Fenestrated and branched endovascular aortic repair for chronic type B aortic dissection with thoracoabdominal aneurysms

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Objective: The treatment of patients with arch and thoracoabdominal aortic aneurysms (TAAAs) and chronic dissections is challenging. We report the results of fenestrated and branched endovascular aortic repair (FEVAR) of such aneurysms.

Methods: A single-center prospective FEVAR trial enrolled 356 patients (2006 to 2011), of whom 30 had chronic dissections with arch aneurysm or TAAAs, or both. Patients were divided into group A, 15 patients (mean age, 58 years) with extensive dissections extending from the arch through the visceral segment, and group B, 15 patients (mean age, 74 years old) with focal dissections and no extension into the thoracic aorta. Inclusion criterion was aneurysm size >5.5 cm in diameter. Customized grafts were implanted into the true lumen, and branches were extended into the true lumen of the supra-aortic trunk (arch branch devices) and visceral vessels. Patients were monitored annually with clinical, imaging, and laboratory studies. Outcome analyses included survival, rupture, spinal cord ischemia, endoleak, morbidity (cardiac, renal or pulmonary), reinterventions, dissection, and aneurysm growth.

Results: The mean time from the onset of dissection to the FEVAR performed in group A was 10.4 years. The mean maximum aneurysm diameter was 60 mm. Follow-up averaged 1.7 years. There were no perioperative deaths. One aortic-related death occurred at 87 days due to progression of a pre-existing untreated arch dissection. No ruptures, cardiac, renal, pulmonary, or spinal cord ischemia complications occurred. Despite the initially narrow true lumen dimensions, stent grafts expanded to their nominal diameters after implantation without any blood flow disturbance of branched visceral vessels and distal aorta. No graft compression occurred. Post-FEVAR growth was noted in two patients, related to type II endoleaks. Sac regression was similar (~6.8 vs –11.4 mm; P = .43), but early endovascular reinterventions were more common in group A (8 patients). Patients with extensive dissection were younger, and the dissection more likely to be associated with a defined connective tissue disease (Marfan syndrome or Loeys-Dietz mutations, 40% vs 0%; P = .006).

Conclusions: FEVAR is feasible for patients with chronic dissections and TAAA. Concerns regarding visceral vessel access and graft compression resulting from narrow true lumen diameters were not relevant in our experience. Favorable sac and lumen morphologic changes, coupled with a low mortality and complication risk, makes this an attractive means of handling this clinical problem. (J Vasc Surg 2013;58:625-34.)
skepticism of this technology as an effective modality for chronic dissections. This report describes our experience with chronic aortic dissections and TAAAs.

METHODS

Patient population. A prospectively collected study was analyzed for patients who underwent FEVAR for juxtarenal and thoracoabdominal aneurysms during an 11-year period (2001 to 2011). Patients were enrolled into a physician-sponsored investigational device exemption trial for juxtarenal/thoracoabdominal aneurysms (National Institutes of Health study number: NCT00583050). However, chronic dissection patients were not treated with these devices until 2006; thus, only patients with evidence of dissection requiring visceral or arch branch incorporation between 2006 and 2011 are included. Informed consent approved by our Institutional Review Board was obtained for each patient. Inclusion criteria for this study have been previously published. Patients undergoing emergency repair were excluded.

Definition and classification of chronic dissection. Patients were divided into two groups by the extent of their dissection. Patients in group A had extensive dissections, which were defined as extending through the visceral segments with type II or type III TAAA, in accordance with the Crawford classification. If the arch was also involved, such as cases with residual arch dissections after open proximal dissection repair, this was noted. Group B patients had focal dissections, which included intimal tears with or abutting the visceral segment that did not extend proximally into the thoracic aorta.

Anatomic assessment and device design. High-resolution computed tomography (CT) scans of the chest, abdomen, and pelvis were obtained. Centerline of flow algorithms based on the true lumen were used to define the longitudinal and radial relationships of the relevant visceral or supra-aortic trunk vessels. Reinforced fenestrations were preferentially used for all renal arteries and the visceral vessels in patients with small true lumens. Helical side-arm branches were used for the visceral vessels in extensive dissections with TAAA when the lumen size was >35 mm. Only three patients (group A) were treated with helical side branches, and the rest were treated with reinforced fenestrations. In the setting of side branches, Fluency stent grafts (C. R. Bard, Murray Hill, NJ) were used to mate the aortic component with the target vessel, and reinforced fenestrations were mated with Jomed (Abbott Vascular, Santa Clara, Calif) and used in accordance with standard methods of mating branches and fenestrations with the visceral vessels. Adjunctive stents were used selectively in areas of kinking or severe tortuosity and invariably were nitinol self-expanding stents.

