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Treatment of Post-implantation Aneurysm Growth by Laparoscopic Sac Fenestration: Long-term Results

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WHAT THIS PAPER ADDS

- Sac growth after endovascular aneurysm repair, reportedly occurring in 40% of patients, is an important finding which may influence prognosis. In case of sac growth and the presence of type II endoleaks or endotension, clipping of side branches and subsequent sac fenestration was suggested in the past as a therapeutic alternative. Long-term efficacy of this procedure is unknown. The current study provides the single largest case series and longest follow-up after this procedure. Based on our results, sac fenestration is not advisable as primary treatment in patients suffering from sac growth in the presence of type II endoleaks or endotension.

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ABSTRACT

Objectives: Sac growth after endovascular aneurysm repair (EVAR) is an important finding, which may influence prognosis. In case of a type II endoleak or endotension, clipping of side branches and subsequent sac fenestration has been presented as a therapeutic alternative. The long-term clinical efficacy of this procedure is unknown.
Methods: The study included eight patients who underwent laparoscopic aortic collateral clipping and sac fenestration for enlarging aneurysms following EVAR. Secondary interventions and clinical outcome were retrieved from hospital records. Sac behaviour was evaluated measuring volumes on periodical computed tomography angiography (CTA) imaging using dedicated software.
Results: Follow-up had a median length of 6.6 (range 0.6–8.6) years. During this time, only three patients successfully achieved durable aneurysm shrinkage ($n = 2$) or stability ($n = 1$). The remaining patients suffered persistent ($n = 2$) or recurrent sac growth ($n = 3$), all regarded as failure of fenestration. A total of six additional interventions were performed, comprising open conversion ($n = 2$), relining ($n = 1$) and implantation of iliac extensions ($n = 3$). All additional interventions were successful at arresting further sac growth during the remainder of follow-up.
Conclusions: Despite being a less invasive alternative to conversion and open repair, the long-term outcome of sac fenestration is unpredictable and additional major procedures were often necessary to arrest sac growth.

Endovascular aneurysm repair (EVAR) of abdominal aortic aneurysm (AAA) has developed since 1991[1] and is now frequently the preferred method of treatment. The ultimate goal of EVAR is to prevent death from aneurysm rupture by excluding the aneurysm

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sac from the circulation, thereby relieving it from pressure. After EVAR, most aneurysms will stabilise or shrink in diameter. Some aneurysms, however, will continue to expand.[2–4]

Continued sac expansion after EVAR can have several explanations, but endoleaks and graft porosity (endotension) are frequently cited as culprits. In the case of sac growth, most physicians propose additional treatment to prevent the aneurysm from rupturing or to prevent aortic dilatation near the proximal or distal sealing zones, giving rise to possible migration and/or type I endoleaks. When an endoleak is associated with growth, a secondary endovascular procedure or conversion to open repair is usually performed. When no endoleak is found, the solution is more challenging, as the cause of continued aneurysm expansion is frequently unclear.

Previously, laparoscopic fenestration of the aneurysm sac was suggested as treatment for patients with an enlarging aneurysm sac after EVAR, with clipping of aortic sac collaterals.[5] Although the early results were promising, long-term durability of this treatment remains unknown. The aim of the current study was to evaluate the long-term effects of this treatment on sac behaviour, to provide guidance in future decision making.

Methods

Patient selection

From June 1999 to October 2005, a total of 143 AAA patients underwent an EVAR procedure in our hospital. During follow-up, sac growth was observed in 34 patients (23.8%). Type II endoleaks were detected in 21 cases (14.7%). These were either observed or treated with percutaneous interventions, such as coil-embolisation, glue injections and endoscopic clipping of lumbar arteries, depending on sac behaviour. In case of a growing aneurysm sac where no endoleak was detected or when an endovascular approach of type II endoleak was technically unsuccessful or failed to arrest growth, an alternative approach was proposed. Laparoscopic fenestration of the aneurysm sac was then performed, which was preceded by clipping of patent inferior mesenteric artery (AMI) and lumbar arteries. To evaluate the effect of fenestration on sac behaviour, all patients who underwent this procedure were included. The sole exclusion criterion for this study was the lack of a minimum two post-fenestration imaging studies, as that would make observations on sac behaviour impossible. The study was conducted in agreement with the Institutional Medical Ethics Committee guidelines.

