Original Article

Archives of Clinical & Experimental Surgery

Predictive Factors for Mortality and Morbidity of Ruptured Abdominal Aortic Aneurysm Repair

Manabu Shiraishi, Kei Aizawa, Yasuhito Sakano, Yuichiro Kaminishi, Shin-ichi Ohki, Tsutomu Saito, Yoshio Misawa

Division of Cardiovascular Surgery
Saitama Medical Center
Jichi Medical University
Saitama, Japan

Received: February 18, 2012
Accepted: March 14, 2012
Arch Clin Exp Surg 2012; 1: 94-101
DOI: 10.5455/aces.20120314112444

Corresponding author:
Manabu Shiraishi
Division of Cardiovascular Surgery
Saitama Medical Center
Jichi Medical University
1-847 Amanuma, Omiya-Ku
Saitama-City 330-8503
Japan
manabu@omiya.jichi.ac.jp

Abstract

Purpose: This study aimed to determine the morbidity and mortality of infrarenal ruptured abdominal aortic aneurysm (rAAA) repair, and to investigate the changes in perioperative laboratory values, as well as clarify the specific independent predictive factors for mortality and morbidity. We retrospectively evaluated patients who were treated in Jichi Medical University Hospital.
Methods: A consecutive fifty-six patients with rAAA between April 2007 and August 2010 were studied. The possible predictive values of various patient-related variables on outcomes (mortality, major morbidity and renal insufficiency) were assessed by univariate and multivariate analysis.
Results: The overall in-hospital mortality was 16.1%. In univariate logistic analyses, lactate dehydrogenase, alanine aminotransferase, as well as pre-operative and post-operative serum creatinine levels were significantly related to mortality. Blood transfusion volume, white blood cells and C-reactive protein levels were significantly related to major morbidity. Intraoperative blood loss, white blood cells, C-reactive protein levels, lactate dehydrogenase and pre-operative serum creatinine levels were significantly related to renal insufficiency.
Conclusions: Emergency open repair can be safely performed in patients for infrarenal rAAA. In particular, we identified specific independent predictive factors of clinical examination and laboratory studies for mortality, major morbidity and renal insufficiency.

Key words: *Ruptured abdominal aortic aneurysm, open repair, predictive factor, renal insufficiency*

Introduction

Abdominal aortic aneurysm (AAA) is defined as an enlargement of the aortic diameter of at least 150% of the diameter at the orifice of the renal arteries[1]. Elective treatment is recommended when AAA size reaches 55 mm in diameter because of higher rates of rupture. 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. However, for emergency treatment of a ruptured abdominal aortic aneurysm (rAAA), open repair has always been considered among the most major of surgical procedures, and the potential complications are highly morbid.

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 morbidity and mortality of open surgery for rAAA, and investigated the changes in peri-operative laboratory values in each different outcome (mortality, major morbidity and renal insufficiency). 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. 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 rAAA repair. All treatments were performed in an operating room under general anesthesia. This study was approved by the institutional review board. Devices used for open AAA repair included a Dacron prosthesis (Hemashield; Meadox Medicals, Oakland, NJ). The low systolic blood pressure (blood pressure < 80 mmHg) on arrival, 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 perioperative mortality and major morbidity (bleeding, stenosis or occlusion of the graft, myocardial infarction, stroke, congestive heart failure, renal insufficiency, bowel obstruction, paralysis). 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), as well as pre-operative and post-operative serum creatinine (SCr) levels. In the initial post-operative period, laboratory data were measured on post-operative 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 post-operative period (at least 30 days) for post-operative 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 values were assessed. Between-group differences in each outcome 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 prob-

Table 1. Demographic characteristics of the patients

				*P value		
	Elective (n=151)	Emergency (n=56)	EVAR (n=100)	Elective versus Emergency	Elective versus EVAR	Emergency versus 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

Values are mean (standard deviation or percentages). *Chi-square test and Unpaired Student's t test. Elective: elective open repair, Emergency: emergency open repair, EVAR: endovascular aneurysm repair. The laboratory values recorded in the postoperative course were the worst ones.

ability (P) < 0.05 was considered statistically significant. All of these potential variables were assessed in univariate analyses. Variables that had a direct effect (P < 0.05 in each outcome) 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

Fifty-six patients underwent emergency open infrarenal rAAA repair. Demographic characteristics and peri-operative details 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%).