Endovascular procedure. Procedures were performed in a hybrid operating room with fixed imaging equipment. Preservation of the left subclavian artery and pelvic circulation was emphasized by using extra-anatomic bypass (carotid-subclavian) or distal branched grafts (iliac branched devices). Endografts were always sized to the aorta proximal to the dissection with a 10% to 15% oversizing and not tapered to accommodate any changes in the true lumen dimensions. Cerebrospinal fluid drainage with appropriate hemodynamic management was established when the region of aortic coverage was >20 cm. Access was transfemoral except when conduits were required as a result of small or calcified iliac arteries, and such procedures were often staged.

FEVAR was performed using custom-made Zenith branched and fenestrated endografts and standardized bifurcated and iliac branch devices (Cook Medical Inc, Bloomington, Ind). If the dissected lesion involved the arch and the arch was aneurysmal, the total arch endovascular repair using a custom arch branched graft (Cook Medical) incorporating with two arch vessels (brachiocephalic and left common carotid artery) was performed for patients presenting in 2010 and later (Fig 2). Cerebrospinal fluid drainage was used when >20 cm of the thoracic aorta was covered or in the setting of a compromised internal iliac artery and was maintained for 48 to 72 hours. Technical success was defined as a successful branched graft deployment with all target vessels incorporated and 24-hour survival in the angiographic absence of type I endoleak.

Variables studied. Preoperative variables collected included demographics, maximum aortic diameter, and extent of required aortic repair. The true lumen diameter was not measured because it was very dynamic. Therefore, there was not a size cutoff for a minimal true lumen diameter in this study. Three-dimensional modalities in CT angiography, including multiplanar reconstructions and centerline of flow analyses, were used to measure the maximum diameter of the aneurysm sac and the presence of endoleaks at all time points, in accordance with Society for Vascular Surgery reporting standards. In the absence of specific contraindications, CT scans were performed at 1, 6, and 12 months, and yearly thereafter. Triple-phase contrast studies were used with the delayed phase (5 minutes), which was preferentially to the arterial phase in the determination of the status (patent/thrombosed) of the false lumen. In addition, duplex ultrasound imaging was used to assess branch vessel status (patency, stenosis, residual distal branch dissection). Laboratory studies were obtained annually. All information was stored in an Oracle Clinical database (Oracle Corp, Redwood Shores, Calif), which provided most of the demographic, morphologic, and follow-up data for this patient population. Medical records and radiographic studies were reviewed to supplement information specific to this analysis.

Outcomes. Outcomes included all-cause mortality, rupture, morbidity (cardiac, renal, pulmonary and spinal cord ischemia), endoleaks (primary/secondary), reinterventions, and aortic morphologic changes. Deaths and other variables (unless noted) were classified in accordance with the Society for Vascular Surgery reporting standards. In addition to the review of medical records and quarterly phone calls, the United States Social Security Death Index was automatically queried every 3 months to supplement results. Adverse clinical events
(death, cardiac, respiratory and renal failure, stroke, and spinal cord ischemia) occurring during the postoperative period and throughout follow-up were recorded.

**Statistical analysis.** Demographic and perioperative characteristics are presented as number with percentage or mean ± standard deviation and, when appropriate, were compared with t-tests. Categoric variables, expressed as percentages, were analyzed with the χ² test. Actuarial survival was estimated with Kaplan-Meier analysis curve. The log-rank test was used to evaluate differences between Kaplan-Meier curves. SAS 9.0 software (SAS Institute Inc, Cary, NC) and S-plus 7.0 software (Insightful Corp, Seattle, Wash) were used for statistical analyses.

