Treatment of ruptured abdominal aortic aneurysm after endovascular abdominal aortic repair: A comparison with patients without prior treatment

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Objective: **A retrospective analysis of immediate outcomes following aneurysm rupture (rAAA) in two groups: patients previously treated at our center with primary endovascular repair (EVAR) and patients without previous EVAR for abdominal aortic aneurysms (AAA) in an 8-year period.**

Methods: **Fourteen patients with a confirmed rAAA identified throughout the follow-up period following primary EVAR repair at our center (from a population of 820 AAA treated at our center in election) were retrospectively compared with 155 patients without previous EVAR in the same time period, from the introduction of an intention-to-treat protocol with EVAR for rAAA in January 1999. Primary study outcomes included 30-day mortality and severe systemic complications following rAAA correction with both open and EVAR treatments.**

Results: **In the 14 patients secondary interventions were necessary throughout follow-up prior to rupture in 43% (6/14). The mean time to rupture was 50.23 months (9-113). The mean increase in maximum aneurysmal diameter at rupture was 18.39 mm. Type of endoleaks observed at rupture: 35.7% I proximal, 35.7% III contralateral stump disconnection, 14.3% I distal, 14.3% III midgraft tear: treatment at rupture included five EVAR corrections with aortouniiliac endografts, four EVAR corrections with extensions, and five surgical conversions. Thirty-day mortality between the two groups, 28.5% (patients with prior EVAR) 38.7% (patients without prior EVAR), and severe systemic complications, 50% vs 37.6%, were not found to be statistically significant. Hemodynamic instability, 36% (patients with prior EVAR) 63% (patients without prior EVAR), was found to be an independent predictor of 30-day mortality ($P < .0001$), whereas severe systemic complications, 50% vs 33.5%, did not influence the same outcome ($P = .852$).**

Conclusions: **In terms of mortality, it would be logical to expect a protection from the endograft in patients with previous EVAR. A trend seems to confirm this assumption, but no statistical significance was found, which may be due to the small population size. (J Vasc Surg 2009;49:582-8.)**

Protecting patients with abdominal aortic aneurysms (AAA) from rupture and aneurysm associated death is the main goal of endovascular repair (EVAR). The treatment aims to completely exclude the aneurysmal sac from circulation. The traditional surgical technique dictates the surgical opening of the aneurysmal sac and the replacement of the arterial wall with a vascular prosthesis, theoretically avoiding the risk of rupture.[1] Comparatively, EVAR has an incidence of incomplete exclusion throughout the follow-up period, which in literature ranges from between 6% to 50% of patients,[2] the most frequent reason being caused by endoleak, what has been termed the "Achilles heel" of EVAR.[3] Graft related endoleaks (type I and III) are associated with a late risk of rupture, which is assumed but not proven to be associated with significant pressurization of the aneurysmatic sac.[4]

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Since the introduction of EVAR, complications related to device failures and endoleaks following EVAR have led some authors to urge caution in the widespread application of EVAR.[5,6] Recent studies have begun to publish more advantageous long-term results,[7,8] especially associated with second generation devices, but an undefined durability of EVAR still exists.

At rupture, an independent risk factor associated with immediate mortality is the patients' hemodynamic stabilty.[9] This led us to question that if in the case of rupture, the presence of an endograft constitutes a protection in terms of improved hemodynamic stability and therefore mortality.

METHODS

From January 1999, our center has followed a protocol for ruptured abdominal aortic aneurysms (rAAA) with an intention-to-treat with EVAR for all anatomically suitable patients, excluding young patients (less than 65-years-old).

From January 1999 to December 2007, a total of 169 consecutive patients with rAAA were retrospectively evaluated according to prior primary EVAR for AAA at our center (14, one patient was subsequently treated for rAAA at another center) or patients without any prior AAA treatment (155). Patients with acute or symptomatic but intact aneurysms shown at computed tomography (CT) were excluded.

