## Title

Early and intermediate outcome of emergency endovascular aneurysm repair of ruptured infrarenal aortic aneurysm; a single center experience of 90 consecutive patients.


## Authors

Holst Jan, Resch Tim, Ivancev Krassi, Björses Katarina, Dias Nuno, Lindblad Bengt, Mathiessen Sven, Sonesson Björn, Malina Martin.

## Affiliation

Vascular Center Malmö / Lund, Malmö University Hospital, S-205 02 Malmö, Sweden

## Corresponding author

Jan Holst, Vascular Center Malmö / Lund, Malmö University Hospital, S-205 02 Malmö, Sweden, e-mail: jan.holst@med.lu.se, telephone: +46-40 33 10 00, fax: 46-40 33 80 96


## Category

Original article

## Running head

Ruptured AAA and emergency EVAR

## Abstract

**Objective** To evaluate the early and intermediate outcome of a consecutive series of emergency endovascular aneurysm repairs (eEVAR) of CT-verified infrarenal ruptured abdominal aortic aneurysm (rAAA) at a single tertiary referral center.

**Methods** Prospectively collected data of patients operated between April 2000 and Oct 2007 were retrospectively reviewed and all their pre, intra and postoperative imaging were re-evaluated. Patient and procedural data were analyzed using a Cox multiregression model.

**Results** 90 patients (86% men), 76 (±7) years were identified and included in the analysis. Symptom duration was <3h in 22% of patients, 3-24h in 39% and >24h in 39%. Mean aneurysm diameter was 73 (±14) mm. All patients were treated with the COOK Zenith® stentgraft (56% bi iliac and 44% uniiliac). 61% were heamodynamically unstable on presentation and 26% required an intra operative aortic occlusion balloon to maintain hemodynamic stability.

The 30 day and one year mortality was 27% and 37%, respectively. One-year aneurysm related mortality was 33%. 28% of patients required re-interventions during the follow up. The use of an aortic occlusion balloon and the presence of cerebrovascular disease or obstructive lung disorder correlated significantly with 30-day mortality in the multivariate analysis.

**Conclusion** EVAR is a valid treatment option for rAAA when used as a first line method for all comers.

***Key words***

Ruptured AAA, emergency EVAR, outcome, prediction

## Introduction

Despite advances in operative technique and peri-operative care over the past 50 years, the results for emergency open surgical repair (eOR) of ruptured abdominal aortic aneurysms (rAAA) remain dismal with mortality rates in the 50% range[1].

Endovascular AAA repair (EVAR) was described in the early 1990's by Parodi and Volodos and brought aortic aneurysm repair into the age of minimally invasive surgery[2, 3]. The use of EVAR for ruptured and symptomatic aneurysms (eEVAR) was described a few years later[4,5] and it is presently being promoted as the treatment of choice in the emergency setting[6]. A number of small series report mortality rates from 0-45% for eEVAR[5,7-16] but large variability both in device selection and different pathologies treated, make results hard to interpret. Non-randomized studies comparing eEVAR to eOR indicate a lower 30-day mortality for the former (0-34% vs. 0-54%)[7-27] but the number of eEVAR patients is small. The only prospective, randomized controlled trial published had a 53% 30-day mortality for both treatment modalities[28].

The aim of the present study is to report the early and intermediate results of 90 consecutive eEVAR procedures from a single referral center.

## Material and Methods

All patients with a CT confirmed rAAA treated with eEVAR between April 2000 and Oct 2007 were included in this study. Pseudoaneruysms, symptomatic aneurysms without evidence of rupture as well as fenestrated and custom-made devices were excluded.

At our institution, all EVAR procedures are performed in a hybrid operating room. Dedicated endovascular ancillary staff is available at all times. Since eEVAR has been the treatment of choice for rAAA at our department since 2001, the vast majority of patients with a rAAA are evaluated with CT upon clinical suspicion of aneurysm rupture.

Data were prospectively registered in a local database. For the purpose of the present study, all charts, CT-scans and angiograms were re-evaluated and verified and additional endpoints collected. Data were then retrospectively analyzed. . Mortality data were cross-referenced with the Swedish National Population Registry.

