



chapter 6

Endoscopic type II endoleak repair following
endovascular aortic aneurysm repair:
acute results and follow-up experience

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ABSTRACT

Objective: To evaluate immediate and long-term results of endoscopic type II endoleak repair (EER) following endovascular abdominal aortic aneurysm repair.

Methods: A retrospective review of electronic and paper medical records of patients admitted or referred to our institution for EER.

Results: Between July 1999 and October 2007, eight consecutive patients underwent EER. Mean operative time was 190 (104–355) min. One patient died per-operatively, due to profuse venous bleeding. One procedure was redone due to a missed pair of lumbar arteries. Mean hospital stay was five (2–10) days. During mean follow-up, 50 (29–91) months, one patient required additional coil embolization for a persistent type II endoleak. Four patients were diagnosed with a type I and one with a type III endoleak; three of these patients required an additional procedure.

Conclusion: in this small series EER proved not to be beneficial.

Introduction

Since the first report by Parodi et al¹ in 1991, endovascular aortic aneurysm repair (EVAR) has become a widely accepted treatment for abdominal aortic aneurysms (AAA). Compared to conventional open repair, EVAR has the advantage of a less extensive operative exposure, circumventing transcavitary incision.

However, EVAR has complications not seen with open abdominal aortic aneurysm repair. The most frequent mechanism of failure after EVAR is the occurrence of an endoleak, i.e., persistent blood flow outside the endograft and within the aneurysm sac. The most prevalent is a type II endoleak that is a result of retrograde filling of the aneurysm sac through small aortic branches, such as the lumbar arteries and the inferior mesenteric artery (IMA). Reported incidence during follow-up roughly ranges from 10-25%.²⁻⁸ Persistent type II endoleak is associated with an increased incidence of adverse outcomes, including aneurysm sac growth, and rupture.^{2,6,9}

The aim of this study was to evaluate retroperitoneal endoscopic endoleak repair (EER) as a minimal invasive surgical treatment option for persistent type II endoleak after EVAR as well as to describe the mid and long-term results of this procedure.

Methods

We performed a retrospective review of electronic and paper medical records of patients admitted to our hospital with a diagnosis of a type II endoleak after EVAR who were treated with EER.

Operative technique

All procedures were conducted by the same surgeons (an experienced vascular surgeon (W.W.) and an experienced endoscopic surgeon (M.A.C.)). The operative technique has been described elsewhere.¹⁰ In short, the patients were placed on a "bean bag" in a 30 to 60-degree right lateral decubitus position. The tip of the twelfth rib was palpated, and an incision of 2 cm was made anteriorly. Blunt dissection was performed with the help of a clamp and index finger to create a small retroperitoneal space in two directions. A dissecting balloon (Origin Medsystems, Menlo Park, CA, USA) was introduced and insufflated sequentially in the two directions to enlarge the retroperitoneal space. The balloon was replaced

with a 10-mm trocar, and the retroperitoneum was insufflated with carbon dioxide up to a pressure of 14 mm Hg. Two accessory trocars with a 10-mm diameter each were placed, the first close to the iliac crest in the posterior axillary line, and the second near the costal margin in the anterior axillary line. A 0-degree videoendoscope was used. Branch arteries, were either clipped using medium-sized titanium clips (Endoscopic Rotating Multiple Clip Applier, Ethicon Endo-Surgery, Cincinnati, Ohio, USA) or ligation was performed using the LigaSure system (Valleylab, Tyco Healthcare, Boulder, CO, USA). First, the left-sided lumbar arteries were clipped and divided, in this manner the aneurysm could be tilted slightly to expose the right-sided lumbar arteries, which were then clipped.

Mean intra-aneurysmal pressure (IAP) was measured by means of insertion of an endoscopic needle attached to a pressure monitor before and after clipping. In one patient intraoperative duplex ultrasound imaging was used for accurate identification of patent branch arteries and control after ligation.

All patients underwent computed tomography-angiography (CTA) on the first post-operative day. Standard follow-up with CTA, magnetic resonance angiography (MRA) and/or duplex ultrasound imaging was carried out at 6 and 12 months and then yearly thereafter.

Results

Between 1999 and 2007, 8 patients with a mean age of 76 years (70-83) were referred to our institution with a persistent type 2 endoleak more than six months following EVAR (table 1). Mean AAA enlargement following EVAR was 8.5 mm (1-23). Six patients underwent one or more preceding coil embolization procedures of which four also underwent percutaneous thrombin injection. One patient was diagnosed with a type 4 endoleak as well, which was treated with placement of an extension graft. Angiography revealed one (n=2), two (n=2), three (n=2) or four (n=1) patent lumbar arteries, whereas the inferior mesenteric artery was patent in four patients. A patent middle sacral artery was found on one occasion. Three patients had developed vague abdominal and back pain, the remaining were asymptomatic.

