pre-TEVAR versus latest FU.
TL: true lumen; FL: false lumen; TEVAR: thoracic endovascular aortic repair.
A thrombosis of the FL at the level of the aortic segment I (thoracic aorta from LSA ostium to the level of the left atrium) was noted in 76.4% of the investigated patients.

A complete FL thrombosis along the entire extension of the aorta from Segment I to Segment III (renal arteries ostia) was observed in 17.6% of the patients.

No differences in terms of FL thrombosis extension and distribution were noticed in patients treated with or without bare metal stent extension.

Table 7 summarizes the TL and FL preoperative and follow-up measurements at different aortic levels (defined by the origin of the major arterial branches). We have documented a significant increase in the minimal TL diameter and a significant reduction in the FL maximal diameters at every thoracic and abdominal level.

At the various abdominal aorta levels, there was a significantly higher regain of the TL diameter in patients undergoing endovascular treatment with bare metal stent extension (Table 8).

**DISCUSSION**

Malperfusion syndrome, as a result of type B aortic dissection, carries a heavy burden of mortality and morbidity, and will involve at least half of patients referred for treatment [1–7]. The goal of TEVAR in complicated type B aortic dissection is to cover the most proximal entry tear and reduce FL perfusion enhancing TL expansion. After endoprosthesis deployment, and proximal entry tear coverage, a TL expansion should be observed and maintained along the entire aortic length, including the ostia of the ischaemic arterial branches. Distal malperfusion is often a dynamic event, resulting from the siphoning of the dissection lamella and the alternate occlusion of the arterial branch ostia originating from the TL. Extension of the endoprosthesis with bare metal stents has been proposed with Provisional ExTension To Induce COmplete Attachement (PETTICOAT) after stent graft placement in type B aortic dissection [4, 8–11].

Concern has been raised about the iatrogenic risk of aggravating the visceral ischaemia as a result of further intra-aortic manipulation and stenting extension across the ostia of the major abdominal arterial branches. In a recent review on the management of complicated type B aortic dissection using aortic extension with bare metal stenting, a total rate of 17.6% of additional endovascular interventions on ischaemic visceral branches was reported [4]. Faure et al. have recently described an experimental study in a human ex vivo model of type B aortic dissection [5]. They documented a high rate (54.5%) of pressure gradient drop in visceral branch vessels after bare metal stenting, whenever those arteries were supplied by the FL.

We believe that, in order to avoid iatrogenic malperfusion, PETTICOAT should be adopted in a patient tailored fashion, and only whenever preoperative malperfusion is not resolved after initial deployment of the covered endoprosthesis.

In our experience, malperfusion was present in over 70% of the referred patients and, in up to 90% of these patients, we reported a dynamic malperfusion. The arterial branches were originating from the aortic TL and were either occluded by the ‘flapping’ intimal dissection membrane, which at times extended within the arterial branches as well, or were malperfused as a result of a proximal diastolic collapse of the TL.

After deployment of the aortic endoprosthesis, and coverage of the most proximal entry tears, distal malperfusion persisted, and was documented at intraoperative angiography in almost 50% of our patients. For this reason, elongation of the aortic endoprosthesis with bare metal stents was selectively performed. We have never treated with bare stent extension patients without previously diagnosed malperfusion. Generally speaking, intraoperative findings, if correctly interpreted, correlate with preoperative clinical status. Thanks to this tailored approach, iatrogenic visceral ischaemia was a rare occurrence and immediate further endovascular intervention on the visceral arteries was necessary just in one case of static preoperative renal artery occlusion. In a second patient, coeliac artery and mesenteric artery stenting was performed to solve recurrent malperfusion that occurred 30 days after the endovascular procedure.

In summary, our findings confirm that, whenever visceral ischaemia occurs as a result of dynamic malperfusion, and possibly TL collapse, bare metal stent elongation may enhance distal perfusion of those branches originating from the TL. Although as already emphasized in our experience, all patients treated with extension had ‘abdominal branches’ originating from the TL, PETTICOAT in patients with origin of arterial branches from the FL should be considered cautiously and would most probably lead to branch occlusion unless there is evidence of a large more distal re-entry tear.

