Spontaneous Delayed Sealing in Selected Patients with a Primary Type-Ia Endoleak After Endovascular Aneurysm Repair


WHAT THIS PAPER ADDS
This is the first publication specifically analyzing the outcome of untreated primary type-Ia endoleaks. In patients with adequate anatomy planning and execution, most will seal spontaneously and not recur, which is novel insight into the natural history of this type of complication. Study limitations do not allow for strong recommendations, but suggest a role for conservative management of self-limiting primary type-Ia endoleaks in selected cases.

Objective: Direct additional therapy is advised for type-Ia endoleaks detected on completion angiography after endovascular aneurysm repair (EVAR). Additional intraoperative endovascular procedures are, however, often challenging or not possible, and direct open conversion is unattractive. The results of a selective, conservative strategy for patients with primary type-Ia endoleak has been analysed.

Methods: This was a retrospective, single-centre study (UMC, Utrecht, NL). From 2004 to 2008, all patients with a primary type-Ia endoleak and suitable anatomy for EVAR, stentgraft oversizing ≥15%, and optimal deployment were included. Complications during follow-up were studied and all sequential CTA scans were reviewed. These were compared with the remaining patients, treated during the same period.

Results: Fifteen patients were included (14 male, median age 77, range 67–85) with a median aneurysm diameter of 60 mm (48–80), an aneurysm neck diameter of 26 mm (21–32), a neck length of 29 mm (11–39), and infrarenal angulation of 49° (31–90). One patient suffered rupture 2 days after EVAR — leading to the only AAA-related death. Eight of the 15 type-Ia endoleaks disappeared spontaneously on the first postoperative CTA, obtained within 1 week of EVAR. On the second postoperative CTA, obtained a median of 5 months (1–12) after EVAR, all remaining endoleaks had sealed. One recurrence occurred at 4.85 years. During a median follow-up of 3.3 years, there were five secondary interventions. Compared with controls, there were more secondary (or recurrent) type-Ia endoleaks (13% vs. 4%), endograft migrations (13% vs. 3%), sac growths (33% vs. 16%), and secondary interventions (33% vs. 23%). None of these differences however, were statistically significant.

Conclusions: All but one of the primary type-Ia endoleaks sealed spontaneously. Until sealing, the risk of rupture persisted, but subsequently only one recurrence of type-Ia endoleak was seen. In selected patients, a conservative approach for primary type-Ia endoleaks may be justified.

INTRODUCTION
The aim of endovascular aneurysm repair (EVAR) is to prevent aneurysm rupture by placement of a stentgraft, thereby eliminating pressure on the wall of the aneurysm. The proximal fixation and sealing of the stentgraft is often considered the Achilles’ heel of the EVAR procedure. Incomplete proximal sealing results in a proximal type-I endoleak in about 4% of all patients treated with EVAR.

5 A proximal type-I endoleak can be dangerous as high-pressure blood flow persists in the aneurysm sac. Primary type-Ia endoleaks are proximal type-I endoleaks visible immediately after stentgraft deployment on the completion angiogram. Prompt treatment for these endoleaks is advocated, either by additional endovascular means (ballooning or placement of a cuff or balloon expandable stent) or by conversion to open surgery. Endovascular correction of the endoleak is, however, often challenging and even impossible in many cases, either because there is no possibility to extend the stentgraft proximally without compromise of renal or mesenteric vessels or because the diameter of the neck is too large for additional (balloon expandable) stent placement. Immediate conversion to open surgery, on the other hand, is associated with a high
mortality rate of up to 20–40%, although this rate has declined over the years.9,10 Spontaneous sealing of primary type-Ia endoleaks is also anecdotally reported, but many believe that even spontaneously sealed type-I endoleaks require therapy, as the chance of recurrence is unacceptably high.11

We hypothesised that if a patient’s anatomy (specifically neck length, diameter, tortuosity angles, and shape) is considered suitable for EVAR, preoperative measurements have been performed precisely (with the use of a centre lumen line), the stentgraft is ≥15% oversized (using outer-to-outer diameter measurements as reference) and the stentgraft is deployed at the optimal position below the lowermost renal artery, primary type-Ia endoleaks may resolve spontaneously in most cases. Furthermore, these type-Ia endoleaks may not recur, making additional treatment unnecessary. The objective of this study was to analyse the results of a selective conservative approach for primary type-Ia endoleaks in patients with EVAR-suitable anatomy and adequate stentgraft sizing and deployment.