In-hospital mortality of rAAA was 16.1% (overall in hospital mortality of AAA was 3.3%). Nine patients died in the emergency open repair group of rAAA; death was cardiac-related in three patients, and was caused by multiple organ failure in five patients and by pneumonia in one patient. The number of patients who had major morbidity was nineteen. Complications were as follows: paroxysmal atrial fibrillation (one patient), renal insufficiency (ten patients), ischemic enteritis (one patient), duodenal ulcer (one patient), prosthesis infection (one patient), pneumonia (four patients), intestinal necrosis (one patient), intestinal obstruction (three patients), stroke (one patient), and retroperitoneal abscess (two patients).

Table 2A. Comparison of the perioperative details in mortality

Prognostic factor	alive case (n=47)	dead case (n=9)	Odd's ratio (95% CI)	*P value
Age (year)	72.2 (10.6)	75 (6.39)	0.967 (0.888-1.054)	NS
Blood pressure <80 mmHg	11 (23.4%)	5 (55.6%)	0.244 (0.056-1.072)	<0.1
Aneurysm diameter (mm)	62.5 (18.9)	70.1 (17.2)	0.978 (0.940-1.019)	NS
Duration of operation (min)	250.5 (71.8)	211 (98.2)	1.008 (0.997-1.018)	NS
Blood loss (ml)	1255.9 (1606.4)	2290.6 (2389.9)	1.000 (0.999-1.000)	NS
Blood transfusion (ml)	2066.9 (2016.7)	2846.1 (2143.1)	1.000 (0.999-1.000)	NS
WBC (/μ)	14859.6 (5602.3)	16575 (11801.7)	1.000 (1.000-1.000)	NS
CRP (mg/dl)	22.2 (7.4)	22.5 (13.2)	0.995 (0.894-1.108)	NS
LDH (mU/ml)	570.9 (714.7)	2212.7 (2747.8)	0.999 (0.999-1.000)	<0.05
CPK (U/l)	4561.8 (14571.3)	4455.6 (4525.2)	1.000 (1.000-1.000)	NS
K (mmol/l)	4.7 (0.7)	5.4 (1.5)	0.477 (0.211-1.077)	<0.1
AST (mU/ml)	221.9 (547.7)	1522.7 (3140.7)	0.999 (0.999-1.000)	NS
ALT (mU/ml)	111.6 (276.7)	490.5 (699.9)	0.998 (0.997-1.000)	<0.05
pre-SCr (mg/dl)	1.2 (0.7)	2.3 (1.7)	0.411 (0.185-0.915)	<0.05
post-SCr (mg/dl)	1.8 (1.7)	3.6 (3.0)	-	-
(post-SCr)-(pre-SCr) (mg/dl)	0.5 (1.5)	0.7 (1.9)	-	-

Values are mean (standard deviation) or number (percent). 95% CI: Values in parentheses are 95 per cent confidence intervals. *Values found at analysis with univariate logistic regression model. 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: pre-operative and post-operative serum creatinine. The laboratory values recorded in the postoperative course were the worst ones.

Statistically significant differences in each outcome were observed in WBC, CRP, ALT, lactate dehydrogenase, pre-operative SCr and post-operative SCr values by univariate analysis (Table 2 A-C). Multivariate logistic analysis showed that WBC values significantly increased major morbidity (Table 3).

Discussion

Overall in-hospital mortality of rAAA was 16.1%. The rate of mortality was lower, compared to the current reports. Mani K et al. reported 7040 ruptured AAA repairs, and revealed that perioperative mortality was 31.6%[2]. Further investigation showed that various factors were predictive for mortality, major morbidity and renal insufficiency in univariate analysis. Relevant predictors for mortality by univariate analysis were lactate dehydrogenase, ALT, and pre-operative SCr.

Lactate dehydrogenase is a cytoplasmatic enzyme present in essentially all major organ systems. 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[3]. Therefore, we considered that the degree of rise in the serum lactate dehydrogenase level reflected destruction of cells by operative invasion, but a nonspecific test.