Table 1: Patient characteristics

Patient	Age (yr)	Sex	ASA scale	AAA diameter at time of EVAR (mm)	AAA Enlargement (mm)	Prior interventions
1	79	M	II	60	12	-
2	72	M	II	55	1	CE + PTI
3	80	F	III	73	7	CE + PTI
4	83	M	II	55	23	CE + PTI
5	73	M	III	80	1	CE
6	77	M	II	56	10	CE
7	70	M	III	56	4	CE
8	74	M	III	55	10	-

ASA: American Society of Anaesthesiologists, CE: coil embolization, PTI: percutaneous thrombin injection

30-day results

There was one operative death. Dissection of the aorta was difficult due to peri-aortitis with dense adhesions between the aortic wall and the vena cava. The endoscopic procedure had to be converted due to a significant bleeding at the dorsal aorta. During conversion a tear in the vena cava was found at the level of the aortic bifurcation. No immediate homeostasis could be achieved, for religious reasons, blood transfusion was declined. The patient died on the operating table.

For all successfully completed operations (n=7), mean operative time was 190 minutes (median 169, range 104-355), median blood-loss was 50 ml (range 30-250). Mean IAP decreased from 65.8 (range 30-130) to 22.8 (range 0-35) mmHg. In one patient IAP measurement was unsuccessful; it revealed a straight line, without a pulse wave or other fluctuations. Peroperative duplex was performed, which revealed no flow in the aneurysm-sac. Four (n=2), six (n=3) or eight (n=1) lumbar arteries were either clipped or ligated. The IMA was treated on five occasions. In one patient, a large patent middle sacral artery was clipped, this required a small retroperitoneal incision to facilitate difficult dissection. All patients were removed from mechanical ventilation immediately postoperatively without the need for admission to an intensive care unit. The post-operative course was

uncomplicated in six patients; one patient was diagnosed with a pulmonary embolism 2 days after discharge. Regular diet was resumed one (n=6) or two (n=1) days after the operation. Mean hospital stay was 5 (2-10) days. CTA on the first post-operative day revealed no remaining endoleak in five patients. In one a pair of unclipped patent lumbar arteries was seen just at the aortic bifurcation. Following immediate re-operation via the same approach the remaining pair of lumbar arteries was clipped, and thrombin was injected into the aneurysm. Following CTA confirmed complete thrombosis of the aneurysm sac. In another patient a minor type II endoleak was seen, which at that time was left untreated.

Long-term follow-up

With a mean follow-up of 50 months (29-91), 6 patients have remained free from type II endoleak. In the one patient with the type II endoleak that was left untreated, minimal aneurysm sac enlargement (1 mm) was seen. And while the endoleak remained persistent more than two years after EER coil embolization was performed. Four patients were diagnosed with a type I endoleak at the proximal (n=2) or one of the distal (n=2) sites. One patient was treated with an extension graft at the right iliac artery site, while another received a fenestrated cuff at the proximal site with two covered stents for both renal arteries¹¹. While there was no aneurysm size enlargement in both other patients, they were left untreated. In one patient MRA revealed a type III endoleak at the left branch two years after EER. Subsequently the patient was treated a uni-iliac endograft (Talent, Medtronic), which was deployed within the old prosthesis, followed by a femoral-femoral crossover.

Discussion

The occurrence of type II endoleak after EVAR remains a vexing dilemma. Interventionalists debate their significance, as some have been associated with AAA stability or even shrinkage, while others resolve spontaneously. Traditional believe is that further intervention is required when an increase in aneurysm size is diagnosed, or when type II endoleaks remain persistent more than 6 months during follow-up.^{2,3,5,7,12-14}

Table 2: Results

Patient	OT (min)	HS (days)	Follow-up (months)	Ligated arteries	Complications	Adjunct procedure
1.	130	10	91	6 LA + IMA	type 1 + 2 endoleak	re-operation with ligation of 2 LA
2.	204	2	41	8 LA	type 1 endoleak	-
3.	355*	-	-	-	conversion + peroperative death	-
4.	255	7	41	4 LA + MSA	type 1 endoleak	extension graft for right iliac
5.	190	4	55	6 LA + IMA	-	-
6.	130	4	48	6 LA + IMA	type 3 endoleak	uni-iliac endograft placement prox.
7.	104	3	44	IMA	type 1	fenestrated cuff placement
8.	148	4	29	4 LA+ IMA	type 2 endoleak	coil embolization

OT: operating time, HS: hospital stay, LA: lumbar artery, IMA: inferior mesenteric artery, MSA: middle sacral artery.