Hemodynamic instability was defined as loss of consciousness or systolic blood pressure <80 mmHg before induction of anaesthesia. Permissive hypotension was used until exclusion of the aneurysm was achieved. Blood pressure was regarded as satisfactory as long as the patient remained conscious[29]. Symptom duration was defined as the time passed since presentation of first clinical sign of rupture (i.e. pain, loss of consciousness etc.)

Only the commercially available bi- or uniiliac Zenith® stentgraft (Cook Inc, USA) was used. The indication for choosing a uniiliac device was technical (e.g. unilateral iliac occlusion or extreme tortuosity), clinical (extreme urgency) or logistical (appropriate bi-iliac system not available). Patients treated with an aortouniiliac device also received a femorofemoral bypass.

Standard iodized contrast (Omnipaque® 140-200 mg/ml, GE Healthcare, www.gehealthcare.com)

was used in most cases. In patients with renal insufficiency, standard contrast was replaced partially or fully by $CO_2$ at the surgeon's discretion. No fixed creatinine level was used for determining the choice of contrast. A percutaneous approach was preferred using either a "preclose" technique applying catheter delivered sutures (Perclose®/Proglide®, www.abbott.com) or a fascial closure technique[30].

If circulatory collapse occurred, an aortic occlusion balloon (Latex, Large Diameter Occlusion balloon or Coda® balloon catheter, www.cookmedical.com) (fig 1), was inserted transfemorally and inflated in the supra visceral aorta as described in detail previously[29].

Follow up consisted of clinical exams, contrast enhanced CT scans and plain abdominal X-rays at 1 month, 12 months and then annually for the remaining life time of the patient.

Morphological data were captured from thin slice CT scans (mostly 0.75-3 mm) performed with and without intravenous contrast. Care was taken to locate the most orthogonal projection to measure the shortest axial vessel diameter to avoid overestimation of vessel size due to tortuosity.

The local ethics committee at Lund University approved the study.

All continuous variables were normally distributed, except for the data regarding ICU and hospital stay. Continuous data are presented as mean and ± standard deviation. Comparisons were performed with standard Student's t-test and the corresponding paired t-test when appropriate. Non-normally distributed data are presented as median and interquartile range. Binary data were compared using the Fischer Exact test. Cox multivariate regression analyses and Kaplan-Meier for survival curves were performed. All tests were two-sided, p-values $<0.05$ were considered significant. The SPSS software, version 13.0 was used (www.spss.com).

## Results

90 eEVAR procedures were identified and included in the analysis. Patient data, AAA morphology, procedural data, outcome and re-intervention rates are summarized in tables 1-4. 32 patients (36%) had proximal aneurysm necks 15mm or shorter. No graft related endoleaks (type 1 or 3) were left untreated.

None of the 90 eEVAR procedures were converted to open repair. The all cause 30-day mortality was 27% (24/90). Six patients died peri operatively and another six within 24 hours. The remaining 12 patients died within 30 days due to multiple organ failure (MOF) or myocardial infarction (MI).

The patients had a mean of 4 (±2) co morbidities and risk factors, table 1. No single patient was without any co morbidities or risk factors. In a multivariate regression analysis testing the variables presented in table 1, cerebrovascular disease (p=0.007, rr: 3.8, 95% ci: 1.4-10.5) as well COPD (p=0.003, rr: 4.3, 95% ci: 1,7-11.0) significantly correlated to 30-day mortality. Among morphological and procedure related variables (specified in tables 2 and 3), only the use of aortic occlusion balloon correlated significantly to 30 day outcome (p=0.003, rr: 4.8, 95% ci: 1.7-13.0) illustration 1.

*Re-interventions*

24 (27%) patients underwent re-interventions during follow up (table 4). Eight open re-interventions were performed, all in the immediate postoperative phase. Seven of these underwent exploratory laparotomy for bowel ischemia (n=3), abdominal compartment syndrome (n=3) or bleeding (n=1). Of these, only one survived more than 30 days. One patient underwent common femoral patch repair due to occlusion. Seven patients underwent endovascular re-intervention; 6 during late follow up (2 additional stenting of endograft limbs due to kink, 2 embolization due to type 2 endoleaks and expanding aneurysms, 2 proximal Palmaz stent placement due to type 1