**RESULTS**

Of 356 patients with TAAAs, FEVAR was used to treat 30 consecutive patients (26 men [87%]) with aortic dissections affecting the thoracoabdominal segment. Extensive dissections were present in 15 patients (group A), four of whom had a surgical history of arch repair and one of whom had chronic proximal dissection with arch dilation requiring branched arch graft. Focal dissections were treated in 15 patients (group B). The mean follow-up was 1.7 ± 1.5 years (range, 1 month-5.2 years) and postoperative aneurysm diameters were 60 ± 15 mm. The mean time from the onset of dissection to the FEVAR was 10.4 ± 9.9 years (range, 2-34 years) for group A. The time from dissection to FEVAR was not calculated for group B because most of the patients never presented with symptoms of an acute dissection.

Baseline patient demographics are detailed in Table I. Technical success was achieved in all patients, although the ability to detect type I and III endoleaks in patients with extensive dissections and many branches was limited. Significant differences between the two groups existed. Group A consisted of younger patients with a higher likelihood of having genetic confirmation of a connective tissue disease (\( P = .006 \)). More patients in group A also had undergone thoracic aortic repairs involving the root, ascending aorta or arch, and descending repairs compared with group B (proximal thoracic: 67% vs 20%; \( P = .01 \); descending thoracic: 80% vs 7%; \( P < .001 \)).

**Mortality.** There were no 30-day deaths. There were four late deaths. Three were unrelated to aortic issues and one resulted on postoperative day 87 from progression of an untreated portion of a prior arch dissection that was remote from the endovascular repair. The three unrelated deaths included congestive heart failure (a condition pre-existing the FEVAR), sepsis, and Wegener granulomatosis. Actuarial survivals at 12, 24, and 36 months were 85%, 85%, 85% for group A and 100%, 100%, 75% for group B (log-rank test, \( P = .50 \); Fig 3).

**Morbidity.** No patients developed spinal cord ischemia, respiratory failure, renal failure, or stroke. The Marfan syndrome patient who died as a result of congestive heart failure on postoperative day 65 had previously undergone multiple cardiac and aortic procedures and had a baseline ejection fraction of 15%. He was undergoing the endovascular TAAA repair (which was his sixth aortic operation) with the intent of subsequently receiving a heart transplant.
There were no endoleaks in group B and 10 primary endoleaks (five type I and five type III) in group A. All endoleaks were to the visceral portion of the repair (see Reintervention below). One secondary type III endoleak occurred due to distal sealing failure at a re-entry site within a dissected right renal artery (Table II).

Of 10 primary endoleaks, eight patients (five type I and three type III) underwent repair, with four patients requiring multiple endovascular procedures (Table III). One patient with a type III endoleak attributed to the right renal artery did not undergo reintervention because of favorable morphologic changes at 6 months (decreased sac size and markedly decreased endoleak volume). Persistent type II endoleaks from multiple intercostal arteries into aneurysmal sac and false lumen were treated with translumbar glue embolization in two of these patients.

**Aneurysm sac behavior.** Of the 21 patients (10 patients in group A and 11 patients in group B) who were monitored for >6 months, no aortic growth was noted in 19 (90%) (8 [80%] in group A and all of group B). Overall mean sac regression was similar between the two groups (−6.8 mm in group A vs −11.4 mm in group B; \( P = .43 \); Fig 4). Both patients with growth in group A had connective tissue diseases (one Marfan syndrome and the other a TGFβR2 mutation), and both underwent FEVAR to treat visceral patch aneurysms that developed after open surgical repairs of type II TAAAs. Both have undergone multiple reinterventions in an effort to control the aneurysm morphology, and one has recently been noted to have a decreasing sac size after his last intervention.

**DISCUSSION**

The current treatment paradigm for TAAA in the setting of a chronic dissection involves open surgical repair, or more recently, hybrid endovascular repair.
Both these techniques are associated with significant risk, and many patients are not candidates for either procedure. The outcomes of open repair or hybrid endovascular procedure for TAAA in literature are reported in Table IV. Hybrid endovascular procedures\textsuperscript{29,32} have been thought to be less invasive than open repair for TAAA because of the minimal chest dissection and lack of aortic cross-clamping. However, the 30-day mortality (8.5%-24%) and incidence of spinal cord ischemia (4.3%-14%) remain significant. Clearly, there is a need for a less invasive solution for dissection with TAAA, and FEVAR seems to be a promising possibility.