These patients were further categorized according to their hemodynamic stability in the period between presentation at hospital through to admittance into the operating suite; as hemodynamically unstable (defined as unconscious and/or with a systolic blood pressure constantly less than 80 mm Hg, following fluid resuscitation) or hemodynamically stable (conscious and/or with a systolic blood pressure constantly greater than 80 mm Hg, with or without fluid resuscitation). All patients were assisted by a vascular surgeon and an anesthesiologist from their presentation at emergency through the operating room. Permissive hypotension was practiced with prudent fluid resuscitation to keep systolic blood pressure around 80 mm Hg. All patients were assessed by contrast enhanced spiral computed tomography (CT) 5 mm slices of the abdomen before entering the operating suite, apart from those less than 65 years of age or with severe hypotension (unconscious or with a systolic blood pressure less than 50 mm Hg, with or without fluid resuscitation), who were transported directly into the operating suite and assessed for EVAR in the operating room if a hemodynamic response was achieved with an occlusion balloon. Rupture with CT was defined as extravasation of blood surrounding the aneurysm as evident by the scan, and in the cases of patients sent immediately into the operating room, rupture was confirmed by intraoperative angiography and intravascular ultrasound (IVUS), or directly during traditional surgery. During the CT scanning, the operating room was prepared, in order to further reduce delay. In the case of a CT scan, the patients' anatomic suitability for emergency EVAR was directly determined by the vascular surgeon from the screen.

Our EVAR procedures have been previously described.[9] All endografts used in the EVAR correction for rAAA were commercially available, detailed in Table I. The endografts were chosen according to the patients' anatomical characteristics, as interpreted by the vascular surgeon from the preoperative diagnostic examination.

Open repair was performed in the traditional fashion as is described in literature.[10-12] All grafts used in the study were commercially available (Table I).

The EVAR follow-up scheme consisted of routine CT before discharge, at 3 and 12 months and annually thereafter, and echo duplex scanning at 1 and 6 months and annually thereafter. From January 2007, the annual CT scan was replaced with an echo duplex scan and a subsequent CT scan if required, in order to reduce radiation exposure and the administration of contrast media, plain X-ray previous to discharge, at 6 months and annually thereafter. Open follow-up consisted of an echo duplex scan at 6 months and annually thereafter.

Data are expressed as mean (range), if continuous, and as absolute and relative frequency, if categorical. Comparisons between continuous variables are performed by means of *t* test for unpaired samples, while categorical variables by means of χ^2 or Fisher exact test, as appropriate. Univariate binary logistic regression is used to find out predictors of 30-day mortality. All two-sided *P* value $< .05$ are considered statistically significant.

Table I. Devices and surgical techniques used in the treatment of rAAA

Variables	*rAAA with prior primary EVAR*	*rAAA without prior EVAR*
Patients, n	14	155
Endografts, extensions and grafts:		
Powerlink	1 (1 ext)	4 (2 Bif, 1 AUI, 1 Tube)
Talent	1 (1 AUI)	11 (1 Bif, 10 AUI)
Zenith	5 (4 AUI, 1 ext[a])	32 (2 Bif, 30 AUI)
Excluder	3 (7 ext[b])	7 (5 Bif, 2 Tube)
Hemashield Gold	—	90
Bard	—	11
EVAR conversion – banding	1	—
Conversion with bypass	4	—

EVAR, Endovascular repair; *rAAA,* ruptured abdominal aortic aneurysms; *Bif,* bifurcated endograft; *AUI,* aortouniiliac endograft; *Tube,* straight tube endograft; *ext,* extension.
Powerlink (Endologix Inc, Irvine, Calif); Talent (Medtronic Vascular, Santa Rosa, Calif); Zenith (Cook Inc., Bloomington, Ind); Excluder (W.L. Gore and Associates, Flagstaff, Ariz); Hemashield Gold (Boston Scientific, Natick, Mass); Bard (Bard Inc. Murray Hill, NJ).
[a]One patient was treated for a rAAA at primary treatment.
[b]One patient was treated with both a Cook and Gore extension.