endoleak) and one early (diagnostic CO2 angiography only) due to worsened renal function postoperatively. 9 patients underwent combined open and endovascular re-interventions postoperatively; six in the acute phase (2 diagnostic angiography and exploratory laparotomy due to homodynamic instability, both negative; 2 SMA stenting and bowel resection due to iatrogenic SMA coverage by the aortic stentgraft, both died in-hospital; 1 iliac stentgraft and femoral interposition graft due to external iliac artery rupture; 1 iliac stenting, open balloon embolectomy and fasciotomy due to stentgraft limb occlusion) and 3 in the late phase ( 1 fenestrated proximal stentgraft extension due to proximal type 1 endoleak and femoral patch plasty; 1 open balloon embolectomy and re-stenting of limb; 1 patient underwent multiple re-interventions for type 1 endoleak and limb occlusions resulting finally in axillo-bifemoral bypass and lower limb amputation 3 years after the rAAA)

The mean clinical follow up was 27 months (±18) and the mean CT follows up was 24 months (±15). No patient was lost to follow up.

The cumulative survival data are presented in fig 2. The one-year all cause mortality was 37% (33 patients). Nine patients died between the second and 12$^{th}$ month. Six of the deaths were related to the procedure and included MOF (2), sepsis (1) MI (1) heart failure (1) and renal failure (1)). Three deaths were unrelated to the rAAA (2 malignancies, 1 gastroenteritis). Hence the one-year aneurysm related death was 33% (30/90). The three-year survival was 50%.

## Discussion

The majority of patients with rAAA die before reaching the hospital and it is estimated that only 10% reach the operating room alive[31]. Despite advances in open surgical repair and preoperative care, the mortality for open repair of rAAA remains between 45-50%[1]. The massive surgical trauma to the already massively traumatized patient with a rAAA is a lethal combination. The theoretical advantages to an endovascular approach are manifold. eEVAR can be performed percutaneous in local anaesthesia thus avoiding the vasodilating and negatively inotropic effects of general anaesthesia. Transfemoral access avoids the devastating effects of a large, midline laparotomy on muscle wall tonus , which can lead to further circulatory compromise. In addition, surgical dissection in the setting of a large retroperitoneal hematoma is avoided and blood loss minimized. Lastly, EVAR also allows for continuous aortic flow avoiding the effects of aortic cross clamping with potential reperfusion injury. The potential downsides of eEVAR include technical, anatomical and logistical issues. A preoperative CT is preferred, if not mandatory, and can cause unnecessary delay in treatment. Several studies, however, indicate that this plays a much lesser role for the outcome of these patients than previously believed. EVAR is also presently limited to patients with suitable aneurysm necks and adequate access and this will preclude a number of patients from an endovascular option if strict criteria are used for inclusion. Logistically, the need for appropriate intraoperative imaging, fully trained staff as well as the availability of correct endograft inventory also limits the widespread use of the technique.

Our data, with a 30-day mortality of 27%, correspond well to the previously published smaller series with respect to patient-, aneurysm-, procedure and outcome/complication -related data. Furthermore, they correspond to the recent meta-analyses by Lovegrove et al of 463 patients treated with eEVAR for rAAA[37]. This is somewhat surprising considering the very high rate of

heamodynamically unstable patients (61%) reflected in the high use of aortic occlusion balloon (26%) in which the outcome proved to be the worst. These acceptable results in the face of a high proportion of unstable patients might be due to selection bias and the fact that the report is not intention-to-treat and can only be validated with further studies.

The limitations of the present series are the inherent ones for single center retrospective reports and therefore any attempt at generalization must be done with caution. In addition, no comparison to eOR under the same study period was made. During the study period there has been a gradual shift towards eEVAR as presented earlier[24] and currently the amount of eEVAR by far exceeds eOR. Therefore any comparison may be unjust. Furthermore, the precise number of rAAA patients where no attempt at repair, open or endovascular, was made is not known. It might be argued that since we perceive eEVAR to be less traumatic for patients with rAAA, we apply it more liberally than we would with open repair. This will potentially affect results negatively if older, sicker patients are treated and thus produce worse outcome for EVAR than if stricter inclusion criteria were applied. Conversely, the morphological and logistical issues might in some cases disqualify patients for EVAR, forcing the unsuitable patients to undergo open repair, perhaps thereby negatively affecting outcome for this group. Obviously these issues can be resolved only if randomized studies are undertaken. There are, however, ethical issues being raised regarding a randomized trial. If the results of EVAR were truly as good as have been indicated in some studies and the results of OR are quite stable with mortality rates of 40-50% would it be ethically responsible to randomize patients? Ultimately, this decision remains with the individual physician or perhaps institution that has to scrutinize his or her own experience and the setting in which treatment is offered.