FEVAR is an established treatment for nondissected TAAA.\textsuperscript{20,33,34} Concerns specific to dissection repair include the potential for true lumen stent graft compression, inadequate end-organ blood flow to visceral vessels supplied from the false lumen, and more challenging technical issues with implantation.\textsuperscript{35} From our data, it appears that the key to successful false lumen obliteration is to ensure that the endovascular repair extends well above and below the dissection flaps. Thus, in a focal dissection where the flap is limited to a portion of the visceral segment, one simply incorporates the visceral vessels into the repair within the true lumen, the flap and false lumen collapse, and the proximal and distal sealing zones are extended above and below the dissection. Success was noted in all group B patients. There were no instances of graft compression, no endoleaks, and no branch compromise, and repair was always associated with favorable morphologic behavior on follow-up.

This principle is much more difficult to apply in a patient with extensive dissections because the anatomy and patient presentations are markedly more complex. Six patients in group A underwent treatment for proximal (ascending) dissections with interposition grafts in the ascending aorta, with only the “normal segment” of target

### Table I. Patient demographics

<table>
<thead>
<tr>
<th>Variable\textsuperscript{a}</th>
<th>Group A (extensive) (n = 15)</th>
<th>Group B (focal) (n = 15)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>58 ± 11 (33-71)</td>
<td>74 ± 8 (53-84)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Male sex</td>
<td>14 (93)</td>
<td>12 (80)</td>
<td>.28</td>
</tr>
<tr>
<td>Aneurysm diameter, mm</td>
<td>64 ± 13 (43-97)</td>
<td>56 ± 17 (24-93)</td>
<td>.15</td>
</tr>
<tr>
<td>Comorbidities</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>13 (87)</td>
<td>12 (80)</td>
<td>.62</td>
</tr>
<tr>
<td>Hyperlipidemia</td>
<td>7 (47)</td>
<td>12 (80)</td>
<td>.06</td>
</tr>
<tr>
<td>Smoking (ever)</td>
<td>10 (67)</td>
<td>10 (67)</td>
<td>&gt;.99</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>1 (7)</td>
<td>3 (20)</td>
<td>.28</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>2 (13)</td>
<td>11 (73)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Renal insufficiency\textsuperscript{b}</td>
<td>2 (13)</td>
<td>3 (20)</td>
<td>.62</td>
</tr>
<tr>
<td>Prior paraplegia</td>
<td>3 (20)</td>
<td>0</td>
<td>.07</td>
</tr>
<tr>
<td>COPD</td>
<td>0</td>
<td>2 (13)</td>
<td>.14</td>
</tr>
<tr>
<td>Connective tissue disease</td>
<td>6 (40)</td>
<td>0</td>
<td>.006</td>
</tr>
<tr>
<td>Marfan syndrome</td>
<td>4 (27)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Loyez-Dietz syndrome</td>
<td>2 (13)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Aortic disease</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TAAA</td>
<td>15 (100)</td>
<td>5 (33)</td>
<td>.003</td>
</tr>
<tr>
<td>Crawford type</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>I</td>
<td>1 (7)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>II</td>
<td>10 (67)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>III</td>
<td>0</td>
<td>2 (13)</td>
<td></td>
</tr>
<tr>
<td>IV</td>
<td>3 (20)</td>
<td>3 (20)</td>
<td></td>
</tr>
<tr>
<td>Extensive aorta (arch + TAAA II)</td>
<td>1 (7)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Juxtarenal abdominal aorta</td>
<td>0</td>
<td>9 (60)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Common iliac artery</td>
<td>0</td>
<td>1 (7)</td>
<td>.31</td>
</tr>
<tr>
<td>Previous aortic surgery</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Proximal thoracic aortic repair</td>
<td>10 (67)</td>
<td>3 (20)</td>
<td>.01</td>
</tr>
<tr>
<td>Bentall operation</td>
<td>6 (40)</td>
<td>1 (7)</td>
<td></td>
</tr>
<tr>
<td>Ascending aorta with arch repair</td>
<td>4 (27)</td>
<td>2 (14)</td>
<td></td>
</tr>
<tr>
<td>Descending thoracic aortic repair</td>
<td>12 (80)</td>
<td>1 (7)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Open surgery</td>
<td>8 (53)</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>TEVAR</td>
<td>4 (27)</td>
<td>1 (7)</td>
<td></td>
</tr>
<tr>
<td>Abdominal aortic repair</td>
<td>6 (40)</td>
<td>4 (27)</td>
<td>.44</td>
</tr>
<tr>
<td>Open surgery</td>
<td>4 (27)</td>
<td>2 (13)</td>
<td></td>
</tr>
<tr>
<td>EVAR</td>
<td>2 (13)</td>
<td>2 (13)</td>
<td></td>
</tr>
<tr>
<td>Cerebrospinal fluid drainage used</td>
<td>11 (73)</td>
<td>3 (20)</td>
<td>.003</td>
</tr>
</tbody>
</table>

