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Predictive Factor for Mortality and Morbidity of Abdominal Aortic Aneurysm Repair

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Abstract

Purpose: This study aimed to determine the procedure-related morbidity and mortality of infrarenal abdominal aortic aneurysm (AAA) repair, to investigate the changes in perioperative laboratory values, and to clarify the specific independent predictive factors for mortality and morbidity. We retrospectively evaluated patients who were treated in Jichi Medical University Hospital.
Methods: Consecutive patients with AAA between April 2007 and August 2010 were studied. The possible predictive values of various patient- and operation-related variables on outcomes (mortality, duration of stay in hospital (>7 days), and major morbidity) were assessed by multivariate analysis.
Results: Overall in-hospital mortality was 3.3%. Statistically significant differences, all in favor of endovascular aneurysm repair (EVAR), were observed in the intraoperative and post-operative data. In multivariate logistic analysis, potassium, serum creatinine and C-reactive protein levels were significantly related to outcomes.
Conclusions: Open repair and EVAR can both be safely performed in patients treated for elective and emergency infrarenal AAA. EVAR has perioperative advantages of reduced blood loss and blood transfusion, as well as decreased mortality and duration of post-operative hospital stay. In particular, we identified specific independent predictive factors of serum chemistry values for mortality and renal insufficiency.

Key words: *Abdominal aortic aneurysm, endovascular aneurysm repair, open repair, predictive factor, renal insufficiency*

Introduction

An abdominal aortic aneurysm (AAA) is defined as an enlargement of the aortic diameter with at least 150% of the diameter at the orifice of the renal arteries [1]. Elective treatment is recommended when the AAA size reaches 55 mm in diameter because of higher rates of rupture. Open repair of an AAA has always been considered among the most major of surgical procedures, and the potential complications are highly morbid. However, operative management has improved, and advances in critical care have reduced operative morbidity and mortality. Endovascular aneurysm repair (EVAR) is performed via limited access and incisions in femoral arteries and it reduces the need for blood transfusions. It has repeatedly demonstrated decreased perioperative complications, hospital stay and mortality, compared to open repair. In particular, the usefulness of EVAR for patients with chronic renal insufficiency has been reported from some institutions [2,3].

During the last 4 years, our institution has offered EVAR and open repair to patients undergoing elective and emergency treatment of their infrarenal AAA. This study reviewed the procedure-related morbidity and mortality of EVAR and open surgery, and investigated the changes in perioperative laboratory values in the different procedures. The main objective of this study was to clarify the specific independent predictive factors for mortality and morbidity, to ultimately be used for prognostic improvement.

Materials and Methods

We reviewed the data of patients who underwent AAA repair between April 2007 and August 2010. Repair of infrarenal AAA was performed in 307 consecutive patients. The type of repair was decided by the primary vascular surgeon and patient preference. Patients who had emergency repair of symptomatic, ruptured AAAs, false aneurysms, and iliac artery aneurysms were included. All treatments were performed in an operating room under general anesthesia. This study was approved by the institutional review board. To determine the effect of the selection of treatment, three groups were classified according to the type of repair (elective open repair, emergency open repair and EVAR). Emergent EVAR was not performed. Devices used for open AAA repair were a Dacron prosthesis (Hemashield; Meadox Medicals, Oakland, NJ), and for endovascular treatment we used an Excluder (W.L. Gore, Flagstaff, AR) and a Zenith (Cook, Bloomington, IN). The duration of the operation, intraoperative blood loss (the amount of suctioned blood by Cellsaver was excepted from intra-operative blood loss), blood transfusion volume and postoperative course were recorded. Intraoperative and perioperative outcomes were assessed. They included perioperative mortality, post-operative hospital stay, and major morbidity (bleeding, stenosis or occlusion of the graft, myocardial infarction, stroke, congestive heart failure, renal insufficiency, bowel obstruction, paralysis and endoleak). Endoleak was classified as types I through IV, according to standard definitions. Follow-up included clinical examination and laboratory studies, including measurement of white blood cells (WBC), C-reactive protein (CRP), lactate dehydrogenase, creatine phosphokinase (CPK), potassium (K^+), aspartate aminotransferase (AST), alanine aminotransferase (ALT), and preoperative and postoperative serum creatinine (SCr) levels. In the initial postoperative period, laboratory data were measured on postoperative days 1 and 2. Patients with worsening data were observed until they improved, and their data were recorded. The laboratory values recorded in the postoperative course were the worst ones. All patients were followed up closely in the early postoperative period (at least 30 days) for postoperative complications assessment. All patients with the difference between pre-operative SCr and post-operative SCr values greater than 1.0 mg/dL were considered to have post-operative renal insufficiency. The possible predictive value and operation-related variables of the three groups (elective open repair, emergency open repair and EVAR) were assessed. Between-group differences in categorical variables were tested by the chi-square test and the unpaired Student's t-test for continuous variables. Data are expressed as the mean value ± standard deviation (SD) or as frequencies and percentages. A value of probability (P) < 0.05 was considered statistically significant. All of these potential variables were assessed in univariate analyses. Variables that had a direct effect after univariate analysis were entered in a multivariate logistic regression model. The odds ratio (OR) and 95% confidence intervals (CI) were also calculated. Statistical analysis was performed using StatView 5.0 (SAS Institute, Cary, NC) software.

