Endovascular repair versus open surgery in patients with ruptured abdominal aortic aneurysms: Clinical outcomes with 1-year follow-up

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Objective: **To compare the clinical outcomes of treatment after endovascular repair and open surgery in patients with ruptured infrarenal abdominal aortic aneurysms (AAAs), including 1-year follow-up.**
Methods: **All consecutive conscious patients with ruptured infrarenal AAAs who presented to our tertiary care teaching hospital between January 1, 2001, and December 31, 2005, were included in this study (n = 55). Twenty-six patients underwent endovascular repair, and 29 patients underwent open surgery. Patients who were hemodynamically too unstable to undergo a computed tomography angiography scan were excluded. Outcomes evaluated were intraoperative mortality, 30-day mortality, systemic complications, complications necessitating surgical intervention, and mortality and complications during 1-year follow-up. The statistical tests we used were the Student *t* test, χ^2 test, Fisher exact test, and Mann-Whitney *U* test (two sided; $\alpha = .05$).**
Results: **Thirty-day mortality was 8 (31%) of 26 patients who underwent endovascular repair and 9 (31%) of 29 patients who underwent open surgery ($P = .98$). Systemic complications and complications necessitating surgical intervention during the initial hospital stay were similar in both treatment groups (8/26 [31%] and 5/26 [19%] for endovascular repair, respectively, and 9/29 [31%] and 8/29 [28%] for open surgery, respectively; $P > .40$). During 1-year follow-up, two patients initially treated with endovascular repair died as a result of non–aneurysm-related causes; no death occurred in the open surgery group. Complications during 1-year follow-up were 1 (5%) of 20 for endovascular repair and 4 (16%) of 25 for open surgery ($P = .36$).**
Conclusions: **On the basis of our study with a highly selected population, the mortality and complication rates after endovascular repair may be similar compared with those after open surgery in patients treated for ruptured infrarenal AAAs. (J Vasc Surg 2006;44:1148-55.)**

Mortality in patients with a ruptured abdominal aortic aneurysm (AAA) treated with open surgery remains high. Among patients who arrive in the hospital alive and undergo open surgery, the reported mortality rates vary between 32% and 70%, and the morbidity rates vary between 30% and 50%.[1,2] Since 1994, endovascular aneurysm repair in patients with a ruptured AAA has been proven to be feasible.[3] Recently, this technique has become routine practice in Europe, and it is increasingly performed in the United States. Several studies have demonstrated a reduction in mortality and morbidity rates with endovascular repair compared with conventional open surgery in patients with ruptured AAAs.[4-16] Most of these studies, however, included hemodynamically unstable patients in the open surgery group, whereas in the endovascular group mostly hemodynamically stable patients were included. To assess the clinical effectiveness of endovascular repair and open surgery in patients with a ruptured AAA, it is essential to compare both treatments in a homogeneous group of patients. Therefore, in the absence of a randomized controlled clinical trial, we compared endovascular repair and open surgery in patients who were hemodynamically stable enough to undergo a computed tomography angiography (CTA) scan before the procedure.

Several advantages and disadvantages of endovascular repair over open surgery exist. Important advantages of endovascular repair are potential avoidance of general anesthesia and minimization of invasiveness. During endovascular repair, the aorta is not clamped, and blood loss is considerably less than with open surgery. Patients treated with endovascular repair, however, are expected to have complications in the long run as a result of graft failure, such as endoleak and graft migration.[17-21] Therefore, follow-up after endovascular repair is essential. Patients treated with open

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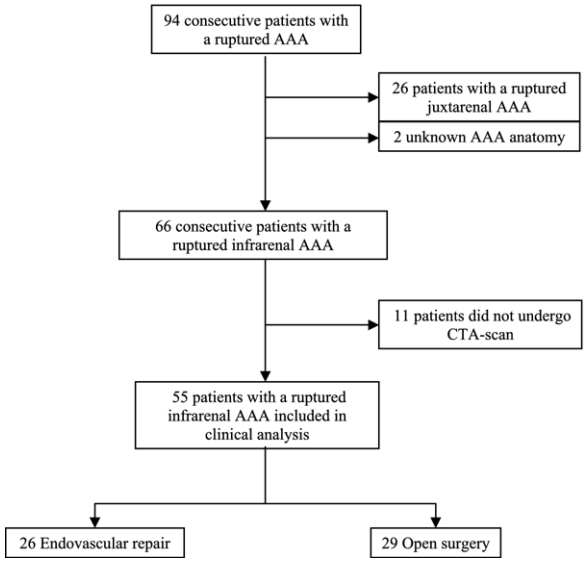

Fig. Flowchart of patients with a ruptured abdominal aortic aneurysm *(AAA)* included in the analysis. *CTA,* Computed tomography angiography.

surgery may have more severe complications during and immediately after the procedure, such as bleeding, cardiac and pulmonary complications, and ischemia of the sigmoid, whereas during follow-up complications are rare after open surgery. Thus, to evaluate the clinical effectiveness of both procedures, it is important to determine these complications both during the hospital stay and during follow-up.