\textsuperscript{COPD}, Chronic obstructive pulmonary disease; \textit{EVAR}, endovascular abdominal aortic repair; \textit{TAAA}, thoracoabdominal aortic aneurysm; \textit{TEVAR}, thoracic endovascular aortic repair.

\textsuperscript{a}Continuous data are shown as mean ± standard deviation (range) and categoric data as number (%).

\textsuperscript{b}Defined as creatinine >1.5 mg/dL.
aorta relegated to the surgical graft within the ascending aorta or other surgically implanted graft. However, in the absence of arch dilation, it seems inappropriate to treat the entire aortic segment. All but one of these patients also had thoracic repairs providing a segment within the thoracic aorta providing a landing zone in that region that was dissection-free, providing an optimal proximal landing zone for an endovascular graft. There were no issues with proximal sealing, fixation, or migration in cases where a prior surgical or endovascular landing zone had been successfully created. When we were forced to use the native aorta for a landing zone, we tried to locate the landing zone in the healthiest segment possible (straight, nonsurgical, nonatheromatous) and maintain at least 2 cm of sealing within the healthiest aorta ascertainable.

Aside from the proximal sealing and fixation zone, there exist three regions of potential failure: the intergraft joints, the interface between a branch and the distal visceral or brachiocephalic artery, and the distal sealing zone. The

Table II. Endoleaks after fenestrated and branched endovascular aortic repair (FEVAR) for chronic type B aortic dissection in group A (no endoleaks in group B)

<table>
<thead>
<tr>
<th>Endoleak</th>
<th>Group A (extensive) (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Primary</td>
<td>10 (66.7)*</td>
</tr>
<tr>
<td>Type I</td>
<td>5 (33.3)</td>
</tr>
<tr>
<td>Type IA</td>
<td>1 (6.7)</td>
</tr>
<tr>
<td>Type IB</td>
<td>4 (26.6)</td>
</tr>
<tr>
<td>Type III</td>
<td>5 (33.3)</td>
</tr>
<tr>
<td>Secondary</td>
<td>1 (6.7)</td>
</tr>
</tbody>
</table>

SMA, Superior mesenteric artery.

*Staged endoleaks (two endoleaks were found separately) (n = 3).

latter situation becomes challenging when the dissection extends into and beyond the iliac vessels, often necessitating the incorporation of the internal iliac arteries into the repair. In such a case, we found that the off-the-shelf helical iliac branch device (Cook Medical Inc) version worked well with respect to the ability to cannulate the internal iliac artery within small true lumens (smallest was 5 mm) while allowing for treatment deep into the dissected internal iliac artery systems using self-expanding stent grafts.

When the dissection extends deep into a visceral vessel, particularly when it involves secondary and deeper branches, the repair becomes more challenging. Unlike dissection within the internal iliac artery, where the anterior or posterior division may be sacrificed and a seal established within one of the internal iliac trunks, the side branches on the visceral vessels may not be occluded without potentially significant repercussion (mesenteric or renal insufficiency). Unfortunately, we have no optimal solution in such circumstances. If a seal cannot be established solely within the true lumen, the objective becomes preventing aneurysm perfusion derived from retrograde flow through the false lumen.

One patient with dissection deep into the left renal and superior mesenteric arteries (SMAs) required an innovative solution. The left renal dissection was amenable to extension of the covered stents deep into the artery to the level of the renal bifurcation. However, the SMA dissection extended to the ileocolic vessel, and the false lumen supplied many proximal small intestinal branches. Thus, once the aortic repair was completed and the SMA branch reinforced with two stent grafts, a lumbar puncture was performed. The false lumen of the SMA was accessed and occluded using embolization techniques (Fig 5). This excluded the aortic aneurysm, but the fate of the SMA

Fig 3. Actuarial survival curves of fenestrated and branched endovascular aortic repair (FEVAR) in the Kaplan-Meier analysis are shown for group A (n = 15; black line) and group B (n = 15; dotted line), with the error bars showing the standard error.