As a result of the patients’ complex comorbid profile, and the technical difficulties encountered during the endovascular procedure, the total 30-day morbidity rate after combined TEVAR and distal aortic stenting for complicated type B aortic dissection may

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**Table 8: Aortic true lumen diameter after endovascular intervention with and without bare stent elongation**

<table>
<thead>
<tr>
<th>Measuring point</th>
<th>Lumen</th>
<th>1-year follow-up w/o bare stents (n = 16)</th>
<th>1-year follow-up w/o bare stents (n = 14)</th>
<th>P-value</th>
<th>Latest follow-up* w/o bare stents (n = 17)</th>
<th>Latest follow-up* w/o bare stents (n = 18)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left common carotid artery (mm)</td>
<td>TL</td>
<td>31.3 ± 2.0</td>
<td>31.7 ± 5.8</td>
<td>0.8</td>
<td>31.2 ± 2.1</td>
<td>31.8 ± 6.0</td>
<td>0.7</td>
</tr>
<tr>
<td>Left sub-clavian artery (mm)</td>
<td>TL</td>
<td>30.6 ± 3.3</td>
<td>28.4 ± 5.0</td>
<td>0.1</td>
<td>30.5 ± 3.2</td>
<td>28.6 ± 4.6</td>
<td>0.1</td>
</tr>
<tr>
<td>Coeliac trunk (mm)</td>
<td>TL</td>
<td>21.5 ± 4.5</td>
<td>14.0 ± 6.2</td>
<td>0.001</td>
<td>22.0 ± 4.2</td>
<td>16.3 ± 7.3</td>
<td>0.009</td>
</tr>
<tr>
<td>Superior mesenteric artery (mm)</td>
<td>TL</td>
<td>20.4 ± 4.4</td>
<td>13.3 ± 5.8</td>
<td>0.002</td>
<td>20.5 ± 3.8</td>
<td>15.1 ± 6.4</td>
<td>0.006</td>
</tr>
</tbody>
</table>

*16.6 ± 13.7 months. TL: true lumen.
increase. Canaud et al. reported a pooled rate of severe morbidity of 33.3% (36/108 included in four separate studies) spanning from 0 to 65% [4, 8–11]. More recently, Hofferberth et al. have compared outcomes between combined proximal descending aortic endografting plus distal bare metal stenting and conventional proximal descending aortic stent graft repair in a group of 63 patients with type A and type B aortic dissection [12]. At follow-up, they report a significantly lower unplanned reintervention rate (11%) and aortic mortality (5%) in the PETTICOAT group.

They conclude that combined proximal descending aortic endografting plus distal bare metal stenting provides favourable short-term outcomes and decreases late distal aortic complications compared with conventional endovascular repair [12].

Although some authors have proposed a contained major complication rate of 11.1% in acute and chronic dissection patients undergoing isolated TEVAR without bare stent extension [6], results should be reinterpreted in the light of patients’ clinical complexity. In fact, in a recent multicentre experience in highly comorbid patients with type B aortic dissection treated with emergent TEVAR, without additional distal bare stenting, Wiedemann et al. have reported an in-hospital complication rate of 36% (32/110), including retrograde type A aortic dissection, neurological deficits, renal dysfunction and intestinal malperfusion [7].

Interestingly, the most common complications occurring in our series were protracted mechanical ventilation (14.2%) and sepsis (11.4%). This is possibly the result of the fact that over 70% of our patients were treated emergently for organ malperfusion (mainly gut and renal ischaemia) that may have resulted in bacterial translocation and volume overload leading to sepsis and respiratory impairment.

A comment apart should be given to neurological and aortic complications. We have reported a 5.7% cerebrovascular accident rate and a similar paraplegia rate. All patients recovered full functionality, including independent ambulation and balance. Our findings are similar to those proposed in the most recent literature, including patients treated with and without bare metal stent extensions [4, 7]. Interestingly, in spite of the fact that, in order to achieve maximal coverage of distal aortic re-entries, we had an average aortic coverage length of over 200 mm, we could contain the neurological occurrences thanks to a systematic approach towards preoperative cerebral perfusion evaluation.

Concerning the aortic complications, retrograde dissection and aortic rupture are the leading causes of aortic morbidity and mortality after TEVAR. We had just one occurrence of retrograde aortic dissection in the acute phases, in a patient undergoing total arch debranching. Wiedemann et al. have proposed a multicentre 5.4% rate of retrograde aortic dissection after sole TEVAR for acute type B dissection [7]. They confirm that extension of the proximal landing zone to the aortic arch and ascending aorta may contribute to the increased rate. Canaud et al. have reported, in a registry of over 1000 TEVAR patients, a 1.6% rate of retrograde aortic dissection [13]. They confirmed that the main determinant for such a treacherous occurrence was treatment for acute type B aortic dissection [13]. In any case, we should emphasize that, retrograde aortic dissection and aortic rupture may occur even at mid-term follow-up, as documented in our experience. Four patients died as a result of aortic complications (ascending aorta haematoma, dissection and aortic rupture) occurring from 35 to 160 days after the indexed procedure. All these occurrences are possibly unrelated to the additional instrumentation required for distal bare stenting and are resulting from the dynamic evolution of the aortic pathology.