Fenestration procedure

The technical details of this intervention were described previously.[5] In summary, all visible lumbar arteries were clipped endoscopically through a retroperitoneal approach, and a patent inferior mesenteric artery (AMI) was clipped laparoscopically. Cleared from all patent side branches, the aneurysm was then fenestrated. During this phase of the operation, the operators could check for residual back-bleeding and suture any remaining type II endoleaks. Also, the sac contents were removed at this time and an omentum slip was inserted whenever technically possible in the sac to prevent immediate closure of the fenestration, reduce exposure of the bare endograft to the small intestines and possibly facilitate resorption of hygroma in the early stages after fenestration.

In one case, the procedure was converted to open suturing of all patent side branches and fenestration of the sac. The primary operator during all procedures was the same, experienced vascular surgeon (J.H.), who was assisted by an experienced laparoscopic surgeon.

Efficacy of fenestration

At the time of these procedures, sac growth was a phenomenon that was aggressively treated. Therefore, the preferential outcome of this treatment at the time was to achieve sac stability or shrinkage. Primary end point of the current study is therefore persistent or recurrent sac growth, which is considered failure of treatment. Aneurysm-related death and additional vascular interventions were recorded as secondary end points. Information on survival and the cause of death was retrieved from hospital records.

Analysis of sac behaviour

Measurement of the aneurysm sac was performed on computed tomography angiography (CTA) images. The first CTA, within 48 h after the fenestration, was considered the baseline for future follow-up. CTAs were then performed approximately every 6–12 months, according to institutional protocol. All hospital records were reviewed for additional interventions and rationale behind treatment decisions. Sac behaviour was scored by two complementary methods.[6,7] First, the single largest diameter of the aneurysm sac was measured. Second, the total sac volume was quantified on each CTA and plotted in time-related curves, regarding the first measurement after fenestration as baseline. All measurements were performed on a workstation with dedicated software (3Mensio Vascular v4.2; 3Mensio Medical Imaging B.V., Bilthoven, The Netherlands) and using centre-lumen line (CCL) reconstruction. Volume measurements were obtained according to a standardised and previously validated protocol.[8] Sac growth was defined as >5% increase in volume compared to baseline or in a 12-month interval. All data was subsequently analysed using the Statistical Package for the Social Sciences, version 17.0 (SPSS Inc., Chicago, IL, USA).

Results

Study population

In the presented time window, a total of nine patients with a growing aneurysm after EVAR underwent aneurysm sac fenestration. One patient died of non-Hodgkin lymphoma 3 months after the procedure, having received only one CTA after the procedure, and was therefore excluded from the current study. From the remaining eight patients (seven men), one patient suffered from a common iliac artery aneurysm rather than an AAA, but was similarly treated by EVAR and later fenestration for continued growth. In one patient, the endoscopic procedure was converted to an open fenestration procedure, as described.[5] At the time of fenestration, the eight patients had a median age of 65.2 (range 55.1–74.3) years. Patient baseline characteristics are detailed in Table 1. There was no perioperative mortality.

Table 1
Descriptive statistics for the study population.

Baseline characteristics	All patients ($n = 8$)
Age in years, median (range)	65.2 (55.1–74.3)
Female gender, *n* (%)	1 (12.5)
Ischaemic heart disease, *n* (%)	2 (25)
Diabetes mellitus, *n* (%)	1 (12.5)
History of stroke, *n* (%)	0
Congestive heart failure, *n* (%)	1 (12.5)
Renal dysfunction, *n* (%)	1 (12.5)
Cardiac arrhythmias, *n* (%)	1 (12.5)
Hypertension, *n* (%)	4 (50)
History of smoking, *n* (%)	3 (37.5)
COPD, *n* (%)	0

COPD = chronic obstructive pulmonary disease.