The objective of our study was to compare the clinical outcomes of treatment after endovascular repair and open surgery in patients with ruptured infrarenal AAAs, including 1-year follow-up.

PATIENTS AND METHODS

Patients and protocol. All consecutive patients treated for a ruptured AAA in our university-based tertiary care center between January 1, 2001, and December 31, 2005, were evaluated (n = 94). The study period started in 2001, because January 2001 marked the initiation of endovascular repair of ruptured AAAs in our hospital. To make comparable groups of patients treated with endovascular repair and patients treated with open surgery, we excluded patients with a juxtarenal or suprarenal AAA (necessitating suprarenal clamping; n = 26), those with unknown AAA anatomy (n = 2), and those who were hemodynamically too unstable (ie, systolic blood pressure <70 mm Hg and no adequate verbal reply) and therefore were unable to undergo a CTA scan before the procedure (n = 11; Fig). Hemodynamically too unstable patients (n = 11) were immediately transported to the operating room, where they all underwent open surgery. In this study, 55 patients with ruptured infrarenal AAAs were included in the analysis.

The records of all patients with a ruptured AAA were identified from the medical registry by using operation codes. These codes are valid for our institution specifically. Patient data were obtained partly retrospectively and partly prospectively. Retrospectively data were retrieved from a computerized hospital database and, subsequently, from medical records. To obtain information about patients' readmissions, we verified patients' medical records and the computerized database of our hospital or of the hospital to which they were readmitted. From December 2004 onward, patients (n = 16) were prospectively enrolled in our study. Institutional review board approval was obtained, and informed consent to verify patient data was waived because we analyzed only patient data documented as part of routine clinical care and collected from the medical records. Note that in The Netherlands, patients' formal written informed consent is not obtained for good clinical practice. Use of new or emerging therapies can be applied after institutional review board approval of the hospital.

Once the emergency department was informed that a patient with a ruptured AAA was in transport to the hospital, a team consisting of a vascular surgeon, an interventional radiologist, and an anesthesiologist was waiting for the patient in the emergency room. This team was available 24 hours a day, 7 days a week. Upon the patient's arrival in the emergency room, an abdominal ultrasound scan was performed to confirm the diagnosis of an AAA. Furthermore, an electrocardiogram was performed, and laboratory findings were assessed to determine the patient's clinical condition. Hemodynamically stable patients were transported to the computed tomography (CT) suite to perform an abdominal CTA scan to assess whether the AAA was ruptured or not and to decide whether the AAA was suitable for endovascular repair. An infrarenal AAA was considered ruptured in the presence of leakage on the preoperative CTA. The anatomic inclusion criteria for endovascular repair were a proximal neck longer than 15 mm, neck diameter less than 28 mm, angulation <90°, and accessibility of the iliac arteries. After the CTA-confirmed rupture of the AAA, the patient was immediately transported to the operating room, where endovascular repair was performed if the AAA was suitable; otherwise, open surgery was performed. In our protocol, the decision to treat patients with endovascular repair or open surgery was not based on intraoperative aortography only. Hemodynamically too unstable patients (n = 11) were immediately transported to the operating room for open surgery and were excluded from this analysis. The median time interval between arrival in the emergency room and arrival in the operating room was 43 minutes (range, 15 minutes to 59 hours) for patients with a ruptured AAA. In total, in our analysis we included 55 patients with a ruptured infrarenal AAA. One-year follow-up was completed for 45 patients who underwent operation between January 1, 2001, and December 31, 2005.

In patients treated with endovascular repair, 1 Cook (Zenith, Bloomington, Ind) endograft and 24 Excluder (Gore, Flagstaff, Ariz) endografts were used. In patients treated with open surgery, Sulzer/Terumo (Vascutek, Renfrewshire, Scotland) vascular prostheses were used. In

our local setting, a large variety of endografts and vascular prostheses is on stock. Endovascular repair of a ruptured AAA was performed in the operating room by a vascular surgeon (four involved; on average, 4 years of experience in endovascular AAA repair and 20 years of experience in open AAA repair, as of 2001) and an interventional radiologist (two involved; on average, 4 years of experience in endovascular AAA repair, as of 2001). Patients with ruptured AAAs who underwent endovascular repair preferentially received local or regional anesthesia. According to our protocol, we accepted hypotension (ie, systolic blood pressure lower than 90 mm Hg) without massive fluid resuscitation (permissive hypotension) in order to prevent further bleeding. Patients treated with open surgery received general anesthesia. In patients treated with endovascular repair, a groin cutdown was performed to obtain access to the common femoral artery. A bifurcated endograft was preferentially used. If iliac occlusions were present on one side, an aortomonoiliac endograft was used. The first intraoperative angiography was performed only after the main body of the endograft was introduced. After completion of the endovascular procedure, control angiography was performed to exclude a type I or type III endoleak. A CTA was performed before discharge to assess complete exclusion of the AAA. If an endoleak was seen on the predischarge CT scan, a reintervention was scheduled, and the patient was treated within 1 month. The management of early endoleaks was similar after ruptured AAA repair and after intact AAA repair.