Results

Of the 251 patients who underwent elective infrarenal AAA repair, 151 had conventional open repair and 100 had EVAR. Fifty-six patients underwent emergency open infrarenal AAA repair. Demographic characteristics of the patients are listed in Table 1. The mean diameter was significantly larger in the emergency open repair group, compared to that in the elective open repair group ($P < 0.05$) and EVAR group ($P < 0.001$). No significant age or sex differences were found in our patient population. In the emergency open repair group, according to Fitzgerald's classification, type 1 accounts for 18 cases (32.1%), type 2 for 11 (19.6%), type 3 for 26 (46.4%), and type 4 for 1 (1.8%).

Overall in-hospital mortality was 3.3%. The in-hospital mortality was 4.8% for the open repair groups (0.7% for the elective open repair group and 16.1% for the emergency open repair group) and 0% for the EVAR group. One patient died in the elective open repair group; the death was related to multiple organ failure. Nine patients died in the emergency open repair group; the death was cardiac-related in three patients, caused by multiple organ failure in five patients and by pneumonia in one patient.

A comparison of the intraoperative details is shown in Table 2. The duration of the operation was shorter, and blood loss and blood transfusion were significantly less in the EVAR group, compared to the elective and emergency open repair groups. In the emergency open repair group, the blood loss is less than the blood transfusion. This is why a massive blood transfusion was necessary to be treated according to the patient's hemo-

Table 1. Demographic characteristics of the patients.

	ELOR	EMOR	EVAR	P value		
				ELOR vs EMOR	ELOR vs EVAR	EMOR vs EVAR
Age	71.4 (7.8)	72.7 (10.1)	73.3 (8.4)	*NS (0.386)	*NS (0.068)	*NS (0.707)
Male	134 (88.7%)	49 (87.5%)	86 (86%)	**NS (0.804)	**NS (0.518)	**NS (0.792)
Aneurysm diameter	57.3 (12.8)	63.6 (17.7)	54.9 (13.2)	*<0.05	*NS (0.152)	*<0.001

Table 2. Comparison of intraoperative details by the type of repair.

	ELOR	EMOR	EVAR	*P value		
				ELOR vs EMOR	ELOR vs EVAR	EMOR vs EVAR
Duration of operation (min)	253.3 (72.0)	244.2 (77.0)	166.4 (8.4)	0.429	<0.001	<0.001
Blood loss (ml)	508.0 (740.1)	1422.2 (1770.9)	129.4 (119.9)	<0.001	<0.001	<0.001
Blood transfusion (ml)	211.9 (539.7)	2192.1 (2037.9)	13.6 (82.0)	<0.001	<0.001	<0.001

Table 3. Comparison of post-operative details by the type of repair.