Questions regarding the general applicability of eEVAR compared to eOR have been raised indicating that if strict criteria for eEVAR are applied, only a minority of patients can be treated. Accepting wider inclusion criteria (shorter, wider necks or access problems) might affect the durability of the procedure negatively. One must keep in perspective, however, that the aim of

elective vs. emergency treatment is quite different. In the former, good short-term outcome is guaranteed for most and thus long-term durability becomes paramount. In the latter, short term outcome is more uncertain and thus tips the scale towards making this much more vital for the choice of repair. Consequently, the role of eEVAR and its ability to reduce immediate mortality speaks in its favour. Neither the question of applicability or durability (as indicated by secondary interventions) was specifically addressed in this series. However, others have looked into the matter. The overall applicability of eEVAR was approximately only 30% of rAAA cases in one study[38]. We estimate that our applicability is considerably higher, mainly due to a more favourable logistical setting and a more aggressive approach. The rate of reintervetion in our study was numerically (28%) higher compared to other reports where this parameter was the main endpoint of the study[15]. This may, at least, be partly due to the policy of having eEVAR as the first line of treatment of rAAA at our department, thus accepting anatomical and clinical parameters that will likely affect durability negatively.

Regardless of which modality is used to treat these very ill patients, it is unlikely that the long term all cause mortality will differ substantially as indicated in the randomized trials of elective EVAR[39,40] as well as the results in the present study with a 50% survival rate three years after the procedure. In the shorter perspective it may even be likely that the early and intermediate results of eEVAR will be less favourable as more and more difficult cases are being attempted. The future will probably reveal a final, appropriate equilibrium between eOR and eEVAR.

We believe that the favourable outcome in this large consecutive series of eEVAR may be attributed to several reasons. Firstly, the endovascular specialists, the vascular surgeons and the properly trained ancillary staff have worked together for a long time as one team accumulating a vast collective experience in the elective setting. Secondly, the in house logistics allow us to evaluate every patient for a possible eEVAR with a CT at all times. Thirdly, we have deliberately chosen to use only one type of modular stentgraft system (Cook Zenith® system), thus allowing the

entire team to become accustomed to a single device. The system is commercially available and we find it versatile in almost cases, as demonstrated by the 100% technical success rate in excluding the rAAA in the acute phase.

It seems logical that in the light of these and others cautiously promising results, the increasing number of elective EVARs with the parallel technical development of both the stentgraft design/delivery and imaging will further increase the use of eEVAR over eOR. Further studies will determine the ultimate role of eEVAR vs. eOR.

**Conflict of interest**

Prof Ivancev: has received grants from Cook Europe. Dr T Resch: proctor and lecturer for Cook Europe. None of the other authors have any disclosures.

References

1. Bown MJ, Sutton AJ, Bell PRF, Sayers RD. A meta-analyses of 50 years of ruptured abdominal aortic aneurysm repair. Br J Surg 2002;**89**:714-30.

2. Parodi JC, Palmaz JC, Barone HD. Transfemoral intraluminal graft implantation for abdominal aortic aneurysm. Ann Vasc Surg 1991;**5**:491-9.

3. Volodos NL, Karpovich IP, Troyan VI, Kalashnikova YuV, Shekhanin VE, Ternyuk NE, Neoneta AS, Ustinov NI, Yakovenko LF. Clinical experience of the use of self-fixing synthetic prostheses for remote endoprosthetics of the thoracic and the abdominal aorta and iliac arteries through the femoral artery and as intraoperative endoprosthesis for aorta reconstruction. Vasa Suppl 1991;**33**:93-5.

4. Yusuf SW, Whitaker SC, Chuter TAM, Wenham PW, Hopkins BR. Emergency endovascular repair of leaking aortic aneurysm. Lancet 1994;**334**:1645

5. Greco G, Egorova N, Anderson PL, Gelijns A, Moskowitz A, Nowygrod R, Arons R, McKinsey J, Morrissey NJ, Kent KC. Outcomes of endovascular treatment of ruptured abdominal aortic aneurysms. J Vasc Surg 2006;**43**:453-9.

