

Factors Affecting the Postoperative Mortality in the Ruptured Abdominal Aortic Aneurysm

Hyo Yeong Ahn, M.D.¹, Sung Woon Chung, M.D.¹, Chung Won Lee, M.D.¹,
Min Su Kim, M.D.¹, Sangpil Kim, M.D.¹, Chang Won Kim, M.D.²

Background: Although patients with a ruptured abdominal aortic aneurysm (RAAA) often reach the hospital alive, the perioperative mortality is still very high. We retrospectively reviewed thirty patients who underwent repair of RAAA to identify the factors affecting postoperative mortality in a single hospital. **Materials and Methods:** Between September 2007 and May 2011, thirty patients with RAAA underwent emergent surgery (n=27) or endovascular aneurysm repair (n=3). Their medical records were retrospectively reviewed regarding three categories: 1) preoperative patient status: age, gender, vital signs, serum creatinine, blood urea nitrogen, hematocrit, and hemoglobin level; 2) aneurysmal status: size, type, and rupture status; and 3) operative factors: interval time to operating room, operative duration, and amount of perioperative transfusion. **Results:** The 30-day postoperative mortality rate was 13.3% (4/30); later mortality was 3.3% (1/30). On multivariate analysis, the initial diastolic blood pressure (BP), interval time to operating room and amount of preoperative packed cell transfusion were statistically significantly linked with postoperative mortality ($p < 0.05$). **Conclusion:** In this study, preoperative diastolic BP, preoperative packed cell transfusion amount and interval time between arrival and entry to operating room were significantly associated with postoperative mortality. It is important to prevent hemorrhage as quickly as possible.

Key words: 1. Aneurysm
2. Rupture
3. Mortality
4. Aorta, surgery
5. Blood transfusion

INTRODUCTION

The incidence of abdominal aortic aneurysm (AAA) has been increasing, due in part to the aging of the population. Although the postoperative mortality of elective AAA repair has been decreased, that of ruptured abdominal aortic aneurysm (RAAA) is still high. According to some reports, the mortality rate of RAAA patients is about 75%, and a half of

them die before reaching the hospital [1,2]. A number of studies have reported the factors affecting the postoperative mortality [1,3-5]. In this study, we reviewed the factors affecting postoperative mortality in RAAA within the text of preoperative patient status, aneurysmal factors and operative factors.

Departments of ¹Thoracic and Cardiovascular Surgery and ²Diagnostic Radiology, Pusan National University School of Medicine

†This study was supported by Pusan National University Hospital grant (2011).

Received: November 3, 2011, Revised: March 7, 2012, Accepted: April 2, 2012

Corresponding author: Sung Woon Chung, Department of Thoracic and Cardiovascular Surgery, Pusan National University Hospital, Pusan National University School of Medicine, 305 Gudeok-ro, Seo-gu, Busan 602-739, Korea
(Tel) 82-51-240-7267 (Fax) 82-51-243-9389 (E-mail) sungwoon@pusan.ac.kr

© The Korean Society for Thoracic and Cardiovascular Surgery. 2012. All right reserved.

© This is an open access article distributed under the terms of the Creative Commons Attribution Non-Commercial License (<http://creativecommons.org/licenses/by-nc/3.0>) which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

MATERIALS AND METHODS

1) Patients

For a period of 44 months from September 2007 to May 2011, thirty patients underwent the repair of RAAA at Pusan National University Hospital (PNUH). The medical records of the patients were retrospectively reviewed. Almost all of them were diagnosed already at local hospitals and transferred to PNUH. All patients had symptoms such as severe abdominal pain, loss of consciousness or unstable vital signs. To confirm RAAA, abdominal computed tomography was performed in most cases, unless vital signs were unstable. Most patients (n=27) underwent open surgical repair through retroperitoneal or transabdominal incision. The other patients (n=3) who have a long neck below the renal artery and an obtuse angle in common iliac artery underwent endovascular aneurysm repair (EVAR).

2) Performed procedures

(1) Open surgical repair: The operation was routinely performed in regular sequence, with the dissection of the AAA and aorta cross clamping. The distal common iliac arteries were clamped unless they had aneurysms, otherwise the femoral arteries were clamped. After resection of aneurysm, a Y-graft was interposed and anastomosed. The inferior mesenteric artery and lumbar arteries were ligated. The graft was wrapped with the remaining aortic wall.

