Original Article

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Laboratory Studies of Perioperative Abdominal Aortic Aneurysm Repair

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Abstract

Purpose: This study aimed to determine the procedure-related major morbidity of infrarenal abdominal aortic aneurysm (AAA) repair, to investigate the changes in perioperative laboratory values, and to clarify the degree of physical strain of surgery and specific independent predictive factors for major morbidity. In addition, in the case of endovascular aneurysm repair (EVAR), we weighed how occlusion of the internal iliac artery related to physical strain of surgery in terms of laboratory values. We retrospectively evaluated patients who were treated at Jichi Medical University Hospital.

Methods: Consecutive patients with an AAA (excluding ruptured AAA) between April 2007 and August 2010 were studied. The effects of various patient- and operation-related variables on outcomes (major morbidity, duration of stay in hospital, renal insufficiency, and endoleakage) were assessed by univariate and multivariate analyses.

Results: Overall in-hospital mortality was 0.4%. Statistically significant differences, mostly in favor of EVAR, were observed in the intraoperative and postoperative data. The presence of an internal iliac artery occlusion did not affect the perioperative laboratory data. Of various patient- and operation-related variables for each outcome, the relative factors were duration of operation, blood loss, white blood cells, C-reactive protein, lactate dehydrogenase, creatine phosphokinase, and potassium. In multivariate logistic analysis, blood loss, C-reactive protein, creatine phosphokinase, and potassium were significantly related to each outcome.

Conclusions: Both elective open repair and EVAR can be safely performed in patients with an infrarenal AAA. EVAR has perioperative advantages of reduced blood loss and blood transfusions as well as a decreased duration of stay in hospital. In particular, we identified specific independent relative factors of laboratory values for major morbidity, duration of stay in hospital, renal insufficiency, and endoleakage.

Key words: *Abdominal aortic aneurysm, morbidity, laboratory data, open repair, endovascular aneurysm repair*

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Introduction

Abdominal aortic aneurysm (AAA) is defined as an aortic diameter enlargement of at least 150% at the orifice of the renal arteries [1]. Elective treatment is recommended when an AAA reaches 55 mm in diameter because of higher rates of rupture. Open repair of an AAA (reconstruction of the abdominal aorta with a synthetic graft) has always been considered to be among the most major of surgical procedures, and the prospective 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 with open repair. In particular, the usefulness of EVAR for patients with chronic renal insufficiency has been reported by 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 outcomes (major morbidity, duration of stay in hospital, renal insufficiency, and endoleakage) of EVAR and elective open surgery, and it investigated the changes in perioperative laboratory values in the different procedures. The main objective of this study was to clarify the specific independent involvement factors for each outcome for ultimate use in prognostic improvement.

Materials and Methods

We reviewed the data of patients who underwent AAA repair between April 2007 and August 2010. Repair of an infrarenal AAA was performed in 307 consecutive patients. The type of repair was determined by the primary vascular surgeon and patient preference. Patients who underwent emergency repair of symptomatic ruptured AAAs and false aneurysms were excluded. All treatments were performed in an operating room under general anesthesia. This study was approved by the institutional review board. To compare the physical strain by the difference in treatment, two groups were classified according to the type of repair (elective open repair and EVAR). Emergent EVAR was not performed. The device used for open AAA repair was a Dacron prosthesis (Hemashield; Meadox Medicals, Oakland, NJ), and those used for endovascular treatment were an Excluder (W.L. Gore, Flagstaff, AR) and a Zenith (Cook, Bloomington, IN). The duration of operation, intraoperative blood loss (the amount of suctioned blood by Cellsaver was excepted from intraoperative blood loss), blood transfusion volume, and postoperative course were recorded. Intraoperative and perioperative outcomes were assessed. They included major morbidity (bleeding, stenosis, or occlusion of the graft; myocardial infarction; stroke; congestive heart failure; renal insufficiency; bowel obstruction; paralysis; and endoleakage) and duration of stay in hospital. Endoleakage was classified as type I to IV according to standard definitions. Follow-up included clinical examination and postoperative 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. All patients were followed closely in the early postoperative period (at least 30 days) and were assessed for postoperative complications. All patients with a preoperative and postoperative SCr difference of >1.0 mg/dL were considered to have postoperative renal insufficiency. The possible predictive value and operation-related variables of the two groups (elective 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 was used for continuous variables. Data are expressed as the mean value ± standard deviation (SD) or as frequencies and percentages. A p-value of < 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 software (SAS Institute, Cary, NC).

