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Conservative Management of Type 2 Endoleaks is not Associated with Increased Risk of Aneurysm Rupture

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Abstract *Objective:* Endovascular repair (EVAR) of abdominal aortic aneurysms (AAAs) has led to a reduction in the perioperative mortality when compared with open repair. However, re-intervention for complications, such as endoleak, may be required in up to 20% of the cases. Controversy exists over the management of Type 2 endoleaks. This study examined the outcomes of patients with Type 2 endoleaks treated conservatively to inform the ongoing management debate.
Methods: All patients with a confirmed Type 2 endoleak after EVAR for an infrarenal AAA were included in the study. Data regarding device details, endoleak and time point, aneurysm sac growth, intervention and outcome were collected retrospectively from case notes and the vascular research database.
Results: Forty-one Type 2 endoleaks were seen in 369 EVARs performed for infrarenal AAA between March 1994 and June 2006. Twenty-five were isolated Type 2 endoleaks and 16 occurred in conjunction with other endoleaks. Of the 25 isolated Type 2 endoleaks, 18 (72%) patients demonstrated no increase in sac size, six (24%) patients showed an enlargement of the sac and one patient was lost to follow-up. Only one patient underwent intervention for an isolated Type 2 endoleak. After a mean follow-up period of 4 years, approximately half of the patients (48%) remain under observation (with an enlarging or stable sac), whilst the others (48%) have spontaneously sealed. Only five patients under surveillance (20%) have an enlarging sac. There were no ruptured aneurysms or aneurysm-related deaths and no patients required conversion to open repair.
Conclusions: In this study, a policy of regular surveillance for Type 2 endoleaks was not associated with any adverse events. We therefore advocate the conservative approach for Type 2 endoleaks.

Endovascular abdominal aortic aneurysm repair (EVAR) was introduced by Parodi in 1991,[1] and since then, the technique has gained popularity due to the significant advantages over open repair. Several large multicentre studies have reported a reduction in morbidity and mortality associated with EVAR.[2–4]

The UK EVAR1 trial randomised 1082 patients to either open or endovascular repair and showed a significant reduction in mortality at 30 days from endovascular repair

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(1.7% vs. 4.7%), with this benefit sustained at 4 years (4% vs. 7%).[2] However, this trial also highlighted a higher complication rate after EVAR due to stent fracture or migration, kinking or graft limb thrombosis and endoleak.[2,5] The re-intervention rate in EVAR1 was 20% at 4 years and approximately 50% of these were for endoleak. The annual cumulative rupture rate after EVAR has been shown to be 1%.[6]

One of the most common complications of EVAR is endoleak, defined as persistent blood flow within the aneurysm sac. Endoleak has been classified into five types depending on the source of origin of the endoleak. Type 1 endoleaks are due to proximal or distal attachment site leaks, Type 2 endoleaks are due to retrograde flow from aortic branches and Type 3 endoleaks are due to component separation or fabric tears. Type 4 endoleaks can only occur within 30 days of stent-graft insertion and are due to an increase in graft wall (fabric) porosity. A further category, known as endotension, has been identified and is described as an increased intra-sac pressure after EVAR without a visualised endoleak on delayed contrast computed tomography (CT) scans.[7] In a recent systematic review, the occurrence of Type 1 and Type 2 endoleaks at 12 months was 3.8% and 14%, respectively.[8]

Types 1 and 3 endoleaks are often grouped together as high-pressure leaks, which require urgent attention to prevent rupture.[6] Type 2 endoleaks, arising from aortic side branches, are the most common type and are regarded as low-pressure leaks. The natural history of Type 2 endoleaks is unclear, with some tending to resolve spontaneously, others persisting without causing clinical symptoms and a small proportion causing a significant increase in sac size.[9–12]

The rupture rate from Type 2 endoleaks appears to be low and there are only 14 reported cases of ruptured AAA associated with a Type 2 endoleak in the world literature in nine studies.[9,11,13–20] Data from the EUROSTAR registry on 2463 patients from 87 European centres suggest an incidence of rupture after Type 2 endoleak of 0.52% (one from 191 patients).[16] These data also suggested that the rupture rates were no different in patients with Type 2 endoleak when compared with patients with no endoleak (0.25%, 5 out of 1975 patients, $P = 0.54$).

There is an ongoing debate as to the management of Type 2 endoleaks, with some authors advocating a conservative approach, some intervening in all cases and others proposing selective intervention for a significant increase in sac size (defined as an 8-mm increase in the EUROSTAR registry).