METHODS

From August 2004 to December 2008, 285 patients underwent planned endovascular aortic procedures at the University Medical Center, Utrecht, The Netherlands. Patients treated for isolated iliac aneurysms, anastomotic or mycotic aneurysms, non-aneurysmal disease, and those with prior abdominal aortic surgery were excluded. This resulted in a cohort of 216 patients. Criteria for treatment were maximum aortic diameter greater than 55 mm, fast growth (>5 mm in 6 months or 10 mm in 1 year), or presence of symptoms. According to the local EVAR protocol, all AAA patients underwent preoperative imaging by computed tomographic angiography (CTA). Preoperative stentgraft sizing was performed, using dedicated software with centre lumen line reconstructions (CLL) for optimal diameter and length measurements during the entire study period, and stentgraft oversizing of 15–20% was considered ideal. Postoperatively, CTAs were performed within 1 week after EVAR (typically at 48–72 hours, before discharge), and yearly thereafter. Additional scans were performed as indicated by the treating physician. All patient data were entered prospectively into a dedicated database of EVAR patients.

Patient selection

A retrospective analysis of the prospective database was performed. All patients with an intraoperative clear proximal type-I endoleak after stentgraft placement and ballooning of the sealing and overlap zones were identified and included in the study group. The remaining patients were included in a control group for comparison of complications and secondary interventions.

Patients with a type-Ia endoleak were only left untreated (and consequently included in this study) if the anatomy of the neck was considered suitable for EVAR, if preoperative stentgraft sizing was performed with the use of a CLL, if the stentgraft was correctly oversized, and if the stentgraft was deployed at the intended, optimal position. The anatomy of an aneurysm neck was considered suitable for EVAR if the length was ≥10 mm, the diameter was ≤32 mm, the infrarenal angulation was ≤60°, and ≤50% of the circumference consisted of thrombus or calcification. Moreover, patients with infrarenal angulation of 60–90° were also considered suitable if the aneurysm neck length was ≥15 mm.

Evaluation

Age, gender, symptoms at presentation, and intraoperative values were noted. Intraoperative values analysed were stentgraft size and type, operation time, volume of contrast agent used, total minutes of fluoroscopy, estimated total blood loss, administration of anticoagulants, activated clotting time, and procedure-related problems. Furthermore, the postoperative course and possible complications or re-interventions were investigated. All pre- and postoperative CTA scans of included patients were transferred to a workstation (3Mensio Medical Imaging B.V., Bilthoven, The Netherlands) for re-evaluation.

CTA scans were performed on a 64-slice helical CT scanner (Philips Medical Systems, Best, The Netherlands) with a standardised acquisition protocol. Slice thickness was 0.9 mm and radiation exposure parameters were 120 kVp and 300 mAs, resulting in a CT dose index (CTDIvol) of 17.6 mGy. Early and late arterial phase scans were obtained.

Measurement

Preoperative CTAs were reviewed for maximum AAA diameter, total aneurysm volume, neck length, neck diameter, supra- (α) and infrarenal (β) neck angulation, and calcification and thrombus lining the neck. All available postoperative CTAs were investigated for maximum AAA diameter, total aneurysm volume, the presence of endoleaks, and stentgraft migration.

All measurements were performed using CLL reconstructions. Volume and angle measurements were performed according to earlier published protocols.12,13 The neck diameter was measured 1 cm caudal to the lowermost renal artery. The presence of calcification and thrombus in the aneurysm neck were also visually quantified at this level. Stentgraft migration was defined as a migration of the most proximal stentgraft ring of ≥10 mm according to the reporting standards for EVAR.1

Endpoints

The primary endpoint was persistent sealing of the type-Ia endoleak. Secondary endpoints were freedom from; secondary intervention, secondary type-Ia endoleak, migration, post-implantation sac growth, AAA-related death, and all-cause death.
**Baseline characteristics**

Basel characteristics were described as counts and percentages (dichotomous variables), or medians and range (continuous variables). Differences were assessed using Fisher’s exact test or Mann–Whitney U test, as indicated. Differences in endograft model were tested with Person’s chi-square test. All tests were two-sided and significance was considered when \( p < 0.05 \). Statistical analysis was performed using the IBM SPSS Statistics 20 (IBM Inc., Chicago, IL, USA).