Results

Of the 251 patients who underwent elective infrarenal AAA repair, 151 underwent conventional open repair and 100 underwent EVAR. Demographic characteristics of the patients are listed in Table I. No significant differences in age, sex, or aneurysm diameter were found in our patient population.

Overall in-hospital mortality was 0.4%. In-hospital

Table 1. Demographic characteristics of the patients.

	Elective open repair	EVAR	*p-value
Age (years)	71.4 (7.8)	73.3 (8.4)	NS (0.068)
Male	134 (88.7%)	86 (86%)	NS (0.518)
Aneurysm diameter (mm)	57.3 (12.8)	54.9 (13.2)	NS (0.152)

Values are mean (standard deviation or percentages). EVAR: endovascular aneurysm repair. *Unpaired Student's t-test.

Table 2. Comparison of intraoperative and postoperative details by type of repair.

	Elective open repair	EVAR	*p-value
Duration of operation (min)	253.3 (72.0)	166.4 (8.4)	<0.001
Blood loss (mL)	508.0 (740.1)	129.4 (119.9)	<0.001
Blood transfusion (mL)	211.9 (539.7)	13.6 (82.0)	<0.001
Postoperative hospital stay (days)	12.1 (4.7)	5.8 (3.8)	<0.001
WBC (/μ)	12148.3 (3747.8)	9856 (2611.0)	<0.001
CRP (mg/dL)	20.4 (6.4)	8.9 (5.9)	<0.001
LDH (mU/mL)	285.7 (102.5)	259.2 (154.4)	NS (0.133)
CPK (U/L)	829.0 (745.7)	169.9 (218.5)	<0.001
K+ (mg/dL)	4.4 (0.4)	4.3 (0.4)	NS (0.108)
AST (mU/mL)	51.3 (62.0)	36.8 (51.2)	<0.05
ALT (mU/mL)	34.9 (31.1)	23.0 (20.6)	<0.001
pre-SCr (mg/dL)	1.1 (0.9)	1.2 (1.4)	NS (0.413)
post-SCr (mg/dL)	1.3 (1.1)	1.3 (1.3)	NS (0.802)
(post-SCr)-(pre-SCr) (mg/dL)	0.3 (0.4)	0.1 (0.3)	<0.001

Values are mean (standard deviation). EVAR: endovascular aneurysm repair. *Unpaired Student's t-test. WBC: white blood cell, 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.

Table 3. Comparison of intraoperative and postoperative details by type of repair in EVAR.

	Coiling	Non-coiling	*p-value
Duration of operation (min)	233.7 (84.7)	157.2 (47.7)	<0.01
Blood loss (mL)	156.7 (122.6)	125.7 (119.8)	NS (0.404)
Blood transfusion (mL)	46.7 (161.7)	9.1 (64.7)	NS (0.442)
Postoperative hospital stay (days)	6 (3.2)	5.8 (3.9)	NS (0.867)
WBC (/μ)	9900 (3332.5)	9850 (2520.5)	NS (0.961)
CRP (mg/dL)	11.1 (8.4)	8.6 (5.4)	NS (0.335)
LDH (mU/mL)	308.5 (177.1)	252.4 (151.0)	NS (0.314)
CPK (U/L)	203.3 (186.5)	165.3 (223.1)	NS (0.575)
K+ (mg/dL)	4.2 (0.4)	4.3 (0.4)	NS (0.480)
AST (mU/mL)	39.8 (22.0)	36.4 (54.0)	NS (0.694)
ALT (mU/mL)	21.3 (19.0)	23.3 (20.9)	NS (0.754)
pre-SCr (mg/dL)	1.4 (1.5)	1.2 (1.4)	NS (0.632)
post-SCr (mg/dL)	1.4 (1.5)	1.3 (1.3)	NS (0.710)
(post-SCr)-(pre-SCr) (mg/dL)	0.03 (0.24)	0.09 (0.33)	NS (0.457)