During follow-up, for patients who underwent endovascular repair, physician visits and CT scans were scheduled 3, 6, and 12 months after the initial procedure. For patients who underwent open surgery, physician visits were scheduled at 3 and 6 months, and at 6 months an ultrasound scan was performed.

Clinical outcomes. The outcomes evaluated were intraoperative mortality, 30-day mortality, systemic complications, complications necessitating surgical intervention, and mortality and complications during 1-year follow-up. Surgical interventions included tracheostomy, bowel resection, and surgical evacuation of an access-site hematoma or infection.

We defined relevant comorbidity factors for patients with a ruptured infrarenal AAA on the basis of published covariates in the literature.[22-25] Comorbidity was assessed by one author by using the patients' medical history (J.J.V.). Patients were stratified by using the Lee risk index.[26] This index was used to identify patients at higher risk for cardiac complications after the procedure. Patients were assigned to risk class II, III, or IV, depending on the presence of a set of risk factors. Patients in a higher risk class had a greater risk of cardiac complications after the procedure than those in a lower risk class. Diabetes mellitus was defined as receiving oral medication and/or insulin therapy for diabetes mellitus. Ischemic heart disease included angina pectoris and myocardial infarction in the medical history. Congestive heart failure included symptoms of congestive heart failure and receipt of medication for this diagnosis. History of a cerebrovascular event included stroke or transient ischemic attack. Hypertension included a systolic blood pressure of 140 mm Hg or more and/or a diastolic blood pressure of 90 mm Hg or more and receipt of at least one antihypertensive drug. Renal dysfunction included a creatine level greater than 2.0 mg/dL. Chronic pulmonary disease included a forced expiratory volume in 1 second less than 70%. Complications were identified by using the "Reporting Standards for Endovascular Aortic Aneurysm Repair" of Chaikof et al.[27]

Data analysis. Patient and lesion characteristics, procedure data, and clinical outcomes during hospital stay and 1-year follow-up of ruptured AAA patients who underwent endovascular repair and open surgery were compared by using the Student t test, χ^2 test, Fisher exact test, and Mann-Whitney U test (two sided; $\alpha = .05$). For data with a skewed distribution, we reported the median. Clinical results were calculated by taking all patients with a ruptured AAA (n = 55) into account, including patients who died. Data on mortality and morbidity were complete for all included patients. A few items related to the patient and procedure characteristics, however, were missing. In the tables, we identified the missing data; in total, less than 1% of the data were missed. Analyses were performed by using Microsoft Excel 2000 (Microsoft Corporation, Redmond, Wash) and SPSS for Windows version 11.0.1 (SPSS Inc, Chicago, Ill).

RESULTS

Patient characteristics. Patient and lesion characteristics are listed in Table I. The mean AAA diameter was similar for patients who underwent endovascular repair and for those who received open surgery (74 vs 76 mm [$P = .64$] for endovascular repair and open surgery, respectively). Reasons to treat patients with open surgery were neck too short (n = 10), too much mural thrombus neck (n = 1), neck angulation greater than 90° (n = 5), conical neck (n = 4), inaccessible iliac tract (n = 4), aneurysm iliac tract (n = 2), and logistic reasons (n = 3). Upon arrival in the emergency room, four patients had systolic blood pressures less than 90 mm Hg (although they were not hemodynamically unstable), of whom two were treated with open surgery and two with endovascular repair.

Procedure. Table II shows the procedure characteristics. Of all patients treated with endovascular repair, two patients were converted to open surgery during the procedure because of persistent blood loss, and one patient underwent a decompression laparotomy for an abdominal compartment syndrome. All three of these patients died. Of the patients with persistent blood loss, the patient with persistent blood loss due to profuse bleeding of the lumbar arteries died during the procedure (ie, the intraoperative mortality rate was 1 [4%] of 26 patients), and the patient with persistent blood loss due to a tear in the aortic wall near the proximal attachment died within 4 hours after the procedure. The patient with abdominal compartment syndrome died 13 days after the initial procedure as a result of sepsis. In addition to these three conversions, one failure