The technical difficulties encountered in the treatment of this unstable aortic condition are reflected also in the high rate of endovascular reinterventions occurring at follow-up. In cumulative experiences using TEVAR with covered endoprostheses plus bare metal stent extension, Type I endoleaks were reported in 4.8% of the patients and overall adjunctive reinterventions in 8.5% (4–13.3%) [4].

Although we report endoleak rate of over 25%, we should remark that after endovascular treatment most of these patients presented with very favourable aortic anatomical and clinical findings, including maintained organ perfusion. As a result, further endovascular intervention was necessary in just 14% of the patients.

It should be noted that, although patients undergoing PETTICOAT had a higher rate of preprocedural malperfusion syndrome and, in spite of the longer and more complex operative sessions required to perform PETTICOAT, 30-day complications rate, including further interventions, remained contained and comparable with that of patients treated with sole endoprostheses.

In any case, the possible benefits of PETTICOAT should be evaluated in terms of late aortic remodelling, FL regression/thrombosis and TL volume regain. In this context, even when using extensive aortic stenting, a complete sealing of the aortic FL is most often difficult to achieve and, at 1 year, FL patency approximates 30% at the thoracic level and is over 80% at the abdominal aorta level [4]. Similar results, with an abdominal aorta FL thrombosis rate as low as 7%, have been proposed when using a more conservative approach, without bare metal stent extension [7, 14]. Our evaluation in four separate aortic segments confirms that, although FL thrombosis of the thoracic aorta from LSA ostium to the level of the left atrium is a frequent occurrence (76.4%), it rarely involves simultaneously all the four aortic segments (9.3%). The clinical importance of this finding in terms of aortic remodelling is difficult to define. In fact, differently from other authors that have recently reported a 16.5% rate of aortic aneurysmal evolvement after TEVAR for acute type B dissection [7], we have noticed a consistent increment of the TL size, accompanied by a FL regression in both the thoracic and abdominal aortic segments.

The favourable aortic remodelling that we have observed does not necessarily imply stabilization of the aortic pathology. Moreover, in our experience, it appears that although PETTICOAT did not increase the FL thrombosis rate, it definitely contributed to a consistent and significantly greater TL gain, particularly at the abdominal aortic level.

Although overall aortic-related mortality has been reported to occur in up to 8% of patients submitted to TEVAR with PETTICOAT for type B aortic dissection [9], data available are still very limited. After TEVAR with isolated covered endoprosthesis, Wiedemann et al. have reported an actuarial survival at 1, 3, 5 and 10 years of 85, 83, 73 and 65%, respectively [7]. In this context, freedom from treatment failure according to the Stanford classification has been reported as low as 59% at 5 years [7]. In our experience, late mortality was most often related to aortic complications (mainly retrograde aortic dissection/ascending aorta haematoma) and late aortic mortality accounted for 14.2%. Moreover, the 3-year freedom from any major adverse vascular events was 67.6%. Both these findings confirm that, independently by the strategy used (with or without bare metal stent extension) and, even when adopting a selective and tailored approach as the one herein proposed, the risk of further catastrophic aortic complications, especially in the proximal segments of the aorta, persists even after the
initial successful endovascular handling of acute aortic type B dissection.

Limitations

Some limitations that are present in the study should be emphasized. The small sample size represents our exposure that, in many ways, is similar to that of many other centres that perform this type of treatment. The lack of randomization is a major limitation and should be addressed in the future to confirm the benefits and drawbacks of this treatment strategy. Finally, the results presented, although encouraging, are still loaded with a burden of aortic morbidity and mortality. It is difficult to say how much this can reflect the complexity of the pathology or can simply result from the use of this still ‘un-coded’ treatment strategy.

CONCLUSION

Endovascular treatment of complicated type B aortic dissection presents decisional and technical challenges. After coverage of the most proximal entry tear, angiographic evidence of TL incomplete expansion and consequent distal malperfusion may persist. In these selected cases, distal extension of the endoprosthesis with uncovered bare metal stents (PETTICOAT) may support TL expansion and distal perfusion regain. Although bare metal stent extension adds a consistent burden of aortic manipulation and instrumentation, the possible benefits seem to outweigh the iatrogenic developments. When a targeted and tailored endovascular strategy is used, the great majority of the treated patients will present acute resolution of peripheral malperfusion and chronic complete thrombosis of the most proximal thoracic aorta, with an overall positive aortic remodelling leading to TL expansion and FL involution. In conclusion, although the results presented in this manuscript are encouraging, randomized data are required in order to confirm if this technique is beneficial in these high-risk patients. Moreover, surveillance for possible complications is required during longer follow-up.

Conflict of interest: none declared.

REFERENCES