Values are mean (standard deviation). EVAR: endovascular aneurysm repair. *Unpaired Student's t-test. WBC: white blood cell, 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.

mortality was 0.7% for the open repair group and 0% for the EVAR group. One patient died in the elective open repair group; death was related to multiple organ failure.

A comparison of the perioperative details is shown in Table II. The duration of the operation was shorter, and blood loss and blood transfusions were significantly less in the EVAR group, compared with the elective open repair group. The same trends in postoperative hospital stay were observed between the groups. Statistically significant differences, mostly in favor of EVAR compared with elective open repair, were observed in WBC, CRP, CPK, AST, ALT, and the difference between preoperative and postoperative SCr. As a result, EVAR was less stressful than elective open repair in laboratory data.

In the EVAR group, comparisons were made among intraoperative details (internal iliac artery coiling embolization; coiling group vs. non-coiling group) (Table III). The duration of the operation in the coiling group was significantly longer than that in the non-coiling group. There were no significant differences between the two groups in postoperative stay, complete blood cell count, or serum chemistry values. Intestinal ischemia was not observed in both groups.

The major morbidity incidence was 17.2% for elective open repair and 22% for EVAR. The details of 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), stroke (one patient), intestinal obstruction (five patients), and paroxysmal incomplete paralysis (one patient). In the EVAR group, there was pneumonia (one patient), intestinal obstruction (one patient), postoperative 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 endoleakage (antegrade flow between the aortic wall and stent graft, four patients), and type II endoleakage (retrograde flow through the lumbar and inferior mesenteric arteries, nine patients). Endoleakage was evaluated at 3 to 5 days by computed tomography. No stent fractures were noted with radiographic evaluation. There were no conversion cases.

The independent involvement factors of elective open repair for each outcome variable identified by univariate logistic regression analysis are shown in Table IV. CRP (24.6 ± 5.2 mg/dL vs. 19.6 ± 6.3 mg/dL, $p < 0.001$), lactate dehydrogenase (325.3 ± 105.9 mU/mL vs. 277.4 ± 100.2 mU/mL, $p < 0.05$), and CPK (1421.5 ± 1297.4 U/L vs. 705.7 ± 494.3 U/L, $p < 0.001$) were

Table 4. Univariate predictors of outcomes among patients undergoing elective open repair.

Outcome measure	Involvement factor	Odds ratio	*p-value
Major morbidity	Duration of operation	0.995 (0.989-1.000)	<0.1
	CRP	0.861 (0.790-0.938)	<0.001
	LDH	0.996 (0.993-1.000)	<0.05
	CPK	0.999 (0.998-1.000)	<0.001
Duration of stay in hospital (>14 days)	Duration of operation	0.994 (0.989-0.999)	<0.05
	CRP	0.913 (0.850-0.982)	<0.05
	LDH	0.997 (0.993-1.001)	<0.1
	CPK	1.000 (0.999-1.000)	<0.1
	K+	0.272 (0.105-0.703)	<0.01
Renal insufficiency	CRP	0.721 (0.580-0.897)	<0.005
	LDH	0.996 (0.990-1.001)	<0.1
	K+	0.094 (0.023-0.387)	<0.005
	pre-SCr	0.537 (0.349-0.826)	<0.005