Table I. Patient and lesion characteristics in patients with a ruptured abdominal aortic aneurysm (n = 55)

Variable	*Endovascular repair (n = 26)*	*Open surgery (n = 29)*	P *value*
Mean age, y (SD)	72.5 (8.4)	73.9 (7.9)	.53
Male sex	25 (96%)	28 (97%)	.51
Mean AAA diameter, mm (SD)	74 (13)	76 (16)	.61
Diabetes mellitus	1 (4%)	0 (0%)	.47
Ischemic heart disease*	7 (27%)	6 (21%)	.64
Congestive heart failure*	2 (8%)	1 (4%)	.37
History of CVA*	4 (15%)	2 (7%)	.22
Hypertension*	13 (50%)	12 (43%)	.60
Renal dysfunction*	5 (19%)	2 (7%)	.14
Chronic pulmonary disease*	7 (27%)	5 (18%)	.42
Systolic blood pressure <90 mm Hg†	2 (8%)	2 (7%)	.39
Medication			
Beta blockers‡	6 (25%)	8 (29%)	.77
Statins§	4 (16%)	0 (0%)	.04
Antiplatelet agents§	4 (16%)	9 (32%)	.17
ACE inhibitors§	5 (20%)	4 (14%)	.25
Calcium-channel blockers§	0 (0%)	2 (7%)	.27
Anticoagulants§	5 (20%)	3 (11%)	.20

AAA, Abdominal aortic aneurysm; *CVA,* cerebrovascular accident; *ACE,* angiotensin-converting enzyme.
*These data were missing for one patient who underwent open surgery.
†Upon arrival in the emergency room.
‡No information on the use of β-blockers was available for two patients who underwent endovascular repair and for one who underwent open surgery.
§These data were missing for one patient who underwent endovascular repair and for one who underwent open surgery.

Table II. Procedure data in patients with a ruptured abdominal aortic aneurysm

Variable	*Endovascular repair (n = 26)*	*Open surgery (n = 29)*	P *value*
General anesthesia	12 (46%)	29 (100%)	<.001
Tubular graft	1 (4%)*	19 (66%)*	<.001
Bifurcated graft	24 (92%)*	9 (31%)*	<.001
Conversion to open surgery during initial procedure	3 (12%)	—	—
Technical failure during initial procedure	4 (15%)	—	—
Additional procedures during initial procedure	0 (0%)	5 (17%)	.04
Intraoperative mortality	1 (4%)	4 (14%)	.18
Median blood loss, mL (range)	<100 (<100-30,000)	6750 (<100-31,000)	<.001
Median transfusion units (EC; range)	1.5 (0-27)	10 (0-31)	<.001
Median transfusion units (FFP; range)	0 (0-20)	10 (0-45)	<.001
Median transfusion units (platelets; range)	0 (0-15)	5 (0-20)	<.001
Median procedure time (min; range)	149 (79-400)	232 (40-434)	<.001

EC, Erythrocyte concentrate; *FFP,* fresh frozen plasma.
*In one patient who underwent endovascular repair, no access was obtained because of calcified iliac arteries. This patient refused conversion to open surgery and subsequently died; one patient who underwent open surgery died before graft placement.

occurred because no access was obtained as a result of calcified iliac arteries. Conversion to open surgery was offered to this patient, but she refused open surgery and subsequently died. One patient initially treated with endovascular repair underwent a decompression laparotomy 7 hours after the initial procedure because of a suspected abdominal compartment syndrome. This patient died 4 days after the initial procedure as a result of respiratory insufficiency. No additional procedure was performed during endovascular repair.

During open surgery, 4 (14%) of 29 patients died as a result of persisting blood loss (coagulopathy leading to continued bleeding [n = 1], nonidentifiable venous bleeding [n = 1], diffuse bleeding after admitting heparin and local thrombolysis with medicinase [n = 1], and bleeding leading to no cardiac output [n = 1]). Additional procedures were performed in five patients during open surgery: resection of the sigmoid (n = 2), thrombectomy of the superficial femoral artery (n = 1), local endarterectomy of the common femoral artery (n = 1), and treatment of a scrotal hernia (n = 1). The median blood loss was lower with endovascular repair compared with that with open surgery (<100 vs 6750 mL; $P < .001$).