6. Ohki T, Veith FJ, Sanchez LA, Cynamon J, Lipsitz EC, Wain RA, Morgan JA, Zhen L, Suggs WD, Lyon RT. Endovascular graft repair of ruptured aortoiliac aneurysms. J Am Coll Surg 1999;**189**:102-12.

7. Greenberg RK, Srivastava SD, Ouriel K, Waldman D, Ivancev K, Illig KA. Shortell C, Green RM. An Endoluminal method of hemorrage control and repair of ruptured abdominal aortic aneurysms. J Endovasc Ther 2000;**7**:1-7.

8. Hincliff RJ, Yusuf SW, Macierewicz JA, MacSweeney STR, Wenham PW, Hopkinson BR. Endovascular repair of ruptured abdominal aneurysm – a challenge to open repair? Results of a single centre experience in 20 patients. Eur J Vasc Endovasc Surg 2001;**22**:528-34.

9. Lachat ML, Pfammatter T, Wittzke HJ, Bettex D, Künzli A, Wolfwensberger U, Turina MI. Endovascular repair with bifurcated stent-grafts under local anaesthesia to improve outcome of ruptured aortoiliac aneurysms. Eur J Vasc Endovasc Surg 2002;**23**:528-36.

10. Doss M, Martens S, Hemmer W. Emergency endovascular interventions for ruptured abdominal thoracic and abdominal aortic aneurysms. Am Heart J 2002;**144**:544-8.

11. Orend KH, Kotsis T, Scharrer-Pamler R, Kapfer X, Liewald F, Görich J, Sunder-Plassman L.

Endovascular repair of aortic rupture due to trauma and aneurysm. Eur J Vasc Endovasc Surg 2002;**23**:61-7.

12. Yilmaz N, Peppelenbosch N, Cuypers PW, Tielbeck AV, Duijm LE, Buth J. Emergency treatment of symptomatic or ruptured abdominal aortic aneurysms: the role of endovasc repair. J Endovasc Ther 2002;**9**:449-57.

13. Resch T, Malina M, Lindblad B, Dias NV, Sonesson B, Ivancev K. Endovascular repair of ruptured abdominal aortic aneurysm: logistics and results. J Endovasc Ther 2003;**10**:440-6.

14. Hechelhammer L, Lachat ML, Wildermouth S, Bettex D, Mayer D, Pfammatter T. Midterm outcome of endovascular repair of ruptured abdominal aortic aneurysm. J Vasc Surg 2005;**41**:752-7.

15. Oranen BI, Bos WTGJ, Verhoeven ELG, Tielliu IFJ, Zeebragts CJ, Prins TR, van den Dungen JJAM. Is emergency endovascular aneurysm repair associated with a higher secondary intervention risk at midterm follow-up? J Vasc Surg 2006;**44**:1156-61.

16. Moore R, Nutley M, Cina CS, Motamedi M, Faris P, Abuznadah W. Improved survival after introduction of an endovascular therapy protocol for ruptured abdominal aortic aneurysms. J Vasc Surg 2007;**45**:443-50.

17. van Sambeek MR, van Dijk LC, Hendriks JM, van Grotel M, Kuiper JW, Pattynama PM, van Urk H. Endovascular versus conventional open repair of acute abdominal aortic aneurysm: feasibility and preliminary results. J Endovasc Ther 2002;**9**:443-8.

18. Veith F, Ohki T. Endovascular approaches to ruptured infrarenal aorto-iliac aneurysms. J Cardiovasc Surg 2002;**43**:369-78.

19. Larzon T, Lindgren R, Norgren L. Endovascular treatment of ruptured abdominal aortic aneurysms: a shift of the paradigm? J Endovasc Ther 2005;**12**:548-55.

20. Vaddineni SK, Russo GC, Patterson MA, Taylor SM, Jordan WD. Ruptured abdominal aortic aneurysm: a retrospective assessment of open versus endovascular repair. Ann Vasc Surg 2005;**19**:782-786.