Procedural details

Four patients were treated with an Excluder AAA Endoprosthesis (W.L. Gore and associates, Flagstaff, AZ, USA), one of which was the low-permeability design introduced in 2004 (Table 2). The remaining implanted grafts were three Zenith AAA Endovascular Grafts (Cook Medical, Bloomington, IN, USA) and one Ancure Graft (Guidant, Menlo Park, CA, USA). Fenestration took place at a median of 1.7 (range 0.5–5.8) years after EVAR. Pre-fenestration sac diameters measured on CTA had a median of 73.2 mm (range 56.5–91.0 mm). The indication for fenestration was persistent or recurrent sac growth for all cases. In 50%, a type II EL could be detected as the possible culprit (Table 2). Attempts to treat these first with glue injections and coil embolisations had been unsuccessful. Upon reviewing the imaging studies in preparation of the procedures, no intense inflammatory component was observed nor was this noticed during the operation. During the procedures, the operators concurred in having achieved proper exposure and the ability to clip all side branches. As confirmation, in only one case residual back-bleeding was observed upon opening the aneurysm sac, which was sutured from within. An omentum slip to leave in the fenestration was available in five out of eight patients.

Aneurysm sac behaviour

Follow-up had a median length of 6.6 (range 0.6–8.6) years. During this time, only three patients experienced durable aneurysm sac shrinkage ($n = 2$) or stability ($n = 1$) and were considered a success. In these three cases where sac growth was successfully arrested, two cases suffered progression of disease leading to dilatation of a common iliac artery (Case #2 and #5). Although this prompted the endovascular extension of one of the distal sealing zones (Table 3), this was not regarded as failure of fenestration.

The remaining five cases suffered persistent sac growth ($n = 2$) or recurrent growth after initial shrinkage ($n = 3$), all regarded as failure of fenestration. The two cases with persistent sac growth comprised one patient with a persistent type II endoleak despite clipping and fenestration, who was converted after 6 months (Case #4), and another patient without detectable endoleaks but an original design Excluder *in situ* (Case #7). This patient was presumed to suffer from endotension, but refused additional treatment until over 5 years after fenestration, when relining of the endograft finally arrested sac growth.

The three cases with recurrent sac growth included one patient that showed shrinkage during the first 7 years, but on the latest CTA suddenly had growth of the aneurysm sac (Case #3) suggesting re-pressurisation, and one patient with a persistent type II endoleak who showed shrinkage at first but recurrent growth within 15 months, spurring conversion (Case #8). In the final case, primary indication for EVAR was a combination of a large iliac aneurysm and a small AAA (Case #6). Sac shrinkage was observed in the first two years after fenestration, but eventually volume and diameter increased again until, finally, contrast was observed in the iliac aneurysm sac, resulting in an extension of the distal dealing zone.

No technical aspects of the procedures or observation made during surgery could be identified as playing a part in the success rate of fenestrations. As mentioned earlier, no (untreated) back-bleeding was observed during the fenestration that could eventually predispose a patient to a residual or recurrent type II endoleak. Furthermore, the impossibility to mobilise an omentum slip for insertion in the fenestration was no predictor for outcome (arresting growth in two, conversion in one).

In summary, six patients underwent additional interventions after fenestration. Two patients were converted to open repair, both suffering from persisting type II endoleaks and early sac (re-) growth. One patient was relined for persistent sac growth, in the presence of an original design Excluder endoprosthesis. In addition, three patients underwent implantation of iliac extensions, one of which suffered from recurrent iliac sac growth and the other two from common iliac artery dilatation due to progression of disease. All secondary interventions after fenestration were successful at arresting further sac growth during the remaining duration of follow-up.