	ELOR	EMOR	EVAR	*P value		
				ELOR vs EMOR	ELOR vs EVAR	EMOR vs EVAR
Post-operative hospital stay (days)	12.1 (4.7)	25.4 (17.6)	5.8 (3.8)	<0.001	<0.001	<0.001
WBC	12148.3 (3747.8)	15109.1 (6720.4)	9856 (2611.0)	<0.01	<0.001	<0.001
CRP	20.4 (6.4)	22.2 (8.0)	8.9 (5.9)	NS (0.133)	<0.001	<0.001
LDH	285.7 (102.5)	756.8 (1205.6)	259.2 (154.4)	<0.01	NS (0.133)	<0.01
CPK	829.0 (745.7)	4548.0 (13660.1)	169.9 (218.5)	<0.05	<0.001	<0.05
K	4.4 (0.4)	4.8 (0.9)	4.3 (0.4)	<0.01	NS (0.108)	<0.001
AST	51.3 (62.0)	390.5 (1253.6)	36.8 (51.2)	<0.05	<0.05	<0.05
ALT	34.9 (31.1)	154.5 (359.9)	23.0 (20.6)	<0.05	<0.001	<0.01
Pre-SCr	1.1 (0.9)	1.4 (0.9)	1.2 (1.4)	<0.05	NS (0.413)	NS (0.316)
Post-SCr	1.3 (1.1)	2.0 (2.0)	1.3 (1.3)	<0.05	NS (0.802)	<0.05
(Post-SCr)-(Pre-SCr)	0.3 (0.4)	0.5 (1.6)	0.1 (0.3)	NS (0.178)	<0.001	<0.05

Values are mean (standard deviation or percentages). *Unpaired Student's t-test. **Chi-Square test. EVAR: Endovascular aneurysm repair. ELOR: Elective open repair. EMOR: Emergency open repair. WBC: White blood cells, CRP: C-reactive protein, LDH: Lactate dehydrogenase, CPK: Creatine phosphokinase, K: Potassium, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, Pre-SCr/Post-SCr: Pre-operative and post-operative serum creatinine.

Table 4. Univariate predictors of outcomes among patients undergoing AAA repair.

Outcome measure	Prognostic factor	Odds ratio	*P value
Mortality	Emergency open repair	0.021 (0.003-0.169)	<0.0005
	Blood loss	0.999 (0.999-1.000)	<0.0005
	Blood transfusion	0.999 (0.999-1.000)	<0.0001
	Renal insufficiency	0.133 (0.031-0.563)	<0.01
	WBC	1.000 (1.000-1.000)	<0.01
	LDH	0.999 (0.998-1.000)	<0.005
	K	0.203 (0.079-0.520)	<0.001
	AST	0.999 (0.998-1.000)	<0.05
	ALT	0.997 (0.995-0.999)	<0.001
	Aneurysmal diameter	0.947 (0.912-0.983)	<0.005
	Pre-SCr	0.727 (0.550-0.960)	<0.05
	Post-SCr	0.687 (0.542-0.870)	<0.005
Duration of stay in hospital (>7 days)	Open repair	156.235 (59.478-410.394)	<0.0001
	Emergency open repair	0.039 (0.005-0.289)	<0.005
	Operation time	0.972 (0.965-0.979)	<0.0001
	Blood loss	0.996 (0.995-0.998)	<0.0001
	Blood transfusion	0.998 (0.996-0.999)	<0.005
	WBC	1.000 (1.000-1.000)	<0.0001
	CRP	0.790 (0.748-0.834)	<0.0001
	LDH	0.995 (0.993-0.998)	<0.005
	CPK	0.993 (0.991-0.995)	<0.0001
	K	0.447 (0.251-0.798)	<0.01
	AST	0.987 (0.977-0.997)	<0.01
	ALT	0.970 (0.956-0.985)	<0.0001
	Aneurysmal diameter	0.981 (0.963-1.000)	<0.05
	Post-pre SCr	0.149 (0.055-0.404)	<0.0005
Major morbidity	Emergency open repair	0.421 (0.221-0.804)	<0.01
	Operation time	0.997 (0.993-1.000)	<0.05
	Blood loss	1.000 (0.999-1.000)	<0.05
	Blood transfusion	1.000 (0.999-1.000)	<0.005
	WBC	1.000 (1.000-1.000)	<0.001
	CRP	0.954 (0.923-0.986)	<0.005
	LDH	0.999 (0.998-1.000)	<0.05
	Pre-SCr	0.761 (0.598-0.970)	<0.05
	Post-SCr	0.583 (0.453-0.750)	<0.0001
	Post-pre SCr	0.103 (0.044-0.237)	<0.0001
Renal insufficiency	Emergency open repair	0.171 (0.066-0.444)	<0.0005
	Blood loss	1.000 (0.999-1.000)	<0.001
	Blood transfusion	1.000 (0.999-1.000)	<0.0005
	WBC	1.000 (1.000-1.000)	<0.0001
	CRP	0.857 (0.794-0.925)	<0.0001
	LDH	0.999 (0.998-1.000)	<0.005
	CPK	1.000 (1.000-1.000)	<0.005
	K	0.185 (0.080-0.426)	<0.0001
	AST	0.999 (0.998-1.000)	<0.05
	ALT	0.998 (0.997-1.000)	<0.05
	Pre-SCr	0.642 (0.480-0.857)	<0.005