**RESULTS**

Fifteen patients with a primary type-Ia endoleak met the inclusion criteria (14 male, median age 77, range 67–85, 6.9% of all EVAR patients, Table 1). Fourteen patients were asymptomatic at presentation, and one patient presented with a symptomatic intact AAA (case 10). The preoperative median aneurysm diameter was 60 mm (48–80) and the aneurysm sac volume was 217 ml (116–552) (Table 2). The median neck diameter was 26 mm (21–32) and neck length 29 mm (11–39). The suprarenal angle (\( \alpha \)) was 31° (13–82) and the infrarenal angle (\( \beta \)) was 49° (31–90). During the study period, there were no direct conversions to open repair and one patient received an aortic cuff to treat a type-Ia endoleak caused by low-deployment. No primary type-Ia endoleaks were left untreated in the control group.

Thirteen patients were treated with a Talent bifurcated stentgraft (Medtronic, Minneapolis, MN, USA), one with both a Talent bifurcated and a Talent aorto-uni-iliac device and one patient with a Zenith bifurcated stentgraft (Cook, Bloomington, IN, USA). Significant differences were observed in the type of grafts used in the study and control groups, where the distribution of types of graft was greater (Table 1). The median stentgraft oversizing was 21% (10–31) and eight patients (53%) were treated with an endograft sized \( \geq 32 \) mm proximally. Patient 11 had an hourglass neck 39 mm in length, and oversizing was 15–20% in the mid-section, despite a 10% oversizing only in the proximal 10 mm.

**Table 1. Baseline characteristics.**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study group ( N = 15 )</th>
<th>Control group ( N = 201 )</th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age — median (range)</td>
<td>76 (67–85)</td>
<td>74 (47–89)</td>
<td>.086</td>
</tr>
<tr>
<td>Male gender — N (%)</td>
<td>14 (93)</td>
<td>180 (90)</td>
<td>1.0</td>
</tr>
<tr>
<td>ASA III/IV — N (%)</td>
<td>9 (60)</td>
<td>116 (58)</td>
<td>1.0</td>
</tr>
<tr>
<td>Endograft model</td>
<td></td>
<td></td>
<td>.01</td>
</tr>
<tr>
<td>Talent bif, N (%)</td>
<td>13 (86)</td>
<td>115 (57)</td>
<td></td>
</tr>
<tr>
<td>Talent AUI, N (%)</td>
<td>1 (7)</td>
<td>7 (3)</td>
<td></td>
</tr>
<tr>
<td>Zenith, N (%)</td>
<td>1 (7)</td>
<td>2 (1)</td>
<td></td>
</tr>
<tr>
<td>Excluder, N (%)</td>
<td>—</td>
<td>43 (21)</td>
<td></td>
</tr>
<tr>
<td>Endurant, N (%)</td>
<td>—</td>
<td>34 (17)</td>
<td></td>
</tr>
</tbody>
</table>

Bif = bifurcated; AUI = aorto-uni-iliac.

All patients received 5000 units of heparin just before catheterisation. The activated clotting time (ACT) was maintained \( \geq 2 \) times normal during all procedures. Stentgraft deployment was successful and uncomplicated in 14 patients. In one patient a bifurcated stentgraft was inadvertently deployed 2 cm below the intended position (case number 7). An aorto-uni-iliac stentgraft was subsequently placed through the bifurcated stentgraft at the intended proximal position, followed by a contralateral iliac occluder and a femoro-femoral crossover bypass. As the second stentgraft was placed at the optimal position, this patient was still included in the study group. Blood loss was \(<150 \) cc during all EVAR procedures and the median operation time was 120 minutes (80–173). Median contrast volume used was 110 ml (70–170), and median fluoroscopy time (in half-dose setting) was 20 minutes (8–43). All 15 patients had a clear, unmistakable, proximal type-I endoleak at the completion angiogram, which was verified by two of three vascular surgeons with extensive experience in EVAR (HV, FM or JvH) (Fig. 1).

In all patients a CTA was obtained within 1 week of EVAR, and all had a minimum of 1 yearly CTA during postoperative surveillance. The type-Ia endoleak had disappeared spontaneously on the first postoperative CTA in 8 of the 15 patients (Table 3). In the other seven patients, a clear type-Ia endoleak was still seen. In one of these patients the...
aneurysm ruptured 2 days after EVAR, causing death before treatment was possible (case 7). This patient had lumbar pain and hypotension, which led to a CTA investigation 48 hours postoperatively, where a large type-Ia endoleak and a posterior retroperitoneal rupture were evident. On the second postoperative CTA, obtained a median of 5 months (1–12) after EVAR, no more type-Ia endoleaks were seen.