Table 2
Details on EVAR follow-up prior to fenestration.

Case	Implanted graft type	Time since EVAR (years)	Sac diameter (mm)	Detected endoleak
1	Excluder OD	1.6	74.7	None
2	Excluder OD	0.7	68.0	Type II
3	Zenith	1.7	83.5	None
4	Zenith	2.6	69.9	Type II
5	Ancure	5.8	71.6	Type II
6	Zenith	2.4	56.5	None
7	Excluder OD	1.6	84.3	None
8	Excluder LP	0.5	91.0	Type II

EVAR = endovascular aneurysm repair, OD = original design, LP = low-permeability design.

Table 3
Details on fenestration follow-up and outcomes.

Case	Baseline volume	Mid-term volume	Latest volume	Follow-up (years)	Sac growth	Status endoleak	Additional intervention
1	152	81	81	8.6	No	n/a	None
2	97	97	96	8.6	No	Treated	Iliac extension
3	263	244	289	8.0	Yes	n/a	None
4	239	239	263	0.6	Yes	Persistent	Conversion
5	239	164	151	6.3	No	Persistent	Iliac extension
6	47	37	43	7.0	Yes	n/a	Iliac extension
7	188	387	254	6.0	Yes	n/a	Relining
8	432	381	431	1.3	Yes	Persistent	Conversion

Volumes are abdominal aneurysm sac volumes in ml. Sac growth was defined as >5% volume change compared to baseline or in a 12-month interval.

Discussion

EVAR has become the preferred method of treatment in many AAA patients, especially when the aortic anatomy is favourable. Despite the early survival advantage, EVAR is associated with greater aneurysm-related complications and therefore most agree on the need for life-long follow-up with imaging studies to evaluate migration, stent integrity, endoleaks and aneurysm size.[9–11] Post-implantation growth has received particular attention because it is observed with relative frequency and suggests continued pressurisation of the aneurysm sac, and therefore failure of treatment (despite relative rarity in clinical consequences).[12] After EVAR, the majority of patients have either a gradual decline or stabilisation of their aneurysm dimensions over the years.[13] When growth occurs, however, a plausible explanation should be sought and treatment promptly offered. While it may be the accepted standard of care that patients with type I or III endoleaks require rapid intervention, opinions vary over the implications of type II endoleaks, especially in cases where the diameter of the aneurysm stabilises or only grows slowly.[14,15] Within the last decade, studies reported that selective surveillance of a type II endoleak is a safe course.[16] Controversially, Jones et al. reported that persistent type II endoleak increases the risk for rupture and the need for conversion,[17] while data from the EUROSTAR registry suggested that it actually seems to protect the patient against rupture.[14] When the current patients were diagnosed with a growing aneurysm after EVAR, endotension and type II endoleaks were aggressively treated. In

2002, Veith et al. reported on a summit with 27 interested leaders who reached a consensus that growing aneurysms without detection of endoleaks should be treated surgically or by repeated EVAR procedure.[17] Concerning type II endoleaks, Steinmetz et al. reported that if no sac growth is seen, no additional intervention is necessary.[15] However, general opinion among the leaders previously mentioned was that persistent type II endoleaks required treatment, either with coil/glue embolisation[18] or laparoscopic clipping.[19]

With that historical backdrop, a series of nine patients with growing aneurysms without detectable endoleaks or with persistent type II endoleaks were treated by laparoscopic clipping of side branches and aneurysm sac fenestration. Although the short-term results were promising,[5] the current study is the first to show that long-term results are sub-optimal in a large proportion of patients, raising doubt over the applicability of this previously described technique. The ultimate goal of the clipping and fenestration procedure was to halt sac growth. Durable aneurysm, sac stability was only achieved in three patients, two of who underwent additional procedures for progression of the disease in the common iliac arteries. Out of the other five cases, two were converted to open repair, one was relined, one was extended at the distal sealing zone and one was diagnosed with recurrent sac growth on the latest scan. In general, the two-step procedure was not particularly successful in achieving its goal of durable sac stability.