Outcomes. In total, 8 (31%) of 26 patients and 9 (31%) of 29 patients treated with endovascular repair and open surgery, respectively, died within 30 days after the initial procedure ($P = .98$; Table III). In addition to the patient who died during endovascular repair, seven patients died within 30 days. The causes of postoperative death were persistent blood loss (ie, due to a tear in the

Table III. Mortality, complications, and admissions in patients with a ruptured abdominal aortic aneurysm

Variable	*Endovascular repair (n = 26)*	*Open surgery (n = 29)*	P *value*
Intraoperative mortality	1 (4%)	4 (14%)	.18
30-d mortality	8 (31%)	9 (31%)	.98
Complications necessitating surgical intervention*	5 (19%)	8 (28%)	.47
Systemic complications†	8 (31%)	9 (31%)	.98
Access-site hematoma‡	2 (8%)	3 (10%)	.34
Access-site infection§	0 (0%)	3 (10%)	.14
Cardiac complications‖	3 (12%)	1 (3%)	.22
Pulmonary complications¶	2 (8%)	2 (7%)	.39
Pulmonary embolism#	2 (8%)	1 (3%)	.36
Renal complications**	0 (0%)	1 (3%)	.53
Sepsis	3 (12%)	3 (10%)	.33
Deep venous thrombosis#	0 (0%)	2 (7%)	.27
Bowel ischemia††	1 (4%)	2 (7%)	.40
Endoleak	5 (19%)‡‡	—	—
Postoperative days in the ICU: mean (SD), median	4.3 (8.6), 0.9	11.7 (15.0), 5.5	.01
Postoperative days in hospital: mean (SD), median	10.9 (17.4), 4.0	26.7 (28.3), 15.3	.003

ICU, Intensive care unit.

*Complications necessitating surgical intervention included surgical evacuation of an access-site hematoma or infection, tracheostomy, and bowel resection.

†Systemic complications included cardiac arrest, progressive heart failure with fatal outcome, pulmonary complications necessitating tracheostomy or with fatal outcome, pulmonary embolism necessitating anticoagulation therapy or with fatal outcome, renal complications necessitating temporary dialysis, sepsis, and deep venous thrombosis.

‡Necessitating surgical evacuation.

§Necessitating operative drainage.

‖Cardiac arrest and progressive heart failure with fatal outcome.

¶Necessitating tracheostomy or pulmonary complications with fatal outcome.

#Necessitating anticoagulation therapy or with fatal outcome.

**Necessitating temporary dialysis.

††Necessitating bowel resection.

‡‡One patient had a type I endoleak and was treated within 1 month after the initial procedure. One patient had a type III endoleak and was treated 2 months after the initial procedure. Three patients had type II endoleaks. They were treated conservatively, and their endoleaks disappeared spontaneously. None of these three patients was treated for endoleaks during 1-year follow-up.

aortic wall near the proximal attachment; n = 1), sepsis (n = 1), respiratory insufficiency (n = 1), pulmonary embolism (n = 1), ventricular fibrillation (n = 1), progressive heart failure (n = 1), and technical failure of the endovascular procedure with refusal of conversion to open surgery, leading to death (n = 1). During the hospital stay, one patient who underwent endovascular repair died 35 days after the initial procedure as a result of sepsis. In addition to the four patients who died during open surgery, five patients died within 30 days. The causes of postoperative death were septic shock (n = 2), renal insufficiency (n = 1), respiratory insufficiency (n = 1), and progressive heart failure (n = 1). During the hospital stay, one patient who underwent open surgery died 109 days after the initial procedure as a result of the inability to wean the patient from mechanical ventilation.

Systemic complications and complications necessitating surgical intervention were not different between treatment groups. In patients treated with endovascular repair, one patient required tracheotomy for respiratory failure. In patients treated with open surgery, two patients required tracheotomy for respiratory failure. Table III shows postoperative complications in more detail. The mean number of postoperative days in the intensive care unit was 4.3 and 11.7 for endovascular repair and open surgery, respectively ($P = .01$). In total, the mean number of postoperative days in the hospital was 10.9 for endovascular repair and 26.7 for open surgery ($P = .003$; Table III). In the endovascular repair group, one patient was discharged to a nursing hospital. In the open surgery group, three patients were discharged to a nursing hospital.

In Table IV we stratified 30-day mortality and systemic complications by the Lee risk index. For patients in Lee risk class II (ie, those at lower risk for cardiac complications), 30-day mortality and systemic complication rates were higher for endovascular repair compared with open surgery, although this was not statistically significant. For patients in Lee risk class III and IV (ie, those at a higher risk for cardiac complications), 30-day mortality and systemic complication rates were lower for endovascular repair compared with open surgery (Table IV).