Values in parentheses are 95% confidence intervals. *Values found at analysis with univariate logistic regression model. CRP: C-reactive protein, LDH: lactate dehydrogenase, CPK: creatine phosphokinase, K+: potassium, pre-SCr: preoperative serum creatinine.

significantly associated with major morbidity. Duration of operation (280.8 ± 101.0 min vs. 246.8 ± 62.0 min, $p < 0.05$), CRP (23.2 ± 5.1 mg/dL vs. 19.8 ± 6.6 mg/dL, $p < 0.05$), and K+ (4.6 ± 0.5 mg/dL vs. 4.4 ± 0.4 mg/dL, $p < 0.01$) were significantly associated with the duration of the stay in hospital (>14 days). CRP (28.3 ± 3.2 mg/dL vs. 20.0 ± 6.3 mg/dL, $p < 0.005$), K+ (5.0 ± 0.6 mg/dL vs. 4.4 ± 0.4 mg/dL, $p < 0.005$), and preSCr (2.3 ± 2.2 mg/dL vs. 1.0 ± 0.7 mg/dL, $p < 0.005$) were significantly associated with the risk of renal insufficiency (>1.0 mg/dL over baseline).

On the other hand, the independent involvement factors of EVAR are shown in Table V. Duration of operation (201.2 ± 8.4 min vs. 156.6 ± 8.4 min, $p < 0.01$), blood loss (207.3 ± 184.5 mL vs. 107.4 ± 83.6 mL, $p < 0.005$), and WBC (10740.9 ± 2597.8 /μ vs. 9606.4 ± 2576.1 /μ, $p < 0.005$) were significantly associated with major morbidity. Age (77.8 ± 7.0 years vs. 72.4 ± 8.4 years, $p < 0.05$), duration of operation (210.2 ± 8.4 min vs. 157.4 ± 8.4 min, $p < 0.01$), and CPK (319.4 ± 439.2 U/L vs. 138.9 ± 118.3 U/L, $p < 0.05$) were significantly associated with the duration of the stay in hospital (>7 days). AST (20.8 ± 5.6 mU/mL vs. 39.2 ± 54.5 mU/mL, $p < 0.05$) and ALT (12.4 ± 4.8 mU/mL vs. 24.5 ± 21.5 mU/mL, $p < 0.05$) were significantly associated with endoleakage.

Multivariate logistic analysis of the independent involvement factors of elective open repair for each outcome variable showed that CRP ($p < 0.001$) and CPK ($p < 0.005$) were significantly associated with major morbidity. CRP ($p < 0.05$) and K+ ($p < 0.1$) were significantly associated with the duration of stay in hospital (>14 days). CRP ($p < 0.01$) and K+ ($p < 0.01$) were significantly associated with the risk of renal insufficiency (>1.0 mg/dL over baseline).

On the other hand, the independent involvement factors for EVAR showed that blood loss ($p < 0.05$) was significantly associated with major morbidity. Age ($p < 0.1$) was associated with the duration of stay in hospital (>7 days). AST ($p < 0.1$) was associated with the risk of endoleakage.

Operative time, blood loss, WBC, and biochemistry data increased in connection with development of major morbidity, long duration of stay in hospital (>7 or 14 days), and development of renal insufficiency. However, AST and ALT were adversely low in the group in which endoleakage was a complication.

Discussion

In the present study, we compared preoperative conditions and perioperative data of the elective open group and EVAR groups, and investigated involvement factors for mortality and morbidity and duration of stay in hospital. All patients were treated in one institution during the same period by the same team of

Table 5. Univariate predictors of outcomes among patients undergoing EVAR.