* extended operating time due to conversion after major venous bleeding

Currently, type II endoleaks can be treated with a variety of methods: conversion to open procedure, transarterial or translumbar coil embolization of feeding branch vessels, direct thrombin injection of the aneurysm sac or endoscopic clipping of lumbar and/or inferior mesenteric arteries. Conversion is a valid option, however is associated with significant complications.^{2,15-17} Moreover, many patients are high-risk or are reluctant to accept conversion. Embolization and thrombin injection can be performed through a transarterial and translumbar route. The success rate of these techniques varies widely; procedures can be difficult and require advanced catheter skills and knowledge of use of microcatheters.^{9,18-22} Sac-embolization alone

does nothing to eliminate the source of the leak. There is no guarantee that just because the aneurysm sac has achieved thrombosis, it is protected from systemic pressure. Moreover, paraparesis and colon ischemia have been reported after thrombin or glue injection.^{7, 23, 24} The transarterial route bares the risk of creating a type I endoleak through catheter manipulation at the proximal and distal ends of the endograft. In this series six patients underwent prior coil embolization, thrombin injection or both. In the remaining two, multiple patent lumbar arteries as well as a patent IMA were present; therefore percutaneous techniques seemed technically difficult and were not attempted prior to EER.

Over the past decade, endoscopic techniques in vascular surgery have progressed from assisted to totally endoscopic procedures.²⁵⁻²⁸ Despite a steep learning curve, endoscopic vascular surgery may offer the same advantages as endoscopic general surgery in terms of patient recovery, postoperative pain, and length of hospital stay.²⁹ Endoscopic endoleak repair (EER) seems an attractive treatment for type II endoleaks. Wisselink et al.¹⁰ first described the technique, which to date has been reported in case-reports only.³⁰⁻³³

In this case-series, similar to Richardson et al.³⁰ there was a false assumption that adequate exclusion had been achieved in two cases. After ligation of multiple pairs of lumbar arteries, CTA revealed persistent contrast in the aneurysm sac. One patient underwent reoperation the next day. Dissection was relatively simple, and a remaining pair of patent lumbar arteries could be easily identified and clipped. In the other patient the endoleak was considered minor, and was left untreated. But while the endoleak remained persistent, after two years it was resolved with coil embolization. This experience has made it clear that to ensure that all lumbar arteries are addressed, the entire posterior surface of the aneurysm needs to be dissected free, allowing a clear, uninterrupted view of the inferior caval vein in the background. In principle, however, only those lumbar arteries that contribute to the endoleak need to be ligated. In future cases, accurate identification of patent branch arteries might allow less extensive dissection. Concurrent fluoroscopy and angiography on the operating table, as well as intraoperative duplex ultrasonography scan, may be used toward this end.

The role of IAP measurement as a mean to judge completeness of EER remains unclear. In one of our cases continuous pressure registration revealed a straight line, without a pulse wave or other fluctuations, this was probably due to the fact that the needle tip was embedded in thrombus. In two other cases, following injection of 0.5 cc of saline, a pressure curve was obtained with a mean pressure

equal to systemic pressure. Furthermore, the patient in whom two lumbar arteries were missed at the first procedure, intra-operative pressure decreased to 0mmHg. CTA the first post-operative day revealed persistent flow in the aneurysm sac. These findings further indicate the need for safe and simple techniques to measure intra-sac pressure.

Remarkably, during follow-up, four patients were diagnosed with a type I endoleak. It is unclear whether these have been new onset leaks or had been missed on prior CTA. However, with this high incidence of type I endoleaks it is not inconceivable that aneurysm manipulation during EER may have played a role. The aneurysm had to be slightly tilted to expose the right-sided lumbar arteries, which might have caused endograft displacement and loss of seal between the graft and vessel. In one patient we believe the type I endoleak had been present prior to the endoscopic procedure, but had not been recognized as such due to the multiple patent branch arteries. Alternatively, it is possible that "latent" type I endoleaks have been unmasked by occluding the outflow of the aneurysm sac by resolving the type II endoleak.

Conclusion

In conclusion, in this small series, endoscopic type II endoleak repair proves a technically challenging procedure, even when performed by two experienced (laparoscopic) surgeons. With two missed type II endoleaks, it proves that adequate per-operative imaging or intra-aneurysm pressure measurement is vital while patent lumbar arteries are easily missed. There seems a high risk for developing a type I endoleak. Although its etiology remains unclear, it is not inconceivable that it is the result of aneurysm manipulation during EER. Furthermore, serious bleeding complications may arise while working in the vicinity of the vena cava when the aorta is dissected free. From this small series, with one operative death, two missed type II endoleaks and five patients requiring additional procedures, we conclude that EER was not beneficial.

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