Statistically significant difference was not revealed, but we also 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

Table 2B. Comparison of the perioperative details in major morbidity

Prognostic factor	major morbidity (+) (n=19)	major morbidity (-) (n=37)	Odd's ratio (95% CI)	*P value
Age (year)	72.8 (10.8)	72.0 (10.4)	0.992 (0.938-1.049)	NS
Blood pressure <80 mmHg	6 (31.6%)	8 (25%)	0.722 (0.206-2.535)	NS
Aneurysm diameter (mm)	68.5 (19.4)	60.2 (19.0)	0.977 (0.948-1.008)	NS
Duration of operation (min)	244.8 (59.8)	256.0 (78.0)	1.002 (0.994-1.011)	NS
Blood loss (ml)	1890.9 (2244.2)	974.2 (1005.0)	1.000 (0.999-1.000)	<0.1
Blood transfusion (ml)	3115.5 (2119.0)	1664.8 (1861.1)	1.000 (0.999-1.000)	<0.05
WBC ($10^3/\mu$)	19.0842 (6.3697)	13.6406 (5.4682)	0.853 (0.758-0.959)	<0.01
CRP (mg/dl)	25.2 (7.2)	20.7 (7.5)	0.916 (0.839-.0999)	<0.05
LDH (mU/ml)	1124.5 (1721.8)	556.5 (764.1)	1.000 (0.999-1.000)	NS
CPK (U/l)	7755 (22064.7)	2819.7 (5061.1)	1.000 (1.000-1.000)	NS
K (mmol/l)	5.0 (1.1)	4.7 (0.7)	0.645 (0.320-1.301)	NS
AST (mU/ml)	700.6 (1945.1)	234.2 (629.8)	1.000 (0.999-1.000)	NS
ALT (mU/ml)	204.2 (423.3)	130.6 (332.4)	0.999 (0.998-1.001)	NS
pre-SCr (mg/dl)	1.8 (1.2)	1.2 (0.7)	0.419 (0.172-1.021)	<0.1
post-SCr (mg/dl)	3.4 (2.7)	1.2 (0.6)	-	-
(post-SCr)-(pre-SCr) (mg/dl)	1.6 (2.3)	0.06 (0.35)	-	-

Values are mean (standard deviation) or number (percent). 95% CI: Values in parentheses are 95 per cent confidence intervals. *Values found at analysis with univariate logistic regression model. 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: pre-operative and post-operative serum creatinine. The laboratory values recorded in the postoperative course were the worst ones.

and high content of K^+, Na^+-K^+ pumps and K^+ channels, skeletal muscles play a central role in the regulation of plasma K^+[4]. 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, 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 and renal insufficiency in the current study.

The result of each outcome is settled briefly, as follows. The values of lactate dehydrogenase, ALT and Pre-SCr in the death group are significantly higher than that of the alive group. Blood transfusion and the values of WBC and CRP in the major morbidity group are significantly higher than that of the non-major morbidity group. Blood loss and the values of WBC, CRP, lactate dehydrogenase and Pre-SCr in the renal insufficiency group are significantly higher than that of the non-renal insufficiency group.

In the present study, there was no statistical difference in the amount of intraoperative blood loss and blood transfusion between alive cases and dead cases. However, Hatori et al. reported that less blood loss and blood transfusions were the factors of a satisfactory outcome in patients of rAAA[5]. Moreover, there was a relationship (P <0.1) in the low systolic blood pressure (blood pressure <80 mmHg) on arrival between alive cases and dead cases in this study. Tambyraja et al. and Vogel et al. reported that blood pressure (<90 mm Hg or <80 mm Hg) is one of the predictive factors that is associated with mortality for rAAA surgery[6,7]. In addition, preoperative anemia (hemoglobin <9 g/dl or <8 g/dl), age, the Glasgow Coma Scale (<15), body temperature on arrival and free rupture are reported as the specific factors[6-9].