Values in parentheses are 95% confidence intervals. *Univariate logistic regression model. WBC: White blood cells, CRP: C-reactive protein, LDH: Lactate dehydrogenase, CPK: Creatine phosphokinase, K: Potassium, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, Pre-SCr/Post-SCr: Preoperative and postoperative serum creatinine.

dynamic stability. The same trends between the groups were observed with regard to post-operative hospital stay (Table 3). Statistically significant differences, all in favor of EVAR compared to the other groups, were observed in WBC, CRP, CPK, AST, ALT as well as the difference between pre-operative SCr and post-operative SCr values (Table 3).

In the EVAR group, comparison of the intraoperative details (internal iliac artery coiling embolization; the coiling group versus the non-coiling group) is investigated. The duration of the operation in the coiling group was significantly longer than that in the non-coiling group (233.7 ± 84.7 vs. 157.2 ± 47.7 min, P <0.01). There were no significant differences between the two groups in post-operative stay, complete blood cell count and serum chemistry values.

Other complications were as follows: in the elective open repair group, there was thrombocytopenia (one patient), acute arterial obstruction (one patient), renal insufficiency (>1.0 mg/dL over baseline, eight patients), pneumonia (two patients), gluteal claudication (three patients), paroxysmal atrial fibrillation (three patients), a stroke (one patient), intestinal obstruction (five patients), and paroxysmal incomplete paralysis (one patient); in the emergency open repair group, there was paroxysmal atrial fibrillation (one patient), renal insufficiency (11 patients), ischemic enteritis (one patient), a duodenal ulcer (one patient), prosthesis infection (one patient), pneumonia (four patients), intestinal necrosis (one patient), intestinal obstruction (three patients), a stroke (one patient), and a retroperitoneal abscess (two patients); and in the EVAR group, there was pneumonia (one patient), intestinal obstruction (one patient), post-operative bleeding (one patient), spinal cord infarction (one patient), kidney infarction (one patient), renal insufficiency (three patients), arterial obstruction (two patients), retroperitoneal hematoma (one patient), prosthesis obstruction (one patient), ischemic enteritis (one patient), type I endoleak (antegrade flow between the aortic wall and stent graft, four patients), and type II endoleak (retrograde flow through the lumbar and inferior mesenteric

Table 5. Multivariate predictors of outcomes among patients undergoing AAA repair.

Outcome measure	Prognostic factor	Odds ratio	*P value
Mortality	Emergency open repair	0.312 (0.017-5.767)	NS
	Blood loss	1.001 (1.000-1.002)	NS
	Blood transfusion	0.999 (0.999-1.000)	<0.05
	K	0.321 (0.117-0.883)	<0.05
	ALT	0.998 (0.996-1.000)	NS
Duration of stay in hospital (>7 days)	Open repair	41.657 (11.740-156.705)	<0.0001
	Operation time	0.990 (0.982-0.999)	<0.05
	Blood loss	1.000 (0.998-1.001)	NS
	WBC	1.000 (1.000-1.000)	NS
	CRP	0.959 (0.885-1.040)	NS
	CPK	0.999 (0.998-1.001)	NS
	ALT	0.982 (0.961-1.004)	NS
	Post-pre SCr	0.164 (0.029-0.925)	<0.05
Major morbidity	WBC	1.000 (1.000-1.000)	NS
	Post-SCr	0.797 (0.572-1.111)	NS
	Post-pre SCr	0.157 (0.063-0.392)	<0.0001
Renal insufficiency	Emergency open repair	0.649 (0.153-2.759)	NS
	Blood loss	1.000 (0.999-1.000)	NS
	Blood transfusion	1.000 (0.999-1.000)	NS
	WBC	1.000 (1.000-1.000)	NS
	CRP	0.900 (0.828-0.979)	<0.05
	K	0.366 (0.139-0.966)	<0.05

Values in parentheses are 95 percent confidence intervals. *Univariate logistic regression model. WBC: White blood cells, CRP: C-reactive protein, LDH: Lactate dehydrogenase, CPK: Creatine phosphokinase, K: Potassium, AST: Aspartate Aminotransferase, ALT: Alanine aminotransferase, Pre-SCr/Post-SCr: Preoperative and postoperative serum creatinine.

arteries, nine patients). Endoleaks were evaluated at 3 to 5 days by computed tomography. No stent fractures were noted with radiographic evaluation. There were no conversion cases.