RESULTS

A total of 169 patients underwent treatment for rAAA at our center in an 8-year period from the introduction of both EVAR and open surgery as treatment options for rAAA at our center; 155 primary rAAA treatment and 14 treated for rAAA after EVAR.

Patient demographic, comorbidities, and preoperative aneurysm anatomical features are displayed in Table II. The two groups were found to be heterogeneous regarding the incidence of hyperlipidemia ($P = .022$) and obesity ($P = .036$).

Analyzing the follow-up from the initial repair through to rupture, a rate of 36% of patients (5/14) who had not followed the correct follow-up protocol was highlighted (Table III). Six patients (43%) had been treated with 8 secondary interventions prior to rupture; 2 type I proximal endoleak (Zenith AUI, proximal AneuRx extension [Medtronic, Minneapolis, Minn]), 1 type I distal endoleak (Zenith extension), 3 type III contralateral stump disconnection (2 Excluder and 1 Passager extensions [Boston Scientific, Galway, Ireland]), 1 type III midgraft tear (Excluder extension), and 1 contralateral branch thrombosis treated with thrombolysis and angioplasty (Table IV).

The mean time between the last consultation and rupture was 21 months. The mean time to rupture from the primary EVAR repair was 50.23 months, ranging from 9 to 113 months (Table IV).The mean AAA diameter increase of 18.39 mm with respect to the preoperative measurement was found to be statistically significant ($P < .0001$). Interestingly, rupture in five cases was associated with a type I proximal endoleak, of which 80% were stable and 20%

Table II. Summary of patient demographics, comorbidities, and preoperative aneurysm anatomical features

Variable	*rAAA with prior EVAR treatment*	*rAAA without prior EVAR treatment*	P *value*
Patients, n	14	155	
Median age, y (range)	82.97 (65-92)	78.57 (53-99)	.154
Sex (M/F)	12/2	125/30	1.000
Stability, % (stable/unstable)	64/36	37/63	.049
Comorbidities, %:			
Diabetes	0	9	.609
Smoker	14	35	.146
Hypertension	71	61	.571
Hyperlipidemia	0	29	.022
Cardiac disease	50	46	.763
Carotid disease	7	6	.589
Renal disease	0	12	.372
Respiratory disease	29	30	1.000
Previous laparotomy	14	11	.660
Obesity	7	37	.036
AAA maximum diameter, mm, mean (range)	81.96 (66-103)	78.23 (60-107)	.159
Neck diameter, mm, mean (range)	23.64 (19-30)	24.92 (17-34)	.253
Neck length, mm, mean (range)	20.73 (15-30)	19.92 (0-50)	.562

EVAR, Endovascular repair; *rAAA,* ruptured abdominal aortic aneurysms; *ASA,* American Society of American Anestheologists; *Bif,* bifurcated endograft; *AUI,* aortouniiliac endograft; *Tube,* straight tube endograft; *ext,* extension.

Table III. Individual patient details: initial AAA intervention details, significant events throughout follow-up period, last check-up prior to rupture, and time to rupture

Pt. No.	*Endograft (year of primary EVAR)*	*AAA maximum diameter preop at initial EVAR*	*Treated complications during follow-up (mo, from initial EVAR intervention)*	*Time from last check-up to rupture (mo)*	*Time to rupture (mo)*
1	Talent (1999)	80	Suspected endoleak at echo-color Doppler, CT refused (84) – untreated	2	61
2	Vanguard (1997)	73	No	36[b]	36
3	Vanguard (1996)	60	Thrombolysis + PTA (6)	68[b]	74
4	Powerlink (2004)	66	No	12	19
5[a]	Zenith (2004)	47	Type I distal (12)	15	36
6	Talent (2004)	65	No	5	14
7	Powerlink (2002)	58	No	31[b]	33
8	Stentor (1996)	60	Type III CSD (36)	12	93
9	Powerlink (2002)	50	No	8	20
10	Powerlink (2007)	78	Type I proximal (1)	4	9
11	Vanguard (1999)	55	No	36[b]	39
12	Vanguard (1996)	60	No	46[b]	46
13	Vanguard (1997)	55	Type I proximal (36)	12	79
14	Vanguard (1997)	80	Type III CSD (48, 84)	12	113