One-year follow-up. One-year follow-up was completed for 45 (82%) of 55 patients (Table V). During follow-up, two patients who were initially treated with endovascular repair died as a result of non–aneurysm-related causes (ie, pulmonary infection and cancer). One patient initially treated with endovascular repair and two patients initially treated with open surgery were readmitted to the hospital because of aneurysm-related complications. The patient treated with endovascular repair was readmitted for endovascular repair of a type I endoleak that was detected during the initial hospital stay. The patients treated with open surgery had aneurysm repair

Table IV. In-hospital systemic complications and 30-day mortality in patients with a ruptured abdominal aortic aneurysm stratified by the Lee risk index

Endovascular repair			*Open surgery*		
Lee risk index	*30-d mortality*	*Systemic complications**	*Lee risk index*	*30-d mortality*	*Systemic complications**
Class II (n = 13)	4 (31%)	5 (38%)	Class II (n = 19)	4 (21%)	4 (21%)
Class III (n = 8)	3 (38%)	2 (25%)	Class III (n = 7)	3 (43%)	3 (43%)
Class IV (n = 5)	1 (20%)	1 (20%)	Class IV (n = 2)	1 (50%)	1 (50%)
Missing (n = 0)	0	0	Missing (n = 1)	1	1

*Systemic complications included cardiac arrest, progressive heart failure with fatal outcome, pulmonary complications necessitating tracheostomy, pulmonary embolism necessitating anticoagulation therapy, renal complications necessitating temporary dialysis, sepsis, and deep venous thrombosis.

Table V. One-year follow-up of patients with a ruptured abdominal aortic aneurysm

Variable	*Endovascular repair (n = 20)*	*Open surgery (n = 25)*	P *value*
Patients at risk	14	16	—
Median number of visits (range)	2.0 (0-6)	2.0 (0-6)	.66
Median number of CTAs (range)	1.5 (0-3)	0.0 (0-1)	<.001
Newly diagnosed endoleaks*	2 (10%)	—	—
Complications†	1 (5%)	4 (16%)	.21
Readmissions	1 (5%)‡	2 (8%)	.42
Died during follow-up	2 (10%)§	0 (0%)	.19
Total deaths at 1-y follow-up	8 (40%)	9 (36%)	.78

CTA, Computed tomography angiography.
*Type II endoleaks diagnosed at 4 months; they were treated conservatively.
†After endovascular repair: a false aneurysm at 3 months, treated conservatively, n = 1. After open surgery: aneurysm of the femoral artery at 4 months, readmitted for aneurysm repair, n = 1; scar hernia at 6 months, treated conservatively, n = 1; abdominal hernia at 7 months, treated conservatively, n = 1; back pain suggestive of aneurysm-related problems at 8 months, readmitted, treated conservatively, n = 1.
‡This patient was readmitted for repair of a type I endoleak that was detected during the initial hospital stay.
§Non–aneurysm-related death.

of the femoral artery and back pain suggestive of aneurysm-related problems.

Juxtarenal AAA and hemodynamically unstable patients. For patients with juxtarenal AAAs, the intraoperative mortality was 4 (15%) of 26, and the 30-day mortality was 9 (35%) of 26. One-year follow-up was completed for 25 patients, of whom 11 (44%) died within 1 year. For patients who were hemodynamically too unstable to undergo a CT scan before the procedure, the intraoperative mortality was 1 (9%) of 11, and the 30-day mortality was 4 (36%) of 11. One-year follow-up was completed for 10 patients, of whom 5 (50%) died within 1 year.

DISCUSSION

Endovascular repair has become an increasingly performed alternative to open surgery in patients with ruptured infrarenal AAAs. In our study, we compared the clinical outcomes of patients treated with endovascular repair vs those of patients treated with open surgery. In the absence of randomization, we focused our comparison on hemodynamically stable patients to make the treatment groups more homogeneous and the comparison more adequate. In our treatment groups, the selection between endovascular repair and open surgery was based on anatomic criteria. Patients who were eligible (ie, had suitable anatomy) for endovascular repair received this treatment; patients whose aneurysm anatomy was not suitable for endovascular repair received open surgery. Note that, in our hospital, vascular surgeons and interventional radiologists are available 24 hours a day, 7 days a week, and that endografts are always on stock. The principal finding of our study was that 30-day mortality and morbidity were similar for endovascular repair and open surgery in patients with a ruptured AAA; this similarity was sustained after 1-year follow-up.

Results from the stratification suggested that patients with a lower risk for cardiac complications (ie, class II) had a better chance to survive and less morbidity after open surgery than after endovascular repair. In addition, patients with a higher risk for cardiac complications (ie, class III and IV) were better off with endovascular repair than with open surgery. It should be acknowledged that 50% of the patients treated with endovascular repair and 32% of the patients treated with open surgery were considered at high risk for cardiac complications (class III and IV). Therefore, it seems that selection criteria other than aneurysm anatomy may have played a role in the treatment choice. It should be noted, however, that these thoughts are highly speculative because of the low number of patients and the highly selected population.