Table III. Reinterventions after fenestrated and branched endovascular aortic repair (FEVAR) for chronic type B aortic dissection in group A (no reinterventions in group B)

<table>
<thead>
<tr>
<th>Reintervention</th>
<th>Group A (extensive) (n = 15)</th>
</tr>
</thead>
<tbody>
<tr>
<td>First reintervention</td>
<td>8 Iliac leg extension</td>
</tr>
<tr>
<td>Type I</td>
<td>5 Hypogastric interruption</td>
</tr>
<tr>
<td>Type III</td>
<td>3 Celiac artery stent</td>
</tr>
<tr>
<td>Second reintervention</td>
<td>4 Renal artery stent with IMA CE*</td>
</tr>
<tr>
<td>Type III</td>
<td>4 Renal artery re-PTA</td>
</tr>
<tr>
<td>Third reintervention</td>
<td>2 Translumbar CE</td>
</tr>
</tbody>
</table>

CE, Coil embolization; IMA, inferior mesenteric artery; PTA, percutaneous transluminal angioplasty; TEVAR, thoracic endovascular aortic repair.

*Coil or glue embolization.
remains uncharted. Dissection deep into the visceral vessels is generally not an access issue, because there is usually a fenestration created by the dissection or one can create an endovascular fenestration, but the long-term management of such vessels is suspect and conceptually leaves much to be desired. Although this prevents growth of the aortic aneurysm, visceral dilation is not necessarily treated with this method.

The interface between graft components can also pose challenges. There are many components in these repairs that frequently transcend extremely tortuous anatomy. Optimally, extensive overlap between components will eliminate joint endoleaks and provide durable repairs, but luminal constraints and target vessel size often prevent this design. The true lumen in a chronic dissection is typically obliquely shaped and narrow, creating technical challenges with stent design and implantation. Thus, in some cases, it seems helpful to optimize the true lumen to allow placement of a branched device. After our experience with TEVAR for chronic thoracic dissections, we used a design strategy of using a long straight (nontapered) graft for the thoracic portion of the disease. The procedure was then staged by a period of 2 months, with the intent of allowing the true lumen to expand but also potentially minimizing the risk of spinal cord ischemia. However, this is only a recent strategy and was used in one of the patients in this series, although many of the group A patients would be considered staged with respect to their surgical thoracic repairs. Consistently, in surgical or endovascular thoracic repairs of chronic dissection, we have seen expansion of the true lumen near the distal seal, but more distally, slow aortic growth is the rule rather than the exception. Thus, in cases where we considered the true lumen in the visceral segment to be too small to form or manipulate catheters, we defaulted to a staged repair whereby TEVAR would initially be completed to the level of the celiac artery.

Extensive dissections, particularly in the setting of the syndromic connective tissue diseases, add yet another level of complexity. The aortic wall in patients with connective tissue disease like Marfan syndrome or Loyez-Dietz

Table IV. Outcomes after open repair or hybrid endovascular repair for thoracoabdominal aortic aneurysm (TAAA) in literature

<table>
<thead>
<tr>
<th>First author</th>
<th>Year</th>
<th>Patients, No.</th>
<th>Operative procedure</th>
<th>Mortality, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cowan25</td>
<td>2003</td>
<td>1542</td>
<td>Open</td>
<td>NA</td>
</tr>
<tr>
<td>Rigberg26</td>
<td>2006</td>
<td>1010</td>
<td>Open</td>
<td>19</td>
</tr>
<tr>
<td>Conrad27</td>
<td>2007</td>
<td>445</td>
<td>Open</td>
<td>6.8</td>
</tr>
<tr>
<td>Coselli28</td>
<td>2007</td>
<td>2286</td>
<td>Open</td>
<td>5</td>
</tr>
<tr>
<td>Smith29</td>
<td>2011</td>
<td>24</td>
<td>Hybrid</td>
<td>12.5</td>
</tr>
<tr>
<td>Lin30</td>
<td>2012</td>
<td>58</td>
<td>Hybrid</td>
<td>24</td>
</tr>
<tr>
<td>Hughes31</td>
<td>2012</td>
<td>47</td>
<td>Hybrid</td>
<td>8.5</td>
</tr>
<tr>
<td>Oderich32</td>
<td>2012</td>
<td>159</td>
<td>Hybrid</td>
<td>16</td>
</tr>
</tbody>
</table>