In Leicester, we have employed a policy of conservative management for Type 2 endoleaks. The emergence of more data about the natural history of Type 2 endoleaks has reinforced our conservative policy and allowed us to implement more stringent criteria. Since 2001, treatment for Type 2 endoleaks has only been performed if there was a documented increase in sac size of greater than 5 mm over a 6-month period, or an overall increase of more than 10 mm from the preoperative measurements.

The aim of this study was to examine the outcome of patients with Type 2 endoleaks following EVAR to guide future management of this complication.

Methods

A retrospective–prospective study of 369 consecutive patients who underwent infrarenal EVAR repair at the Leicester Royal Infirmary between 18 March 1994 and 28 June 2006 was performed. Following EVAR, patients were followed up at outpatients with clinical examination and duplex ultrasound scan (DUSS) at 1, 3 and 6 months postoperatively, and at 6-monthly intervals thereafter.

At the time of DUSS, data were recorded on sac size, the presence or absence of endoleak and other complications such as graft limb occlusion. Follow-up data were recorded on a research database and imaging records were retained for future reference.

DUSS was performed by B-mode imaging of the aneurysm sac in transverse and longitudinal planes to identify the graft and to examine the sac contents (homogeneous or heterogeneous) for any regions that look suspicious for endoleak or show abnormality. The maximum sac diameter was measured in both antero-posterior and lateral planes (side-to-side diameter from the coronal position) so that any changes in sac size could be determined. Colour flow imaging was the principal method used to examine the sac for evidence of endoleak. The colour flow scanner controls were also optimised to detect low-velocity flow from very small endoleaks. Spectral Doppler recordings were also taken from any identified endoleak to examine flow characteristics (pendulum flow suggesting a blind-ending endoleak, or directional flow that might suggest both inflow and outflow). Flow within the graft and iliac arteries was also examined to detect any flow abnormalities, which may suggest any other graft-related complications such as in-graft stenosis or kinking. All DUSSs were performed using a Philips Medical Systems HDI 5000 duplex ultrasound scanner, Bothell WA, USA and a C5-2 MHz broadband curved-array transducer (standard colour DUSS with B-mode imaging, colour flow imaging and spectral Doppler).

In our early experience, clinical decisions regarding patients in follow-up after EVAR were carried out through direct discussion between surgeons and radiologists. More recently, a multidisciplinary team was created (vascular surgeons, vascular technologists, vascular nurse specialists and vascular radiologists). Patients found to have an endoleak on DUSS or where duplex images were suboptimal and not able to exclude endoleak were discussed in the above and clinical management plans formulated.

Patients underwent CT angiography if there was an evidence of a Type 1 endoleak, an indeterminate endoleak or a sac growth of greater than 5 mm in any 6-month period or to a size 10 mm greater than the preoperative size.

Type 2 endoleaks were treated by further endovascular intervention via a transfemoral, translumbar or laparoscopic route.

Results

There were 369 elective infrarenal EVARs performed between 18 March 1994 and 28 June 2006. Seventy-three (20%) were locally developed aortouniliac (AUI) design; the remaining 296 were commercial devices — 86 (out of 369; 23%) were Medtronic Talent grafts, 54 (15%) were Gore

Excluder, 137 (37%) were Cook Zenith and 19 (5%) were other commercial devices.

Seventy-seven endoleaks (of varying types) developed in 68 patients (18%; 68 out of 369) (Table 1). There were 41 Type 2 endoleaks in 39 patients but 16 were associated with another type of leak (14 combined with a Type 1 endoleak and two combined with a Type 3). Therefore, there were a total of 25 Type 2 endoleaks in 25 patients, which form the basis of further analysis (Table 2). Twenty-two patients were male (88%) and three were female (12%). The mean age at operation was 71 years with a mean sac size of 6.6 cm. The timing of diagnosis of the Type 2 endoleak was evenly distributed from 1 month to 12 months and over, although more endoleaks were seen on discharge scans than at any other time point (Table 3).

Out of 24 Type 1 endoleaks, 17 occurred within the first month (71%), compared with seven (29%) detected after 1 month. Only a small number of Type 3 endoleaks were seen in this study ($n = 4$) and all occurred within the first month after the surgery.

Intervention purely to treat a Type 2 endoleak was performed in one patient (one out of 25; 4%). This patient underwent EVAR in 1999 using a Medtronic Talent device. Pre-discharge scans suggested an endoleak from the left limb of the graft, which was corrected by an extension device. Following this, the sac expanded slowly (8.4–9.1 cm in 29 months) but with no evidence of an endoleak. Three years postoperatively, a Type 2 endoleak was identified with sac expansion (10.7 cm). Intervention was attempted using coil embolisation, thrombin injection into the aortic sac and laparoscopic clipping of the inferior mesenteric artery, but all failed. The aneurysm sac continues to grow and the last measurement was recorded at 13 cm (CT scan). The patient has remained completely asymptomatic throughout this period and is unfit for any other form of intervention.