The first step in the procedure was to clip all lumbars and other possible side branches to treat or prevent type II endoleaks. Noticeably, out of four cases presenting with a type II endoleak prior to fenestration in our study, the endoleak persisted in three, despite the subjectively good view on lumbar arteries during this procedure. The only successful elimination of a type II endoleak was achieved in the one patient that was converted, and therefore clipping of collaterals and sac fenestration was performed as an open procedure, reducing the endoscopic success rate of clipping to nil. Interestingly, an open aneurysm sac with a subsequently demonstrable endoleak had no clinical consequences in our series. Although minimally invasive clipping of lumbar side branches has been frequently performed, right-sided lumbar arteries are technically difficult to expose and clip.[20] In some cases, endoscopic clipping may be unsuccessful, resulting in residual type II endoleaks.[21] This could have contributed to the failure of arresting type II endoleaks durably, in the current study. An alternative approach is primary fenestration and subsequent sewing of back-bleeding lumbars from within the sac.[22,23]

In the current study, fenestration was performed after clipping of the side branches, allowing for visual control by scanning for residual back-bleeding, as previously described by Dion et al. in 2001.[24] Only in one case, back-bleeding was still observed, and this was sutured from within the sac. Although sac contents were thoroughly evacuated after fenestration, the residual type II endoleaks could have been masked by mural thrombus or other debris, missed at the time of surgery. This illustrates that laparoscopic fenestration is a demanding procedure and, even in the hands of experienced vascular and laparoscopic surgeons, can lead to underexposure of the inside of the sac, and thus incomplete removal of thrombus and assessment of back-bleeding side branches.

The most logical indication for fenestration would therefore be endotension as a result of increased graft porosity. Transudate of fluid through the graft fabric is well described, particularly after implantation of the original Excluder endograft (W.L. Gore and associates, Flagstaff, AZ, USA).[18] Releasing the hygroma would theoretically result in arrested growth and prolonged success. This idea has also been defended by others, both with open[19] or with percutaneous sac fenestration. In our series, two patients implanted with the Original Design Excluder continued to exhibit growth without detectable endoleaks prior to fenestration. After fenestration, sac stability was observed in one, but sac growth persisted in the other, who later underwent successful relining with a low-permeability graft. This sac growth could be explained by healing of the fenestration, resulting in the recurrence of hygroma, allowing re-pressurisation. Goodney et al. and Kougias et al. have published on their experience with relining, with similar good results at short term.[20,21] This alternative solution, although promising, still lacks long-term data, but is generally accepted as first-line treatment in case of a growing sac with an original Excluder endograft *in situ*, or when graft integrity is thought compromised at a specific location.[22,23] Importantly, standard CTA is not the most sensitive technique for type II endoleak visualisation, and definite diagnosis of endotension is often only possible after opening the aneurysm sac and visualising no bleeding aortic collaterals.[25] Therefore, it is theoretical to reserve this technique for endotension cases.

The current report is limited by its observational design and by the small number of patients. Also, the indication for treatment was individualised and no strict criteria were observed, with potential selection bias. For the purpose of demonstrating the safety and efficacy of the technique, however, these limitations – albeit important – can be accepted to prevent others to subject their patients to this ineffective treatment as well.

In conclusion, the results after fenestration are quite variable and, more importantly, largely unpredictable. Sac growth was observed after fenestration in five out of eight cases, spurring additional interventions in the majority. Therefore, we cannot recommend fenestration as primary treatment for sac growth. Other techniques may hold more promise when minimally invasive interventions fail, risk of rupture is considered high and the patient is too frail for aortic cross-clamping and endograft explantation.

Conflict of Interest/Funding

None.

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