NA, Not available; SCI, spinal cord ischemia.
syndrome is fragile, and one may question the treatment using endovascular techniques. Yet, many of these patients are considered nonsurgical candidates and relegated to die from their dissections or aneurysms. Endovascular repairs have been attempted. Marcheix et al\textsuperscript{37} reported the outcomes of stent grafting of the dissected descending aorta in 15 patients with Marfan syndrome. Primary endoleaks occurred in 5 patients, secondary endoleaks developed in 5, and 7 required conversion to open repair.

Our series included six patients with diagnosed connective tissue diseases. Five of these 6 patients had undergone proximal and distal aortic repairs, providing acceptable landing zones, yet continued growth of untreated aorta was noted over time. Ultimately, it appears that if the patients live long enough, they will require total aortic replacement. The patch aneurysms that were treated did not enjoy a favorable morphologic response and represent a challenging group of patients to deal with. Open surgical techniques have evolved to eliminate the patch in such patients and branches are individually sewn to each vessel given the likelihood of patch failure in connective tissue disease patients.

There were no conversions to open surgery, partly because the patients were considered nonsurgical candidates preoperatively, which is why we are continuing to monitor the one remaining patient with growth and the second patient who only recently was noted to have sac size decrease. Interestingly, both patients had prior open repairs of type II TAAA and presented with large (8-10 cm) patch aneurysms. After endovascular repair, the patch aneurysms continued to expand. Obvious questions include the integrity of the wall in these patients and the ability to successfully protect from rupture using endovascular techniques. One of the two patients suffered an SMA stent separation (multiple stents were required to bridge the gap between the aortic device and the visceral

Fig 5. A persistent endoleak through the distal re-entry of the superior mesenteric artery (SMA; *) resulted in retrograde filling into the false lumen (white arrows) after fenestrated endovascular aneurysm repair for chronic type B aortic dissection is shown in a (A) computed tomography (CT) and (B) angiogram. The translumbar embolization using coils and AMPLATZER Vascular Plug (St. Jude Medical, St. Paul, Minn) plugs (black arrows) was performed to occlude the false lumen near the origin of the SMA. The angiogram shows the (C) right anterior oblique view and (D) the frontal view.
vessel), which, when treated, resulted in some sac size decrease.

Of the 15 patients in group A, there were 10 primary endoleaks (5 type I and 5 type III, 3 in connective tissue disease patients). These leaks were all detected on CT scanning, likely not appearing on angiographic studies given the extensive length of the repair with an inability to image the entire region without excessive amounts of contrast. After the CT diagnosis, efforts were made to repair any type I or III endoleak, which in some patients required multiple procedures. Such patients must understand that multiple procedures and detailed follow-up will be necessary. Yet, alternative open surgical techniques suffer from similar shortcomings.

CONCLUSIONS

FEVAR is feasible for patients with chronic type B aortic dissections with TAAAs. Focal dissections have favorable results, provided that adequate landing zones are located above and below the dissected tissue. The patients with extensive dissections, particularly those with connective tissue diseases and those with dissection deep into the visceral arteries, remain challenging. Such patients will often require multiple procedures, but when aneurysm exclusion is achieved, the aortic morphologic changes appear to be favorable. The behavior of the chronically dissected false lumen appears to parallel that of the more acutely dissected aorta when all of the false lumen fenestrations are properly occluded, implying that a watch-and-wait philosophy is reasonable in the setting of an acute dissection where there is only a potential for the development of a visceral segment aneurysm. This becomes a particularly sound strategy when there are minimally invasive options capable of treating the chronically dissected TAAA.

AUTHOR CONTRIBUTIONS

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Analysis and interpretation: AK, RG
Data collection: AK, RG
Writing the article: AK, RG, ME, TM, ER
Critical revision of the article: AK, RG, ME, TM, ER
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