So far, to our knowledge, no randomized controlled trial comparing endovascular repair and open surgery in patients with ruptured AAAs has been published. In other studies comparing the outcomes of endovascular repair and open surgery in patients with a ruptured AAA, results showed lower mortality for endovascular repair than was found in our study.[5-16] Most of these studies, however, did not report on patients' comorbidity. Therefore, it remains unclear whether this difference in mortality was due to differences in case mix. Furthermore, most other studies reported higher mortality rates for patients treated with open surgery compared with what we found. These studies included hemodynamically unstable patients who underwent open surgery and were not eligible for endovascular

repair, whereas we excluded those patients. A recently published study in which the proportion of hemodynamically unstable patients was equal in both treatment groups showed similar results compared with our study.[28] In addition, follow-up results reported in most other studies demonstrated more complications after endovascular repair than after open surgery because of graft-related problems such as graft migration and endoleak, whereas in our study, the complication rates in follow-up were similar for endovascular repair and open surgery.[17-21]

The results of our study should be interpreted with caution because of several limitations. The patients in our study were not randomly assigned to endovascular repair and open surgery, the sample sizes of both treatment groups were small, and follow-up was limited to 1 year. Despite these limitations, however, our study was a first attempt for a fair comparison in a time when endovascular repair is increasingly performed and data on effectiveness are needed. It is clear that more research comparing endovascular repair with open surgery in patients with ruptured AAAs in larger series with longer follow-up is needed, as suggested in the long-term effectiveness of the trials in elective AAAs.[29,30] As far as we know, one randomized controlled trial is ongoing,[31] but it may take years before results are available. Because endovascular repair is increasingly performed, data from solid research are needed.

In addition, more research focusing on selection criteria and survival chances needs to be performed. As in our study, among patients initially treated with endovascular repair, two were converted to open surgery, and two underwent a decompression laparotomy. All of these patients died. Whether the patients' comorbidities, AAA anatomy, or the delay caused by first performing endovascular repair instead of open surgery played a role in their survival chances remains unknown. These findings, however, do emphasize the need for a predictive tool that can identify patients who may benefit from endovascular repair and those who are unlikely to benefit from this procedure.

In conclusion, on the basis of our study with a highly selected population, mortality and morbidity may be similar for patients with ruptured infrarenal AAAs treated with endovascular repair compared with open surgery, even after 1-year follow-up. In addition to the aneurysm anatomy, other criteria may be needed for endovascular repair to improve clinical outcomes. To obtain more evidence regarding whether endovascular repair or open surgery is better in selected patients with a ruptured infrarenal AAA, more research is needed.

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AUTHOR CONTRIBUTIONS

Conception and design: JJV, JLB, MRHMvS
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Overall responsibility: MRHMvS

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REFERENCES

1. Bown MJ, Sutton AJ, Bell PR, Sayers RD. A meta-analysis of 50 years of ruptured abdominal aortic aneurysm repair. Br J Surg 2002;89:714-30.
2. Bown MJ, Cooper NJ, Sutton AJ, Prytherch D, Nicholson ML, Bell PR, et al. The post-operative mortality of ruptured abdominal aortic aneurysm repair. Eur J Vasc Endovasc Surg 2004;27:65-74.
3. Yusuf SW, Whitaker SC, Chuter TA, Wenham PW, Hopkinson BR. Emergency endovascular repair of leaking aortic aneurysm. Lancet 1994;344:1645.
4. Greco G, Egorova N, Anderson PL, Gelijns A, Moskowitz A, Nowygrod R, et al. Outcomes of endovascular treatment of ruptured abdominal aortic aneurysms. J Vasc Surg 2006;43:453-9.
5. 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;31:345-50.
6. Vaddineni SK, Russo GC, Patterson MA, Taylor SM, Jordan WD Jr. Ruptured abdominal aortic aneurysm: a retrospective assessment of open versus endovascular repair. Ann Vasc Surg 2005;19:782-6.
7. 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.
8. Kapma MR, Verhoeven EL, Tielliu IF, Zeebregts CJ, Prins TR, Van der Heij B, et al. Endovascular treatment of acute abdominal aortic aneurysm with a bifurcated stentgraft. Eur J Vasc Endovasc Surg 2005;29:510-5.
9. Hechelhammer L, Lachat ML, Wildermuth S, Bettex D, Mayer D, Pfammatter T. Midterm outcome of endovascular repair of ruptured abdominal aortic aneurysms. J Vasc Surg 2005;41:752-7.
10. 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.
11. Lee WA, Hirneise CM, Tayyarah M, Huber TS, Seeger JM. Impact of endovascular repair on early outcomes of ruptured abdominal aortic aneurysms. J Vasc Surg 2004;40:211-5.
12. Resch T, Malina M, Lindblad B, Dias NV, Sonesson B, Ivancev K. Endovascular repair of ruptured abdominal aortic aneurysms: logistics and short-term results. J Endovasc Ther 2003;10:440-6.
13. Reichart M, Geelkerken RH, Huisman AB, van Det RJ, de Smit P, Volker EP. Ruptured abdominal aortic aneurysm: endovascular repair is feasible in 40% of patients. Eur J Vasc Endovasc Surg 2003;26:479-86.
14. Peppelenbosch N, Yilmaz N, van Marrewijk C, Buth J, Cuypers P, Duijm L, et al. Emergency treatment of acute symptomatic or ruptured abdominal aortic aneurysm. Outcome of a prospective intent-to-treat by EVAR protocol. Eur J Vasc Endovasc Surg 2003;26:303-10.
15. Castelli P, Caronno R, Piffaretti G, Tozzi M, Lagana D, Carrafiello G, et al. Ruptured abdominal aortic aneurysm: endovascular treatment. Abdom Imaging 2005;30:263-9.
16. Brandt M, Wallusch eck KP, Jahnke T, Graw K, Cremer J, Muller-Hulsbeck S. Endovascular repair of ruptured abdominal aortic aneurysm: feasibility and impact on early outcome. J Vasc Interv Radiol 2005;16:1309-12.