EVAR, Endovascular repair; *rAAA,* ruptured abdominal aortic aneurysms; *AAA,* abdominal aortic aneurysms; *CT,* computed tomography; *CSD,* contralateral stump disconnection.

[a]Original diagnosis and treatment for rAAA.

[b]Patients who had not followed the prescribed follow-up protocol.

unstable, and in five cases a type III contralateral stump disconnection was evidenced with an inversion of hemodynamic condition; 20% stable and 80% unstable (Table V).

Ruptured aneurysms were corrected with EVAR in 9 cases (5 with AUI, 4 with endovascular extensions), with an EVAR conversion in 1 case (surgical banding around the endograft which was not removed), and 4 patients were converted to open repair (3 due to the inability to cannulate the stump through the disconnected limb due to tortuosity in a type III endoleak and an incomplete sealing with a proximal extension for a type I proximal endoleak) (Table IV).

The immediate mortality (within 24 hours) in this group was 14% (2/14); one patient presented stable and during traditional surgery fell into a state of instability and subsequently died for irreversible shock, and one stable patient treated with EVAR died due to multiorgan failure (MOF). A further two deaths occurred within 30 days, for MOF, culminating in a 30-day mortality rate of 28.5% (Table VI).

Table IV. Follow-up, endoleak, and treatment of rAAA patients with previous EVAR treatment

Variable	*rAAA with prior EVAR treatment*
Patients, n	14
Follow-up from initial EVAR treatment through to rAAA:	
Patients who hadn't followed follow-up protocol, %	36 (5/14)
Patients with secondary interventions, %	43 (6/14)
Time to rupture, mo (range)	50.23 (9-113)
Condition of the aneurysm at rupture:	
Endoleak, %:	
Type I proximal	35.7 (5/14)
Type I distal	14.3 (2/14)
Type III midgraft tear	14.3 (2/14)
Type III contralateral stump disconnection	35.7 (5/14)
AAA maximum diameter, mm, mean (range)	81.96 (66-103)
Average increase in AAA maximum diameter, mm (range)	18.39 (1-30)
Treatment of rAAA:	
EVAR correction with AUI	5
EVAR correction with extensions	4
Conversion[a]	5

EVAR, Endovascular repair; *rAAA,* ruptured abdominal aortic aneurysms; *AUI,* aortouniiliac endograft.
[a]One EVAR conversion, one EVAR correction abandoned due to problems associated with the introduction of the device.

Seven patients (50%) suffered severe systemic complications, detailed in Table VI.

Comparatively, the treatment of rAAA without prior EVAR includes 101 patients treated with open surgery and 54 with EVAR. A comparative analysis resulted in a statistically significant difference being found between the hemodynamic stability of the two groups in favor of those previously treated with EVAR ($P = .049$). In the entire population of rAAA with and without prior EVAR, instability was found to be a significant predictor of 30-day mortality (if unstable: odds ratio [OR] = 4.77, 95% confidence interval [CI] 2.3-9.9, $P < .0001$); conversely, the incidence of systemic complications did not significantly influence 30-day mortality (in case of complication: OR = 1.07, 95% CI 0.6-2.1, $P = .852$).

DISCUSSION

The main goal of EVAR for AAA is to prevent rupture and it is well known that EVAR is not completely effective in reaching this target.[13-16] The Eurostar registry, as quoted by Szmidt,[2] found a cumulative annual risk of rupture after EVAR of 2% at 6 years; these results were validated by other studies.[2,7,17]

The mean time to rupture was into the long term (50.23 months); two of these cases, however, were evidenced at 9 and 14 months (Table III). This study not only verifies the need for continued long-term surveillance for device related complications as often quoted in literature,[18-23] but that this surveillance needs to be effective also in the midterm.