An unmistakable recurrence of a type-Ia endoleak was observed in one patient (case 9), 4.85 years after EVAR (Table 4). This was associated with dilatation of the proximal neck beyond the diameter of the implanted endograft. At this time, the previously stable aneurysm sac exhibited growth, and the patient was successfully converted to open repair. In a second patient, sac growth was noted 1.85 years after EVAR (case 13). As a result of neck dilatation and proximal migration (<10 mm) in a previously angulated neck (65° preoperatively), a proximal endoleak was not imaged, but was suspected because of the short proximal seal. This patient underwent successful implantation of a proximal extension cuff. No other recurrences were observed.

After discharge, the median follow-up was 3.27 years (range 0.58–5.52). During this period, five patients had secondary interventions (Table 4). Cases 9 and 13 were for recurrent type-Ia endoleak (case 9) and insufficient proximal fixation and sealing without endoleak (case 13) and have been described above. One patient underwent secondary intervention after 2 years for an aneurysm diameter growth of 16 mm (volume increase of 136 mL in combination with an aortic neck dilation of 8 mm) in the absence of an endoleak (case 1). Conversion to open repair was performed without further complications. During the conversion procedure, there was no visible endoleak when opening the aneurysm sac. One other patient underwent implantation of a proximal extension cuff 1.25 years after EVAR for stentgraft migration of 10 mm. This was probably caused by a neck dilation of 5 mm (case 10). Stentgraft migration occurred in one other patient after 1.85 years (case 12). This was successfully treated by implantation of an aorto-uni-iliac device.

Six patients died during follow-up (Table 3): one as a result of a ruptured aneurysm 2 days after EVAR; three from cancer; one from infection; and one the cause for which could not be determined (Table 3). No other AAA-related deaths occurred in the study group.

In comparison with the control group, patients included in the study group had shorter follow-up time, and shorter time to first intervention, although these differences were not statistically significant (Table 5). There was a greater proportion of patients suffering from secondary (or recurrent) type-Ia endoleak (13% vs. 4%), endograft migration (13% vs. 3%), and sac growth (33% vs. 16%). Similarly, a greater proportion required secondary intervention (33% vs. 23%) and particularly conversion to open repair (13% vs. 5%). None of these differences however, was statistically significant.

**DISCUSSION**

In this study, the conservative management of primary type-Ia endoleaks in patients with adequate anatomy, planning, and implantation led to spontaneous resolution in most cases. A watchful attitude towards primary type-Ia endoleaks in selected patients may be preferable to immediate conversion or complex endovascular techniques. However, our data suggest an increased risk of late complications and secondary interventions that must be factored into the decision of whether to observe or intervene.

The natural history of an untreated type-Ia endoleak remains undetermined. Although common sense suggests that rupture risk is the same as the risk of an untreated AAA of similar diameter, Venermo et al. suggest that EVAR offers protection from rupture despite the presence of sac pressurisation. These authors suggest that growth, rather than the presence of endoleak, is a better predictor of rupture after EVAR. In their series of 21 patients with untreated type-Ia endoleak, only one rupture occurred (2.5 years after EVAR) after a 2 cm enlargement. In this study, one rupture occurred 2 days after EVAR. The cause for this cannot be fully explained. An iatrogenic lesion of the aneurysm wall cannot be ruled out, as the procedure was complex and the temporal relationship between implant and rupture is unusual.
Table 3. Outcome of patients after conservative management of primary type-Ia endoleaks.