17. Tolia AJ, Landis R, Lamparello P, Rosen R, Macari M. Type II endoleaks after endovascular repair of abdominal aortic aneurysms: natural history. Radiology 2005;235:683-6.
18. Prinssen M, Wixon CL, Buskens E, Blankensteijn JD. Surveillance after endovascular aneurysm repair: diagnostics, complications, and associated costs. Ann Vasc Surg 2004;18:421-7.
19. Verhagen HJ, Prinssen M, Milner R, Blankensteijn JD. Endoleak after endovascular repair of ruptured abdominal aortic aneurysm: is it a problem? J Endovasc Ther 2003;10:766-71.
20. van Marrewijk C, Buth J, Harris PL, Norgren L, Nevelsteen A, Wyatt MG. Significance of endoleaks after endovascular repair of abdominal aortic aneurysms: the EUROSTAR experience. J Vasc Surg 2002;35:461-73.
21. Laheij RJ, Buth J, Harris PL, Moll FL, Stelter WJ, Verhoeven EL. Need for secondary interventions after endovascular repair of abdominal aortic aneurysms. Intermediate-term follow-up results of a European collaborative registry (EUROSTAR). Br J Surg 2000;87:1666-73.
22. Kertai MD, Boersma E, Klein J, van Urk H, Poldermans D. Optimizing the prediction of perioperative mortality in vascular surgery by using a customized probability model. Arch Intern Med 2005;165:898-904.
23. Kertai MD, Steyerberg EW, Boersma E, Bax JJ, Vergouwe Y, van Urk H, et al. Validation of two risk models for perioperative mortality in patients undergoing elective abdominal aortic aneurysm surgery. Vasc Endovascular Surg 2003;37:13-21.
24. Shuhaiber JH, Hankins M, Robless P, Whitehead SM. Comparison of POSSUM with P-POSSUM for prediction of mortality in infrarenal abdominal aortic aneurysm repair. Ann Vasc Surg 2002;16:736-41.
25. Steyerberg EW, Kievit J, de Mol Van Otterloo JC, van Bockel JH, Eijkemans MJ, Habbema JD. Perioperative mortality of elective abdominal aortic aneurysm surgery. A clinical prediction rule based on literature and individual patient data. Arch Intern Med 1995;155:1998-2004.
26. Lee TH, Marcantonio ER, Mangione CM, Thomas EJ, Polanczyk CA, Cook EF, et al. Derivation and prospective validation of a simple index for prediction of cardiac risk of major noncardiac surgery. Circulation 1999;100:1043-9.
27. Chaikof EL, Blankensteijn JD, Harris PL, White GH, Zarins CK, Bernhard VM, et al. Reporting standards for endovascular aortic aneurysm repair. J Vasc Surg 2002;35:1048-60.
28. Peppelenbosch N, Geelkerken RH, Soong C, Cao P, Steinmetz OK, Teijink JA, et al. Endograft treatment of ruptured abdominal aortic aneurysms using the Talent aortouniiliac system: an international multicenter study. J Vasc Surg 2006;43:1111-23.
29. Blankensteijn JD, de Jong SE, Prinssen M, van der Ham AC, Buth J, van Sterkenburg SM, et al. Two-year outcomes after conventional or endovascular repair of abdominal aortic aneurysms. N Engl J Med 2005;352:2398-405.
30. Endovascular aneurysm repair versus open repair in patients with abdominal aortic aneurysm (EVAR trial 1): randomised controlled trial. Lancet 2005;365:2179-86.
31. Acute endovascular treatment to improve outcome of ruptured aortoiliac aneurysms. Available at: URL: http://www.trialregister.nl/trialreg/admin/retview.asp?TC=85. Accessed June 14, 2006.

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