Of the 14 patients with rAAA after EVAR, 43% (6/14) had been treated with a reintervention throughout the follow-up prior to rupture (Table III). An average reintervention rate in literature following elective EVAR for AAA is far less at around 14%.[22-24] We have hypothesized two possible explanations for this discrepancy: (1) previous attempted correction had been unsuccessful or (2) that the introduction of catheters and other devices could interrupt endograft stability, leading to endoleak and eventual rupture. Zarins et al[25] in a study of 923 patients treated with EVAR for AAA, demonstrated that the mean maximum aneurysm diameter is a significant independent predictor of rupture, and highlights a percentage of rupture of 3% for aneurysm diameters from 50 to 59mm compared with 7% for those greater than 60 mm. This finding is supported in other studies.[7,17,26-30] The average preoperative aneurysm diameter for the group who subsequently experienced rupture was 63.36 mm, which significantly increased to 81.96 mm ($P < .0001$). Aneurysm sac changes, however, throughout follow-up as a prediction of rupture is controversial and often unreliable.[19,31] Four patients in this study had stable or minimal aneurysm sac enlargement (<5 mm) within a year prior to rupture.

In all ruptured aneurysms following EVAR, an endoleak was observed. Endoleak represents the most frequent cause of aneurysm rupture after EVAR.[3,13] Endoleaks observed were all type I (50%) and type III (50%), with no evidence of type II endoleaks (Table III). Type I and III endoleaks are most commonly associated with rupture,[16,32,33] argued by Hinnen et al[4] as being due to a higher pressurization of the aneurysmatic sac. Further, our study found an association between type III endoleak and hemodynamic instability (4/7) compared with type I endoleak (1/7) (Table IV). The reason for this is unclear. We hypothesize that in case of a rupture due to a type I endoleak, the volume of blood is filtered between the endograft and the aortic wall into the aneurysmal sac, and at point of rupture, the blood volume that passes could be considered less than that which passes at full channel into the aneurysmatic sac through the endograft's stump in the case of a limb disconnection or a large fabric tear. In turn, the quantity of blood loss affects the patients' instability, which is often correlated with poor outcomes.[9,21]

In light of these findings, in the last 3 years we have intensified the advised follow-up, above all for patients who were treated for an aneurysm greater than 60 mm or who required a reintervention. We have substituted the annual CT scan to a bi-annual echo duplex scan, with CT scans requested when more precise imaging is required; such as in the case of increased sac size >5 mm, endoleak, or changes in endograft integrity or suspected migration. However, we must specify that the biannual consultations do not completely eliminate the risk of rupture. Three patients were visited within 6 months prior to rupture, and only one patient had signs of an

Table V. Patients treated for rAAA with previous EVAR compared with those treated for rAAA without previous EVAR

Variable	*rAAA with prior EVAR treatment*			*rAAA without prior EVAR treatment*			*Statistical significance* P *value*
	Total	*Open*	*EVAR*	*Total*	*Open*	*EVAR*	
Patients, n	14	5	9	155	101	54	
Stability %, (Stable/unstable)	64/36	60/40	67/33	37.4/62.6	37/64	39/61	.049
Immediate mortality, % (within 24 h)	14	20	11	27	29.7	22.2	.524
30 day mortality, %	28.5	40	22	38.7	43.6	29.6	.571
Severe systemic complications, %	50	40	55.5	33.5	37.6	26	.216
MOF (fatal)	—	1	2	—	12	1	—
Bowel infarction	—	—	—	—	8 (6 fatal)		—
ACS		—	1	—	1	3	—
Acute renal failure (dialysis)	—	—	—	—	4	—	—
Stroke	—	—	—	—	2 (1 minor)	1	—
Severe respiratory insufficiency	—	—	1	—	8	4	—
Gastrointestinal bleeding	—	—	—	—	1		—
Myocardial infarction	—	1	1	—	2	2	—
Creatinine rise >30%	—	—	—	—	—	3	—
Intestinal ischemia	—	—	—	—	—	1	—
Digestive hemorrhage	—	—	—	—	—	1	—

EVAR, Endovascular repair; *rAAA,* ruptured abdominal aortic aneurysms; *MOF,* multiorgan failure.