<table>
<thead>
<tr>
<th>Pt</th>
<th>Sp. seal (d)</th>
<th>Rec 1aEL (y)</th>
<th>Migration (y)</th>
<th>Sac growth (y)</th>
<th>AAA rupt (y)</th>
<th>Total FU (y)</th>
<th>Death (y)</th>
<th>Death cause</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Y (51)</td>
<td>N</td>
<td>N</td>
<td>Y (1.96)</td>
<td>N</td>
<td>5.52</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>2</td>
<td>Y (2)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y (1.96)</td>
<td>5.51</td>
<td>Y (5.51)</td>
<td>Cancer</td>
</tr>
<tr>
<td>3</td>
<td>Y (112)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>4.71</td>
<td>Y (4.71)</td>
<td>Cancer</td>
</tr>
<tr>
<td>4</td>
<td>Y (2)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>4.05</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>5</td>
<td>Y (370)</td>
<td>N</td>
<td>N</td>
<td>Y (1.99)</td>
<td>N</td>
<td>3.56</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>Y (134)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>2.02</td>
<td>N</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>Y (0.01)</td>
<td>0.01</td>
<td>Y (0.01)</td>
<td>Early rupture</td>
</tr>
<tr>
<td>8</td>
<td>Y (3)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>0.58</td>
<td>Y (0.58)</td>
<td>Undetermined</td>
<td></td>
</tr>
<tr>
<td>9</td>
<td>Y (147)</td>
<td>Y (4.85)</td>
<td>N</td>
<td>Y (4.85)</td>
<td>N</td>
<td>5.31</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>10</td>
<td>Y (4)</td>
<td>N</td>
<td>Y (1.25)</td>
<td>N</td>
<td>N</td>
<td>2.29</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>11</td>
<td>Y (2)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>2.07</td>
<td>Y (2.07)</td>
<td>Infection</td>
<td></td>
</tr>
<tr>
<td>12</td>
<td>Y (2)</td>
<td>N</td>
<td>Y (1.85)</td>
<td>N</td>
<td>4.07</td>
<td>N</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>13</td>
<td>Y (2)</td>
<td>? (2.05)</td>
<td>N</td>
<td>Y (2.05)</td>
<td>N</td>
<td>2.97</td>
<td>N</td>
<td>—</td>
</tr>
<tr>
<td>14</td>
<td>Y (2)</td>
<td>N</td>
<td>N</td>
<td>N</td>
<td>2.17</td>
<td>Y (2.17)</td>
<td>Cancer</td>
<td></td>
</tr>
<tr>
<td>15</td>
<td>Y (238)</td>
<td>N</td>
<td>N</td>
<td>Y (1.65)</td>
<td>N</td>
<td>1.67</td>
<td>N</td>
<td>N</td>
</tr>
</tbody>
</table>

*d* = days; *FU* = follow-up; *Pt* = patient; *Rec 1aEL* = recurrent type-Ia endoleak; *rupt* = rupture; *Sp.* = spontaneous; *y* = years; ? = unconfirmed.

The cause of primary type-1a endoleaks in well planned and executed cases is most likely to be multifactorial. Coagulation abnormalities (antiplatelet + intraoperative anticoagulation with ACT >2), morphological aspects of the neck (shape, angulation, irregularities caused by thrombus or plaques), and structural characteristics of the endograft may contribute to varying degrees. The spontaneous disappearance of type-1a endoleaks after appropriate stentgraft sizing and deployment in this specific patient group is not surprising, once the coagulation abnormalities are (at least partially) corrected and the self-expanding nature of the stentgrafts leads to gradual neck remodeling and improved graft-wall apposition. Late-resolution endoleaks (beyond 1 week) occurred in patients who were either chronically anticoagulated (cases 5 and 15), had angulated and relatively short necks (cases 1 and 3), or had an irregular wall due to thrombus (case 6) (Table 2).

Anecdotal reports have suggested a high chance of recurrence after sealing of primary type-1a endoleaks has occurred.\(^5,15,16\) Only one recurrence was detected after 5 years, probably caused by progression of disease. It is believed that this low recurrence rate is directly associated with the criteria for watchful waiting. Data suggest, however, that patients with spontaneously sealed proximal endoleaks are at high risk of complications and more likely to require secondary interventions. Therefore, particular attention to image surveillance and a low threshold for intervention is advisable, especially for endoleaks persisting beyond 1 week. The subgroup of patients with angulation 60–90° appeared to be at higher risk of complications (Tables 2 and 3), but the contribution of the previously existing type-Ia endoleak is not dissociable from the higher risk resulting from less favourable anatomy alone.

When a device is placed at the intended position just below the lowermost renal artery and a type-1a endoleak persists despite re-ballooning, alternatives to immediate conversion or vigilance are scarce. Placement of a proximal extension cuff is redundant, and (balloon expandable) stent placement (such as “giant” Palmaz stent [Cordis Corp, Miami Flakes, FL, USA]) is limited by maximum diameter and complexity.\(^15\) The Chimney technique potentially allows the resolution of type-1a endoleaks with reasonable results in the mid-term\(^17,18\) and endostaples (Aptus Endosystems, Sunnyvale, CA, USA) are also available, and may be used to enhance proximal fixation and seal. Early experience with this technology suggests high technical success rates and promising results, but the durability of this adjunct remains unknown.\(^19\) Off-the-shelf fenestrated or branched devices have recently been marketed or are under development by most major manufacturers, and may provide a better solution in patients with appropriate anatomy.