Outcome measure	Involvement factor	Odds ratio	*p-value
Major morbidity	Duration of operation	0.987 (0.978-0.996)	<0.01
	Blood loss	0.993 (0.989-.0998)	<0.005
	WBC	1.000 (1.000-1.000)	<0.005
	LDH	0.997 (0.993-1.000)	<0.1
	CPK	0.998 (0.996-1.000)	<0.1
Duration of stay in hospital (>7 days)	Age	0.913 (0.846-0.986)	<0.05
	Duration of operation	0.986 (0.976-0.996)	<0.01
	Blood loss	0.997 (0.993-1.000)	<0.1
	CRP	0.931 (0.857-1.012)	<0.1
	CPK	0.997 (0.994-0.999)	<0.05
Endoleakage	Blood loss	1.004 (1.000-1.008)	<0.1
	AST	0.884 (0.797-0.982)	<0.05
	ALT	0.911 (0.834-0.997)	<0.05

Values in parentheses are 95% confidence intervals. *Values found at analysis with univariate logistic regression model. EVAR: endovascular aneurysm repair. WBC: white blood cell, CRP: C-reactive protein, LDH: lactate dehydrogenase, CPK: creatine phosphokinase, AST: aspartate aminotransferase, ALT: alanine aminotransferase.

vascular surgeons. The lower rates of mortality and duration of stay in hospital in the EVAR group, compared with those in the elective open repair group, 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 group. The in-hospital mortality rate was 0% in the EVAR group versus 0.7% in the elective open repair group; these values are comparable with previously reported ranges of 1.1–4.7% for open repair versus 0–4.7% for EVAR [4].

The patients who underwent EVAR had nearly equal complication rates compared with those in the elective open repair group. However, when complications did occur in the EVAR group, they were less morbid than in the elective open repair group, and patients in the EVAR group had fewer additional hospital stays after these complications, compared with those in the elective open repair group. Specifically, gastrointestinal morbidity was reduced after EVAR compared with open repair. In addition, EVAR had a shorter operative time and duration of stay in hospital as well as a smaller amount of blood loss and blood transfusions than elective open surgery.

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 postoperative 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 preoperative and postoperative SCr in the EVAR group was smaller than that of elective open repair ($p < 0.001$) in the present study. Severe acute kidney injury seems to increase the risk of progressive chronic kidney disease and may increase the risk of death [7]. Minimization of renal dysfunction in the perioperative period is important to prevent a shift to chronic renal insufficiency. These results show that EVAR is a good strategy for an AAA in terms of renal protection. According to the present study's results, we can predict the post-operative renal insufficiency using the value of lactate dehydrogenase, K+ and pre-SCr. We can early evaluate the operative risk prior to surgery, and initiate renal protective treatments (e.g. sufficient fluid administration and injection drug use to keep the renal bloodstream maintenance) to avoid post-operative renal insufficiency.

In addition, statistically significant differences, all in favor of EVAR compared with elective open repair, were observed in WBC, CRP, and CPK. We determined how changes in perioperative laboratory values reflected the degree of physical strain of surgery and evaluated the involvement factors in connection with each outcome (major morbidity, duration of stay in hospital, renal insufficiency, and endoleakage). They are not specific ones to a disease and an organ. However, when the patients have prospective risk guessed from laboratory data for the high mortality and major morbidity, it is recommended that we perform close physician supervision in the intensive-care unit.

Univariate analysis showed that various factors were associated with major morbidity, duration of stay in hospital, renal insufficiency, and endoleakage, and there were a few independent involvement factors in multivariate analysis. Relevant factors for each outcome are described in Tables IV and V as well as the Results section.

Lactate dehydrogenase is a cytoplasmatic enzyme present in essentially all major organ systems. The extracellular appearance of lactate dehydrogenase is used to detect cell damage or cell death. Because of its extraordinarily widespread distribution in the body, serum lactate dehydrogenase is abnormal in a host of disorders [8-10]. It is released into the peripheral blood after cell death caused by ischemia, excess heat or cold, starvation, dehydration, injury, exposure to bacterial toxins, ingestion of certain drugs, and chemical poisonings [8,9]. Enzyme levels in various tissues are about 500-fold higher than those normally found in serum, and leakage of the enzyme from even a small mass of damaged tissue can increase the observed serum level of lactate dehydrogenase to a significant extent [4]. Therefore, we considered that the rise in the serum lactate dehydrogenase level reflected destruction of cells by operative invasion, but it was a nonspecific test.