Six (24%) of the 25 Type 2 endoleaks were associated with a documented increase in sac size; however, only one of these (discussed above) met the criteria for consideration of intervention. Five patients continue to remain under observation for their Type 2 endoleak and one endoleak sealed spontaneously. Although it is difficult to estimate due to the differing length of the follow-up period, the average growth rate was 0.23 mm per month. Of the remaining 19 patients, 18 (72%) have a stable sac with no increase in size and one was lost to follow-up (Table 4). After a mean follow-up of 50.4 months, seven out of 18 endoleaks with a stable sac size remain under observation and 11 have sealed spontaneously (61%).

Table 1 Incidence of endoleak ($n = 77$ in 68 patients).

Type of endoleak	Number of endoleaks (%)
Type 1	24 (31)
Type 2	41 (53)
Type 3	4 (5)
Type 4	1 (1)
Unspecified	7 (9)
Total	77

Table 2 Type 2 endoleaks.

Device type	Number of Type 2 endoleaks (%)
AUI	1 (4)
Medtronic	5 (20)
Gore	10 (40)
Cook	9 (33)
Total	25

Where AUI = aortouniliac device.

To verify the above data, all the patients who had been re-admitted after EVAR were identified through the hospital coding computer system. The notes were then examined to identify the reason for re-admission. Although only eight notes were unobtainable, this re-confirmed that there were no deaths from a ruptured AAA after a Type 2 endoleak.

In summary, only one patient had an increase in sac size that required intervention. Twelve patients remain under observation for their endoleak but only five showed an enlarging sac. Eleven Type 2 endoleaks have sealed spontaneously. There were no conversions to open repair and no ruptured aneurysms among the patients with a Type 2 endoleak.

Two deaths from unknown causes were seen in patients with Type 2 endoleaks. No deaths were due to aneurysm-related complications or rupture. The deaths occurred at 22 months and 33 months postoperatively. Our total survival rate was not assessed during this study but has been previously published. Five-year follow-up data on 58 consecutive patients who underwent elective EVAR between 1 July 1994 and 3 October 2000 revealed 13 deaths, none of which was perioperative death (defined as within 30 days of surgery).[21]

Discussion

The management of Type 2 endoleaks has evolved with time. Initially, many of these endoleaks were treated by radiological or surgical intervention because of the fear of rupture. Recently, a more conservative approach has been adopted because many of them seem to be relatively benign. We have analysed our entire EVAR experience from 1994 to 2006, and during this time, 25 isolated Type 2 endoleaks have been seen. We have intervened only once despite 24% of these endoleaks having an associated increase in sac size. We have not experienced any cases of

Table 3 Type 2 endoleaks according to time point postoperatively.

	Discharge	1 month	3 months	6 months	12 months	>12 months
AUI	0	0	0	0	0	1
Medtronic	2	1	0	1	0	1
Gore	2	3	2	1	1	1
Cook	3	1	1	3	1	0
Total	7	5	3	5	2	3

Where AUI = aortouniliac device.

Table 4 Increase in sac size in association with Type 2 endoleak.

	Increase in sac size requiring intervention	Increase in sac size not requiring intervention	No increase in sac size	Unknown
AUI	0	1	0	0
Medtronic	1	0	4	1
Gore	0	3	6	0
Cook	0	1	8	0
Other	0	0	0	0
Total	1	5	18	1

Where AUI = aortouniliac device.

rupture associated with Type 2 endoleak and we have not performed any open interventions or conversions. Follow-up data over a mean period of 4 years have shown that 48% of these Type 2 endoleaks have sealed spontaneously (12 out of 25), 48% remain under observation (12 out of 25) and one has been lost to follow-up. Currently, only five patients in our surveillance programme have a Type 2 endoleak and an enlarging sac.

Although the natural progression of Type 2 endoleaks is not fully understood, the main concern is rupture. Data from the EUROSTAR Registry show that the annual cumulative rupture rate from EVAR is approximately 1%.[6] However, EUROSTAR also reported (in 2463 patients) that there was no significant difference in rupture rates after EVAR in patients with and without a Type 2 endoleak.[16] Despite this, many centres continue to intervene for Type 2 endoleaks, particularly if the sac is expanding.