Table 2C. Comparison of the perioperative details in renal insufficiency

Prognostic factor	renal insufficiency (+) (n=10)	renal insufficiency (-) (n=55)	Odd's ratio (95% CI)	*P value
Age (year)	73.4 (12.4)	72.5 (9.7)	0.988 (0.923-1.058)	NS
Blood pressure <80 mmHg	4 (40%)	12 (26.1%)	0.556 (0.161-1.919)	NS
Aneurysm diameter (mm)	64.1 (13.2)	63.5 (19.8)	0.986 (0.953-1.021)	NS
Duration of operation (min)	274.1 (77.0)	237.7 (76.3)	1.006 (0.995-1.017)	NS
Blood loss (ml)	2489.3 (2798.8)	1190.2 (1399.4)	1.000(0.999-1.000)	<0.05
Blood transfusion (ml)	2323 (2034.4)	2163.6 (2059.9)	1.000(0.999-1.000)	NS
WBC (/μ)	21680 (6614.9)	13648.9 (5872.3)	1.000(1.000-1.000)	<0.005
CRP (mg/dl)	26.6 (7.5)	21.2 (7.9)	0.884 (0.787-0.992)	<0.05
LDH (mU/ml)	1782.2 (2208.5)	518.3 (667.7)	0.999 (0.999-1.000)	<0.05
CPK (U/l)	13719 (29788.1)	2463.7 (4502.0)	1.000 (1.000-1.000)	NS
K (mmol/l)	5.5 (1.2)	4.7 (0.7)	0.444 (0.191-1.033)	<0.1
AST (mU/ml)	1197.9 (2637.9)	207 (542.7)	0.999 (0.999-1.000)	NS
ALT (mU/ml)	288.7 (561.3)	123.3 (295.9)	0.999 (0.997-1.001)	NS
pre-SCr (mg/dl)	2.2 (1.5)	1.2 (0.7)	0.437 (0.197-0.970)	<0.05
post-SCr (mg/dl)	5.1 (2.7)	1.3 (0.6)	-	-
(post-SCr)-(pre-SCr) (mg/dl)	3.0 (2.5)	0.02 (0.47)	-	-

Values are mean (standard deviation) or number (percent). 95% CI: Values in parentheses are 95 per cent confidence intervals. *Values found at analysis with univariate logistic regression model. 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: pre-operative and post-operative serum creatinine. The laboratory values recorded in the postoperative course were the worst ones.

In the present study, pre-operative SCr was also a significant predictive factor in mortality. On the other hand, a contemporary study by Carpenter et al. demonstrated no significant difference in mortality between patients with and those without elevated SCr [10]. Yet another study failed to demonstrate statistically significant differences in mortality and major complications when accounting for renal insufficiency [11]. However, according to the present study's results, we can predict the mortality and the post-operative renal insufficiency using the value of 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.

Similarly, through using serum chemistry values, we could predict the development of major morbidity. Other laboratory studies (e.g. WBC, LDH and CRP) are values provided after surgery. They are revealed as those elements that reflected the strength of the postoperative inflammatory reaction and destruction of cells. 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.

We could also predict the development of post-operative renal insufficiency. When the rise in values of WBC, CRP and lactate dehydrogenase are recorded, they may be a great risk for post-operative renal insufficiency, similar to the high value of pre-SCr. Furthermore, it is desirable to initiate renal protective treatments previously 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 [12], 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 [13].

A post-operative increase in WBC, CRP, lactate dehydrogenase and pre-operative SCr values gives an indication of the initiation of these treatments. For these

Table 3. Multivariate predictors of outcomes among patients undergoing rAAA repair

Outcome measure	Prognostic factor	Odd's ratio (95% CI)	*P value
Mortality	LDH	1.000 (0.998-1.001)	NS
	ALT	1.000 (0.996-1.005)	NS
	Pre-SCr	0.503 (0.151-1.669)	NS
Major morbidity	Blood transfusion	1.000 (0.999-1.000)	<0.1
	WBC	0.877 (0.773-0.996)	<0.05
	CRP	0.940 (0.850-1.040)	NS
Renal insufficiency	Blood loss	1.000 (0.999-1.000)	NS
	WBC	1.000 (1.000-1.000)	NS
	CRP	0.894 (0.768-1.041)	NS
	LDH	1.000 (0.999-1.001)	NS
	Pre-SCr	0.977 (0.253-3.767)	NS

rAAA: ruptured abdominal aortic aneurysm, 95% CI: 95 per cent confidence intervals. *Values found at analysis with univariate logistic regression model.

reasons, the observation of these clinical and laboratory data can improve operation results of rAAA.