21. Alsac JM, Desgranges P, Kobeiter H, Becquemin JP. Emergency endovascular repair for ruptured abdominal aortic aneurysms: feasibility and comparison of early results with conventional open repair. Eur J Vasc Endovasc Surg 2005;**30**:632-9.

22. Coppi G, Silingardi R, Gennai S, Saitta G, Ciardullo AV. A single-center in open and endovascular treatment of hemodynamically unstable and stable patients with ruptured abdominal aneurysms. J Vasc Surg 2006;**44**:1140-7.

23. Franks S, Lloyd G, Fishwick G, Bown M, Sayers R. Endovascular treatment of ruptured and symptomatic abdominal aortic aneurysms. Eur J Vasc Endovasc Surg 2006;**31**:345-350.

24. Acosta S, Lindblad B, Zdanowski Z. Predictors for the outcome after open and endovascular repair of ruptured aneurysms. Eur J Vasc Endovasc Surg 2006;**33**:277-84.

25. Visser JJ, Bosch JJ, Hunink MGM, van Dijk LC, Hendricks JM, Poldermans D, van Sambeek MRHM. Endovaskular repair versus open surgery in patients with ruptured abdominal aortic aneurysms: clinical outcomes with 1-year follow-up. J Vasc Surg 2006;**44**:1148-55.

26. Najjar SF, Mueller KH, Ujiki MB, Morasch MD, Matsumura JS, Eskandari MK. Percutaneous endovascular repair of ruptured abdominal aortic aneurysms. Arch Surg 2007;**142**:1049-52.

27. Ockert S, Schumacher H, Böckler D, Megges I, Allenberg JR. Early and midterm results after open and endovascular repair of ruptured abdominal aortic aneurysms in a comparative analysis. J Endovasc Ther. 2007;**14**:324-32.

28. Hinchliffe RJ, Bruijstens L, MacSweeney STR, Braithwaite BD. A randomised trial of endovascular and open surgery for ruptured abdominal aortic aneurysm - results of a pilot study and lessons learned for future studies. J Vasc Endovas Surg 2006;**32**:506-13.

29. Malina M, Veith F, Ivancev K, Sonesson B. Balloon occlusion of the aorta during endovascular repair of ruptured abdominal aortic aneurysm. J Endovasc Ther 2005;**12**:556-9.

30. Larzon T, Geijer H, Gruber G, Popek R, Norgren L. Fascia suturing of large access sites after endovascular treatment of aortic aneurysms and dissections. J Endovasc Ther 2006;**13**:152-7.

31. Brown LC, Powell JT, UK small aneurysm trial participants. Risk factors for aneurysm rupture in patients kept under ultrasound surveillence. Ann Surg 1999;**230**:289-97.

32. Wilmink TBM, Quick CRG, Day N. The association between cigarette smoking and abdominal aortic aneurysms. J Vasc Surg 1999;30:1099-105.

33. Schouten O, van Laanen JH, Boesma E et al. Statins are associated with a reduced infrarenal abdominal aortic aneurysm growth. Eur J Vasc Endovasc Surg 2006;**32**:21-6.

34. Sukhija R, Aronow WS, Sandhu R, Kakar P, Babu S. Mortality and size of abdominal aortic aneurysm at long-term follow-up of patients not treated surgically and treated with and without statins. Am J Cardiol 2006;**97**:279-80.

35. Urbonavicius S, Urbonavicius G, Honoré B, Henneberg EW, Vorum H, Lindholt JS. Potential circulating biomarkers for abdominal aortic aneurysm expansion and rupture – a systematic review Eur J Endovasv Surg 2008;**36**:273-80.

36. Wanhainen A, Bylund N, Björk M. Outcome of after abdominal aortic aneurysm repair in Sweden 1994-2005. Br J Surg 2008;**95**:564-70.

37. Lovegrove RE, Javid M, Magee TR, Galland RB. A meta-analyses of 21 178 patients undergoing open or endovascular repair of abdominal aortic aneurysm. Br J Surg 2008;**95**: 677-84.

38. Hoornweg LL, Wisselink W, Vahl A, Balm R. The Amsterdam trial: suitability and application rate for endovascular repair of ruptured abdominal aortic aneurysm. Eur J Vasc Endovasc Surg 2007;**33**:679-83.

39. EVAR trial participants. Endovascular aneurysm repair vs open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. Lancet 2005;**365**:2179-86.