We also considered that the rise in K+ level reflected cell destruction 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 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 [11]. 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 [12]. In adults, hypokalemia is well recognized after stress states and is caused by a combination of the effects of adrenaline and insulin [13]. 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, K+ homeostasis is disturbed and hyperkalemia cannot be corrected by reaccumulation of K+ into the muscle cells via Na+-K+ pumps, thus continuing hyperkalemia. This is why K+ values can be used to estimate the degree of damage in an operation.

Similarly, using serum chemistry values, we revealed other factors associated with major morbidity. The other relevant factors were CRP and CPK values in the elective open repair group and blood loss, WBC, and CPK in the EVAR group. Elevation in WBC is one of the reactions during immediate inflammation. In response to hematopoietic factor production, increases in hematopoiesis or mobilization of WBC from a leukocyte pool to the bloodstream cause WBC increases. CRP is an acute reaction protein that increases in the blood secondary to inflammation. Activated macrophages release cytokines, which promote the formation of CRP by hepatocytes. The elevation of CRP is closely associated with the degree of disease severity. The present study revealed that these elements, which reflected the strength of the postoperative inflammatory reaction, became the involvement factors for major morbidity, duration of stay in hospital, and renal insufficiency.

CPK is an enzyme present in large quantities in skeletal muscles and nerves, and it increases with injury to these tissues. Elevations in CPK reflect the degree of injury to skeletal muscles by operative invasion. Thus, CPK can be used as an index of the degree of operative invasion. Connections with major morbidity and duration of stay in hospital were revealed in both the open surgery group and EVAR group in this study.

Similarly, using serum chemistry values, we could predict the development of postoperative renal insufficiency. CRP ($p < 0.005$), K+ ($p < 0.005$), and pre-SCr ($P < 0.005$) significantly increased the risk of renal insufficiency. Based on these results, when an increase in CRP, K+, and pre-SCr levels is observed, protective measures for renal function should be undertaken before the onset of clinical symptoms for renal insufficiency. 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 [14]. Perioperative administration of nesiritide reduced the risk of an acute postoperative increase in serum creatinine of >0.5 mg/dL or an acute postoperative decrease in GRF of >25% of the baseline by 90% (95% CI 33–99%, $p = 0.001$) in patients with moderate-to-severe preexisting renal dysfunction [15]. Postoperative increases in CRP, K+, and pre-SCr give an indication of the need for initiation of these treatments. There is a 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) [16].

In patients without endoleakage complication in EVAR, AST and ALT were high compared with those in complicated cases. One reason might be that the blood flow to the inferior mesenteric artery and lumbar artery was completely occluded by the stent graft insertion, which caused tissue dysfunction in the perfusion

area. Therefore, the degree of tissue dysfunction in the group without endoleakage was stronger than that in the group with endoleakage.

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 postoperative stay, postoperative complete blood cell count, and serum chemistry values. This finding suggests that internal iliac artery coil embolization was a minimally invasive strategy.

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 in interpretation of the outcome. The relatively short follow-up time could have caused underestimation of reintervention and secondary conversion rates in favor of open repair.

Conclusion

Both elective open repair and EVAR can be safely performed in patients with an infrarenal AAA. EVAR has perioperative advantages of reduced blood loss and blood transfusions, decreased duration of stay in hospital, and reduced invasion of the body, as shown by laboratory data. We identified specific independent predictive factors for major morbidity, duration of stay in hospital, renal insufficiency, and endoleakage. Further prognostic improvement is anticipated with the use of these indices.

Conflict of interest statement

The authors have no conflicts of interest to declare.

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