Table VI. Individual patients hemodynamic situation, diagnosis and treatment and postoperative events

Pt. No.	*Hemodynamic situation at presentation*	*Endoleak*	*Treatment at rupture*	*30-day mortality*	*Systemic complications*
1	Stable	Type I proximal	EVAR AUI (Cook) + Cross over	Irreversible shock	
2	Stable	Type III CSD	Attempted EVAR correction abandoned due to problems associated with the introduction of the device – Conversion aorto-aortico		
3	Stable	Type I distal	EVAR Extensions (2 × Gore)	IMA	Cardiac Arrest
4	Stable	Type I proximal	EVAR AUI (Cook) + Cross over		IRA
5[a]	Stable	Type III midgraft tear	EVAR Conversion − banding		
6	Stable	Type I proximal	EVAR AUI (Cook) + Cross over		ACS
7	Stable	Type I proximal	EVAR Extension (Endologix) + Palmaz stent		
8	Stable	Type III CSD	EVAR AUI (Cook) + extensions (2 × Gore) + cross over		
9	Stable	Type I proximal	Conversion (at another center)	MOF	
10	Unstable	Type I distal	EVAR Extensions (Cook and Gore)		
11	Unstable	Type III CSD	Conversion − aorto-aortico	IMA	IMA
12	Unstable	Type III CSD	Conversion − aorto-iliaco		
13	Unstable	Type III midgraft tear	EVAR AUI (Talent) + Cross over		
14	Unstable	Type III CSD with caval fistula	EVAR Extensions (2 × Gore)		

EVAR, Endovascular repair; *CSD,* contralateral stump disconnection; *AUI,* aortouniiliac endograft; *IMA,* miocardial infarction; *MOF,* multiple organ failure; *IRA,* severe respiratory insufficiency; *ACS,* abdominal compartment syndrome.
[a]Original diagnosis and treatment for rAAA.

endoleak but refused a CT scan and rupture occured within 3 months.

Instability was found to be a significant predictor of 30-day mortality and was less frequently evidenced in patients with prior EVAR. From a theoretical point of view, the presence of the endograft may constitute a type of protection, reducing bleeding.

Obesity and hyperlipidemia were found to be significantly higher in the group without previous EVAR ($P = .036$, $P = .022$; Table II). The reason is unclear but could be associated with findings, which suggest that the incidental identification of an AAA in obese patients (most often affected by dislipidemia) is rendered more complex with the physical impediment for a palpatative examination or an echo duplex image.[34]

Our study is limited by the classification of patients based on blood pressure, which alone is not a sufficient definition of the gravity of a patient's condition. However, in the absence of a precise classification, we adopted the most commonly used criteria in other studies;[35,36] the blood pressure with a cut-off of 80 mm Hg. Further, the low incidence of rAAA after EVAR renders a statistical significance difficult to achieve, limiting in turn an effective conclusion to the study. This study has also made many comparisons with the EUROSTAR registry, which has been criticized for a high drop out to follow-up, a factor of particular importance to a study of rupture after EVAR.

In terms of mortality, it would be logical to expect a protection from the endograft in patients with previous EVAR. A trend seems to support this assumption but no statistical significance was found, which may be due to the inevitable small population size.

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AUTHOR CONTRIBUTIONS

Conception and design: GC, SG, GS, RS
Analysis and interpretation: GC, SG, GS, ST
Data collection: SG, GS
Writing the article: GC, SG, GS
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