40. Blankensteijn JD, de Jong SE, Prinssen M, van der Ham AC, Buth J, van Sterkenburg SM, Verhagen HJ, Buskens E, Grobbee DE; Dutch Randomized Endovascular Aneurysm Management (DREAM) Trial Group. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. N Engl J Med 2005;**352**:2398-405.

Fig 1

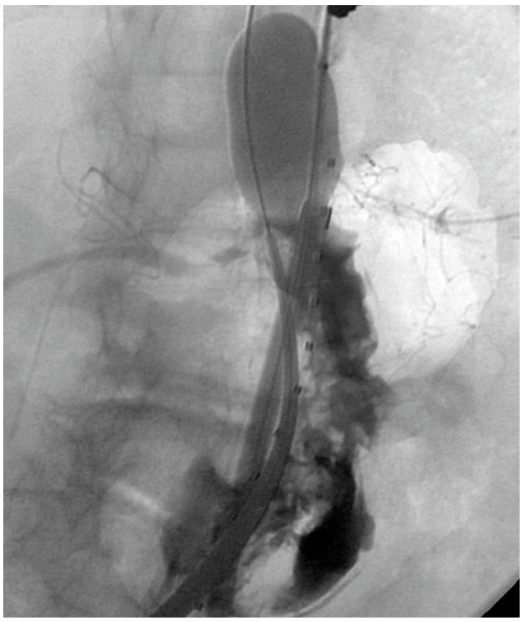


Table 1

Patient data

| | |
|---|---|
| Number of pat | 90 (14% female) |
| Age (years) | 76 ±7 |
| Co morbidities | |
| Coronary | 37% |
| Heart failure | 40% |
| Hypertension | 64% |
| Cerebro vascular | 20% |
| Renal insuff. | 28% |
| Diabetes | 14% |
| COPD | 31% |
| Risk factors[†] | |
| Smoking | 80% |
| Previous abd.surgery | 29% |
| Duration of symptoms (h) | |
| <3 | 22% |
| 3-24 | 39% |
| >24 | 39% |
| Heamodynamically unstable | 61% |
| Pre-op haemoglobin (g/L) | 102 ±19 |
| Creatinine (μmol/L) | 131 ±60 |

Table 2

| AAA morphology (mm) | |
|---|---|
| aorta diameter | 73 ±14 |
| proximal neck diameter | 24 ±4 |
| neck length | 21 ±10 |
| >15mm | n=58 |
| 15 mm or less | n=32 |
| right iliac diameter | 18 ±6 |
| left iliac diameter | 17 ±6 |

Table 3

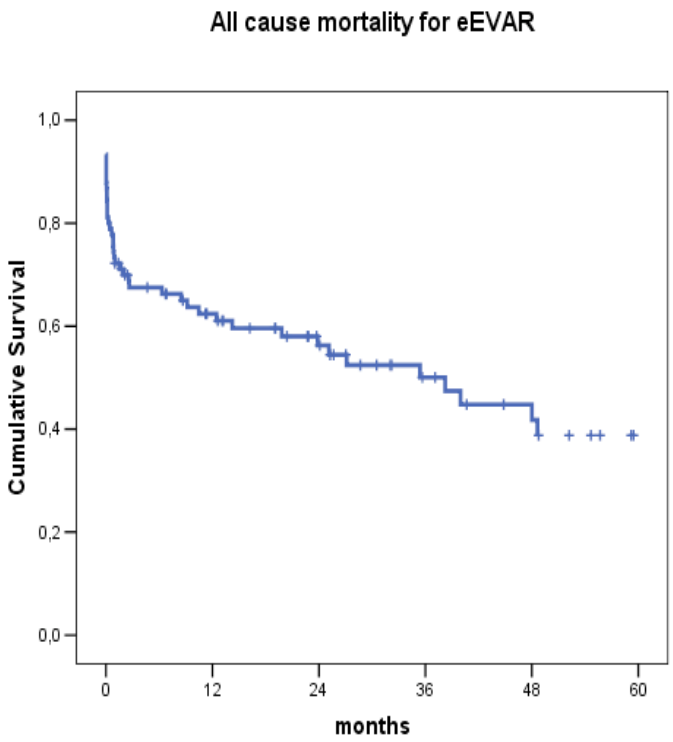
## Procedural data

| | |
|---|---|
| Biiliac system | 56% |
| Uniiliac system | 44% |
| Proximal stentgraft diameter | 31 ±3 |
| Anaesthesia form | |
| General | 50% |
| Local | 50% |
| Occlusion balloon | 26% |
| Procedural time (min) | 207 ±82 |
| Blood loss (mL) | 1170 ±1230 |
| Transfusion (U) | |
| packed red cells | 6 ±5.4 |
| fresh frozen plasma | 4 ±4 |