The independent predictive factors for each outcome variable identified by univariate logistic regression analysis are shown in Table 4. Multivariate logistic analysis showed that blood transfusion (2791.5 ± 1477.9 ml vs. 431.6 ± 0 ml, P <0.05) and post-operative K+ values (5.5 ± 0.28 mmol/L vs. 4.4 ± 0.57 mmol/L, P <0.05) significantly increased mortality. Open repair (192 / 209 cases (91.8%) vs. 6 / 98 cases (6.1%), P <0.0001), operation time (250.6 ± 67.2 min vs. 165.2 ± 20.5 min, P <0.05) and the difference between preoperative SCr and post-operative SCr levels (0.32 ± 6.0 mg/dL vs. 0.11 ± 0.1 mg/dL, P <0.05) significantly extended the duration of the stay in hospital (>7 days). The difference between pre-operative SCr and post-operative SCr (0.78 ± 4.7 mg/dL vs. 0.1 ± 0.93 mg/dL, P <0.0001) significantly increased the risk of major morbidity. CRP (25.4 ± 3.25 mg/dL vs. 16.4 ± 16.0 mg/dL, P <0.05) and post-operative K+ values (5.1 ± 0.42 mg/dL vs. 4.4 ± 0.14 mg/dL, P <0.05) significantly increased the risk of renal insufficiency (>1.0 mg/dL over baseline) (Table 5).

Discussion

In the present study, we compared the preoperative condition, perioperative data of the elective/emergency group and EVAR group, and investigated predictive factors for mortality and morbidity as well as the duration of hospital stay. All patients were treated in one institution during the same period and by the same team of vascular surgeons. The lower rates of mortality and duration of post-operative hospital stay in the EVAR group compared to the elective and emergency open repair groups can be explained by the less invasive procedure. Changes in laboratory data (complete blood cell count and serum chemistry values) of the EVAR group were less than those of the open repair groups. We also found that EVAR was the least invasive procedure. The in-hospital mortality rate was 0% in the EVAR group versus 0.7% in the elective open repair group, which are comparable to previously reported ranges of 0–4.7% for EVAR versus 1.1–4.7% for open repair [4]. The incidence of worsening renal function in patients undergoing open surgical AAA repair with normal preoperative renal function is 5.4%, and it increases two- to three-fold in patients with preexisting chronic renal insufficiency [5,6]. In a nationally representative cohort of patients undergoing AAA repair, EVAR was associated with a 60% reduction in the risk of post-operative renal insufficiency. The protective effect of EVAR was evident among patients with and without chronic renal insufficiency and whether or not dialysis was required for acute renal insufficiency [2]. Furthermore, the difference between pre-operative SCr and post-operative SCr of the EVAR group was smaller than that of elective open repair (P <0.001) and emergency open repair (P <0.05). A severe acute kidney injury seems to increase the risk of progressive chronic kidney disease and may increase the risk of death [7]. To minimize a renal disorder for the perioperative period, it is important not to shift to a chronic renal insufficiency. These results showed that EVAR is a good strategy for an AAA in terms of renal protection.

Patients with extensive aortoiliac aneurysms extending to the iliac bifurcation or involving the internal iliac arteries underwent flow interruption of unilateral or bilateral internal iliac arteries via coil embolization. There were no significant differences between the two groups in post-operative stay, post-operative complete blood cell count and serum chemistry values. This finding suggested that internal iliac artery coil embolization was a minimally invasive strategy.

The patients who underwent EVAR had fewer other complications, compared to the other groups. Additionally, when complications did occur in the EVAR group, they were less morbid than in the open repair groups, and patients in the EVAR group had fewer additional hospital stays after these complications compared to the open repair groups. Specifically, gastrointestinal morbidity was reduced after EVAR was compared to open repair.