A literature search conducted by the authors of this article found only 14 reported cases of ruptured AAA from a Type 2 endoleak in the world literature across nine studies. This represented a cohort size of 2627 Type 2 endoleaks (14 out of 2627; 0.53%), with only six of these appearing to have had an increase in sac size.[9,13,15,17–19] However, a recent study has found 23 ruptures after Type 2 endoleak,[22] although no direct references are given and some of the data may be from duplicate or updated series. Other reports from EUROSTAR found that conversion to open repair or post-EVAR rupture was not associated with Type 2 endoleaks.[12] Similarly, Gelfand showed no association between Type 2 endoleak and rupture and also observed that over 50% of Type 2 endoleaks managed conservatively for a year resolved spontaneously.[23]

The ability to detect endoleaks depends on the sensitivity and specificity of the imaging modality. Currently, CT scanning is the gold standard, but there are concerns about the radiation load, reactions to contrast medium and the risk of worsening renal failure.[24] Although duplex scanning is not as sensitive as CT, a recent study has shown that duplex does detect high-pressure Types 1 and 3 endoleaks but may miss some Type 2 endoleaks. If Type 2 endoleaks are benign, even if the sac is expanding, then duplex may be a more suitable alternative to CT for EVAR follow-up – with reduced risk and lower costs.[25] Whilst DUSS may not be as sensitive at detecting endoleaks when compared with CT scan,[25] the benefits of detecting clinically non-significant endoleaks must be weighed against the added burden on services and subsequent delays in imaging, as well as the extra radiation load and potential contrast-related issues suffered by the patient. In this study, any indeterminate DUSS scans were re-imaged using CT.

Bargellini *et al*. compared the use of DUSS to CT angiogram (CTA) in the post-EVAR assessment of AAA size and detection and monitoring of endoleaks. There was a high level of agreement between the findings observed from DUSS and CTA (k 0.96) in measuring maximum AAA diameter. DUSS detected 76.4% of all endoleaks and of the remaining endoleaks, only three (4.3%) were clinically significant with respect to AAA enlargement.[26] In a similar study, specificity of DUSS when compared with that of CTA for endoleak detection was 67%, mainly due to the large number of false positives from DUSS, whilst sensitivity for DUSS was 86%. All clinically significant endoleaks were identified by both imaging modalities. Despite this, DUSS only had a positive predictive value of 45%, but encouragingly had a negative predictive value of 94%.[27] Another study has quoted the sensitivity and specificity of DUSS as 33.3% and 92.8%, respectively. However, the positive and negative predictive values in this study were similar at 71% and 72%, respectively.[28] By contrast, a systematic review published in 2005 on the same topic concluded that DUSS is not accurate enough in detecting endoleaks. Although the study comprised 711 patients and 1355 paired scan results, these were only from two centres. Data from two studies included in the article were from unpublished studies. The pooled sensitivity of DUSS compared with CT was 69%, with the specificity at 91%.[29]

Whilst some have criticised the ability of DUSS in differentiating between Type 1 and Type 2 endoleaks, it must also be acknowledged that this can also be a problem with other modalities. Simple Type 2 endoleaks from branch vessels are normally easy to identify, especially if they are some distance away from the proximal or distal attachment sites. It can be more difficult if the endoleak is towards the bottom or top ends of the sac. Migration is more difficult to assess using DUSS and is best used when there has been gross movement, such as the neck 'falling' into the sac. For this reason, DUSS is not routinely used to assess migration, and CT scan is the preferred imaging tool.

Several techniques exist for the treatment of Type 2 endoleaks, including embolisation via a transarterial or translumbar route, laparoscopic ligation of aortic sac side branches and vessels and thrombin injection. In a small series of five patients, Steinmetz *et al*.[10] reviewed both the translumbar ($n = 4$) and transarterial ($n = 4$) approaches and reported no recurrence or aneurysm sac growth in either group after the procedure. A larger study[30]

intervened in 33 Type 2 endoleaks (20 through transarterial inferior mesenteric artery embolisation and 13 through translumbar embolisation) and reported failure in 80% undergoing transarterial embolisation (mean follow-up 396 days) compared with an 8% failure rate with the translumbar technique (median follow-up 254 days). Similarly, Stavropoulos reported failure of translumbar embolisation in three out of nine patients (with 11 Type 2 endoleaks).[31]

There are very limited data on the effectiveness of laparoscopic ligation of aortic sac side branches. In total, three studies describe laparoscopic intervention in five patients,[32–34] and in two patients further laparoscopic intervention was required to treat recurrence of the endoleak.[33,34]

Prophylactic intra-operative embolisation of the inferior mesenteric artery together with intra-sac thrombin injection was described by Muthu *et al*.[35] However, there was no statistically significant difference in the subsequent Type 2 endoleak rate between the intervention group and a historical control group.

In conclusion, Type 2 endoleaks appear benign even in the presence of an increasing sac size. The current treatments have a high risk of failure and may not alter overall outcome.

Conflict of Interest

None.

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