Table 4

| Outcome | |
|---|---|
| Conversions | 0% |
| Length of care (days) | |
| ICU | 2 ±3 |
| LOS | 8 ±11 |
| Follow up (m) | |
| Clinical | 27 ±18 |
| CT-angiogram | 24 ±15 |
| 30 day mortality | 27% (24/90) |
| 1-year mortality | |
| All cause | 37% (33/90) |
| Aneurysm related | 33% (30/90) |
| 3 year mortality | 50% |
| Cause of death | |
| 30 days: | uniformly heart/vascular related or multiple organ failure |
| 1 year (non-aneurysm related) | 2 neoplastic, 1 gastroenteritis |
| Creatinine ($\mu$mol/L) | |
| Pre-operatively | 118 ±59 |
| Last FU | 133 ±118 |
| Re-rupture | 0% |
| Diameter change over time (mm) | |
| Pre-operatively | 73 ±14 |
| Last FU | 56 ±15 |
| Detection of endoleak | 4 late type 1 endoleaks treated |
| Migration during FU | 0% |
| Re-intervention | |
| Collectively | 27% (24/90) |
| Open | 8/25 |
| Endovascular | 7/25 |
| Combination | 9/25 |

Fig 2




| time | 0 | 30d | 12m | 24m | 36m |
|---|---|---|---|---|---|
| number at risk | 90 | 66 | 46 | 32 | 20 |

## Legends

Fig 1:

The use of intra operative occlusion balloon[29] (Latex, Large Diameter Occlusion balloon or Coda® balloon catheter, www.cookmedical.com), as illustrated above, due to severe preoperative hemodynamic instability was the only procedure related variable that correlated significantly with 30 day mortality (p=0.003, rr: 4.8, 95% ci 1.7-13.0).

Table 1:

The coronary co morbidity was defined as angina pectoris, history of myocardial infarction, medication to prevent or treat these conditions or previous revascularisation. Renal insufficiency: creatinine >150 µmol/L, n=24/90) or manifest (hemo- or peritoneal dialysis, n=3/90). Diabetes includes both insulin and non-insulin dependent disease. Hemodynamic instability was defined as loss of consciousness or recorded blood pressure <80 mm/Hg at any time pre-op. The value of the pre-operative haemoglobin concentration did not take into account the duration of symptoms or any intravenous fluid given before the intervention.

Cerebrovascular disease and COPD correlated significantly to the 30-day mortality (p=0.007, rr: 3.8, 95% ci: 1.4-10.5 and p=0.003, rr: 4.3, 95% ci: 1,7-11.0).

The use of statins and body mass index was not recorded in the database.

Table 2:

All CT angiograms were re-evaluated and control measurements were performed. The AAA morphology did not influence the outcome and there was no significant gender difference.

Table 3:

The anaesthesia team estimated the procedural time and the blood loss . These parameters include the time and the amount of bleeding when the uniiliac system (44% of the cases) was followed by the femorofemoral bypass. The transfusions are those given during the first 24 hours.

Table 4:

Data on the time of hospitalisation have a skewed distribution, hence presented as median and interquartile range (all other data as mean and standard deviation). The observed creatinine values are from the patients who survived 30 days, patients that were on permanent dialysis pre-operatively excluded. The paired comparison revealed a non-significant ($p=0.343$) increase. The paired comparison between the original CT and latest control revealed a significant ($p<0.001$) shrinkage of the AAA.

Fig 2:

The cumulative survival (Kaplan-Meier) for all cause mortality after 30 days and 1 year was 73% and 63% respectively. The causes of death within the first month were either multiple organ failure or myocardial/vascular. Two out three of the deaths that occurred between the second and the twelfth month were aneurysm related.

After three years half of the patients were alive.