Table 4. Secondary interventions after conservative management of primary type-Ia endoleaks.

<table>
<thead>
<tr>
<th>Pt</th>
<th>Sec. interv. (y)</th>
<th>Cause for sec. interv.</th>
<th>Type of sec. interv</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Y (1.96)</td>
<td>Sac growth without visible EL</td>
<td>Conversion to open</td>
</tr>
<tr>
<td>2</td>
<td>Y (0.01)</td>
<td>Rupture</td>
<td>Conversion to open</td>
</tr>
<tr>
<td>9</td>
<td>Y (4.85)</td>
<td>Proximal type-Ia EL</td>
<td>Conversion to open</td>
</tr>
<tr>
<td>10</td>
<td>Y (1.25)</td>
<td>Migration without visible EL</td>
<td>Proximal extension</td>
</tr>
<tr>
<td>12</td>
<td>Y (1.85)</td>
<td>Migration without visible EL</td>
<td>Conversion to AUI</td>
</tr>
<tr>
<td>13</td>
<td>Y (2.05)</td>
<td>Sac growth without visible EL</td>
<td>Proximal extension</td>
</tr>
</tbody>
</table>

Sec. interv = secondary intervention; *y* = years.

Limitations

Firstly, the study sample is small and consequently the number of events for individual endpoint analysis is restricted. The absence of statistical significance between the study and control groups should be interpreted as a likely type-1 statistical error, and not as proof of equivalence. Also, the majority of patients were treated with the Talent endograft, which is stiffer and has less efficient fixation than most modern alternatives. In these patients, AAA neck configuration was not evaluated, and could help explain the occurrence of primary type-Ia endoleaks in...
otherwise favourable conditions. Lastly, the exact proportion of patients treated successfully with re-ballooning of the proximal attachment site could not be determined.

**Conclusion**

All but one of the primary type-1a endoleaks after EVAR in this specific 15 patient group sealed spontaneously and only one clear recurrence was observed, after 5 years. Although the data suggest that these patients may be at higher risk of complications and may require more secondary interventions, a conservative approach may be justified under strict circumstances, especially when endovascular options are not feasible and direct conversion to open repair is considered high-risk. Future off-the-shelf devices allowing for extension of the proximal sealing zone may present a more durable and elegant solution.

**ACKNOWLEDGEMENTS**

The authors would like to acknowledge the late Jasper van Keulen for his contribution for this study.

**CONFLICT OF INTEREST**

Prof. Hence J. M. Verhagen, Prof. Frans L. Moll and Dr. Joost A. van Herwaarden have received consulting fees from Medtronic AVE.

**FUNDING**

None.

**REFERENCES**


**Table 5.** Comparison between patients after conservative management of primary type-la endoleaks and the remaining EVAR-treated population.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Study group (N = 14)</th>
<th>Control group (N = 201)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total FU (y), median (range)</td>
<td>3.27 (0.58–5.52)</td>
<td>3.93 (0.09–8.88)</td>
<td>.652</td>
</tr>
<tr>
<td>Time to first complic., median (range)</td>
<td>2.03 (0.58–5.51)</td>
<td>2.92 (0.00–8.88)</td>
<td>.824</td>
</tr>
<tr>
<td>Type-la endoleak during FU, N (%)</td>
<td>1 (7)</td>
<td>8 (4)</td>
<td>.843</td>
</tr>
<tr>
<td>Migration, N (%)</td>
<td>2 (13)</td>
<td>5 (3)</td>
<td>.078</td>
</tr>
<tr>
<td>Conversion to open repair, N (%)</td>
<td>2 (13)</td>
<td>9 (5)</td>
<td>.172</td>
</tr>
<tr>
<td>Post-implant rupture, N (%)</td>
<td>1 (7)</td>
<td>1 (0.5)</td>
<td>.134</td>
</tr>
<tr>
<td>Sac growth, N (%)</td>
<td>5 (33)</td>
<td>32 (16)</td>
<td>.145</td>
</tr>
<tr>
<td>Secondary intervention, N (%)</td>
<td>5 (33)</td>
<td>46 (23)</td>
<td>.354</td>
</tr>
</tbody>
</table>

\( y = \text{years.} \)

\( ^a \) Only considering discharged patients.