Further investigation showed that various factors were predictive for mortality, duration of stay in hospital (>7 days), major morbidity and renal insufficiency in univariate analysis, and there were a few independent prognostic factors in multivariate analysis. Relevant predictors for mortality by multivariate analysis were blood transfusion (P <0.05) and K+ levels (P <0.05).

In the present investigation, more than blood transfusion 2791.5 ± 1477.9 ml and post-operative K+ values 5.5 ± 0.28 mmol/L are predictive values of mortality. We considered that the rise in the K+ level reflected destruction of cells by operative invasion. Human skeletal muscles contain the largest single pool of K+ in the body (2600 mmol, 46 times the total K+ content of the extracellular space). Because of their size and high content of K+, Na+-K+ pumps and K+ channels, skeletal muscles play a central role in the acute, min-to-min ongoing regulation of plasma K+. This is important for the maintenance of muscle contractility and heart function. Hyperkalemia may arise from muscle cell damage. This hyperkalemia is rapidly corrected by reaccumulation of K+ into the muscle cells via Na+-K+ pumps, often leading to hypokalemia. The Na+-K+ pumps in skeletal muscles are stimulated by catecholamines and insulin [8]. It is well recognized from adult studies that a stress response follows injury. The sympathoadrenal system responds almost immediately with an increase in catecholamines, and these increases are proportional to the degree of injury severity [9]. In adults, hypokalemia is well recognized after stress states and is due to a combination of the effect of adrenaline and insulin [10]. From the above findings, it is presumed that K+ is mainly released from damaged muscle cells during an operation. Even if a certain amount of K+ is present in the circulation, homeostasis of K+ may be maintained. However, if excessive K+ is released from damaged cells, it disturbs K+ homeostasis, and hyperkalemia cannot be corrected by reaccumulation of K+ into the muscle cells via Na+-K+ pumps, thus continuing hyperkalemia. This is the reason why K+ values can be used for estimating the degree of damage in an operation and K+ values were a factor for predicting mortality in the current study.

A contemporary study by Carpenter et al. demonstrated no significant difference in mortality between patients with and those without elevated SCr [11]. Yet another study failed to demonstrate statistically significant differences in mortality and major complications when accounting for renal insufficiency [12]. In the present study, the difference between pre-operative SCr and post-operative SCr levels was not a significant predictive factor in mortality.

Similarly, using serum chemistry values, we could predict the development of post-operative renal insufficiency. CRP ($P < 0.05$) and K+ values ($P < 0.05$) significantly increased the risk of renal insufficiency. In the present investigation, more than CRP 25.4 ± 3.25 mg/dL and post-operative K+ values 5.1 ± 0.42 mg/dL are predictive values of renal insufficiency. Based on these results, when an increase in K+ and CRP levels is observed, a protective measure for renal function is necessary. Recent reports have suggested that the mainstay of postoperative acute kidney injury prevention is perioperative maintenance of blood volume with adequate cardiac output by hemodynamic monitoring and fluids/inotropes infusion [13], and perioperative administration of nesiritide reduced the risk of an acute postoperative increase in serum creatinine of more than 0.5 mg/dL or an acute postoperative decrease in GRF of more than 25% of the baseline by 90% (95% confidence interval 33%– 99%, $P = 0.001$) in patients with moderate-to-severe preexisting renal dysfunction [14]. A post-operative increase in K+ and CRP levels gives an indication of the initiation of these treatments. There is previous research specializing in ruptured abdominal aortic aneurysm repair. They reported that an increase in lactate dehydrogenase, ALT, and pre-operative SCr, WBC and CRP levels were the possible predictive values on outcomes (mortality, major morbidity and renal insufficiency) [15].

Comparison of treatment groups on the basis of a retrospective study is subject to several flaws, such as selection bias and differences in patient variables. Therefore, this study has its limitations on interpreting outcomes. The relatively short follow-up time could have caused underestimation of reintervention and secondary conversion rates in favor of open repair.

Conclusion

Open repair and EVAR can both be safely performed in patients treated for elective and emergency infrarenal AAA. EVAR has perioperative advantages of reduced blood loss and blood transfusion, decreased duration of post-operative hospital stay, and reduced invasion of the body, as shown by laboratory data. We identified specific independent predictive factors for mortality, duration of stay in hospital (>7 days), major morbidity and renal insufficiency. Further prognostic improvement is anticipated by using these indices.

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Conflict of interest statement

The authors do not declare any conflict of interest or financial support in this study.

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