There is a limitation in the present report. This study was performed in a circulatory center of the university hospital. There are patients directly transferred in shock, but it is a fact that the patients that survived a shock state are often introduced for the purpose of an emergency surgery. Vogel et al. reported that the incidence of mortality within twenty-four hours of emergency surgery for the patients directly transferred was significantly higher in the patients who were transferred from local community centers [7]. Our institution has a high percentage of patients that are in a critical but stable condition, compared to that of local hospitals. Therefore, there may be an institutional bias on that point. Also, examination in more cases is necessary, because there is a little number of cases in the present study.

Conclusion

We identified specific independent predictive factors for mortality, major morbidity and renal insufficiency. Further prognostic improvement is anticipated by using these indices.

Acknowledgments

No external funding was obtained for the work presented here.

Conflict of interest statement

The authors have no conflicts of interest to declare.

References

1. Ruiz CE, Zhang HP, Douglas JT, Zuppan CW, Kean CJ. A novel method for treatment of abdominal aortic aneurysms using percutaneous implantation of a newly designed endovascular device. Circulation 1995;91:2470-2477.
2. Mani K, Lees T, Beiles B, Jensen LP, Venermo M, Simo G, et al. Treatment of abdominal aortic aneurysm in nine countries 2005-2009: a vascunet report. Eur J Vasc Endovasc Surg 2011;42:598-607.
3. Moss DW, Henderson AR. Enzymes. In: Burtis CA, Ashwood ER, eds. Tietz Textbook of Clinical Chemistry, W.B. Saunders Company, Philadelphia, 1986: 735–896.
4. Clausen T. Hormonal and pharmacological modification of plasma potassium homeostasis. Fundam Clin Pharmacol 2010;24:595-605.
5. Hatori N, Yoshizu H, Shimizu M, Hinokiyama K, Takeshima S, Kimura T, et al. Prognostic factors in the surgical treatment of ruptured abdominal aortic aneurysms. Surg Today 2000;30:785-790.
6. Tambyraja A, Murie J, Chalmers R. Predictors of outcome after abdominal aortic aneurysm rupture: Edinburgh Ruptured Aneurysm Score. World J Surg 2007;31:2243-2247.
7. Vogel TR, Nackman GB, Brevetti LS, Crowley JG, Bueno MM, Banavage A, et al. Resource utilization and outcomes: effect of transfer on patients with ruptured abdominal aortic aneurysms. Ann Vasc Surg 2005;19:149-153.
8. Alexander S, Bosch JL, Hendriks JM, Visser JJ, Van Sambeek MR. The 30-day mortality of ruptured abdominal aortic aneurysms: influence of gender, age, diameter and comorbidities. J Cardiovasc Surg (Torino) 2008;49:633-637.
9. Bonardelli S, Cervi E, Maffeis R, Nodari F, De Lucia M, Guadrini C, et al. Open surgery in endovascular aneurysm repair era: simplified classification in two risk groups owing to factors affecting mortality in 137 ruptured abdominal aortic aneurysms (RAAAs). Updates Surg 2011;63:39-44.
10. Carpenter JP, Fairman RM, Barker CF, Golden MA, Velazquez OC, Mitchell ME, et al. Endovascular AAA repair in patients with renal insufficiency: strategies for reducing adverse renal events. Cardiovasc Surg 2001;9:559-564.

11. Mehta M, Veith FJ, Lipsitz EC, Ohki T, Russwurm G, Cayne NS, et al. Is elevated creatinine level a contraindication to endovascular aneurysm repair? J Vasc Surg 2004;39:118-123.

12. Kor DJ, Brown MJ, Iscimen R, Brown DR, Whalen FX, Roy TK, et al. Perioperative statin therapy and renal outcomes after major vascular surgery: a propensity-based analysis. J Cardiothorac Vasc Anesth 2008;22:210-216.

13. Dyke CM, Bhatia D, Aronson S, Moazami N, Mentzer RM Jr. Perioperative nesiritide and possible renal protection in patients with moderate to severe kidney dysfunction. J Thorac Cardiovasc Surg 2008;136:1369-1370.

DOI: 10.5455/aces.20120314112444