(2) Endovascular aneurysm repair: Placing the patient in the supine position, bilateral access was obtained via the common femoral artery. Maintaining an activated clotting time ≥ 300 seconds, a 10F introducer sheath was inserted over the access guidewire after pre-closure of access sites by Perclose device (Abott, Redwood City, CA, USA). A 5F pig-tail catheter was used for aortography. After measuring the length and diameter of main trunk and iliac arteries, the stent graft for main trunk and ipsilateral limb were inserted, and the stent graft for the contralateral limb through the contralateral side. Apposition of each stent graft to the aortic wall was performed by balloon catheter. Patency of stent graft and possible endoleak were checked on the aortogram after EVAR. Bilateral access sites were closed by pre-closure technique.

3) Postoperative management

After repair procedures, patients were sedated until vital signs became stable in the intensive care unit. Extubation was carried out as soon as possible. Oral intake, starting with drinking water, could be initiated when the bowel sounds were heard. Except for cases of unstable vital signs, dyspnea, pulmonary edema, and/or decreased urine output, patients were usually transferred to general wards to carry out ambulation, usually 2 days after extubation.

4) Measures

Variables were considered in the context of preoperative patient status, aneurysmal factors and operative factors. First, the preoperative patient status measures included age, gender, vital signs, the serum creatinine, blood urea nitrogen, hematocrit, hemoglobin level and patients' comorbidities. Second, aneurysmal factors included aneurysmal size, type and rupture status. Third, operative factors analyzed included the interval time to the operating room, the operative duration, and the amount of preoperative transfusion.

Data were analyzed with IBM SPSS (SPSS Inc., Chicago, IL, USA), and statistical significance was assumed for $p < 0.05$. For univariate statistical analysis, unpaired Student's t-test, and chi-squared test were used.

RESULTS

Of the 30 patients included in this study, 28 were male (93.3%) and two were female (6.7%). The mean age was 70.57 years, ranging from 55 to 84 years old. Table 1 shows the preoperative characteristics of the patients. The associated comorbidities of the patients were hypertension (n=21), cerebrovascular attack (CVA) (n=2), peripheral vascular disease (n=1), renal dysfunction (n=3), and arrhythmia (n=1).

Overall, 25 patients survived after their emergency operation and five patients died. Of five patients who died, four patients (13.3%, 3: surgical repair, 1: EVAR) died in early postoperative day (<30 days) and one patient (3.3%, surgical repair) in late postoperative day.

Table 1. Preoperative patient status

Risk factor	Survivor	Death	p-value
Age (yr)	70.88	69.00	0.61
Sex (male:female)	23:2	5:0	0.69
Systolic BP (mmHg)	113.56	108.0	0.358
Diastolic BP (mmHg)	68.52	57.80	0.044
Heart rate (/min)	79.56	80.80	0.989
Hemoglobin (g/dL)	10.90	9.80	0.816
Hematocrit (%)	32.52	28.98	0.582
Creatinine (mg/dL)	1.66	2.06	0.978
Blood urea nitrogen (mg/dL)	26.78	33.52	0.846

Values are presented as mean or number.
BP, blood pressure.

1) Preoperative patient status

The initial mean systolic blood pressure (BP) of those who died was slightly lower than that of the survivors (108 mmHg and 114 mmHg, respectively); however, this difference was not statistically significant ($p=0.358$). The mean diastolic BP of five patients who died was statistically significantly different to that of the survivors (57 mmHg vs. 68 mmHg, $p=0.044$). Baseline heart rate was slightly higher in patients who died, but this difference was not statistically significant ($p=0.989$).

The preoperative hemoglobin and hematocrit values were lower in the mortality group than those in the survivors, at 9.8 g/dL and 10.8 g/dL, and 28.9% and 32.5%, respectively. The differences were not statistically significant ($p=0.816$, 0.582). The preoperative blood urea nitrogen and creatinine of the mortality group were 33.52 mg/dL and 2.06 mg/dL, respectively, and those of the surviving group were 14.9 mg/dL and 12.3 mg/dL, respectively, which was not statistically significant ($p=0.846$ and $p=0.978$, respectively) (Table 1).

2) Aneurysmal factors

Based on the radiologic findings, aneurysm size and rupture status could be estimated retrospectively. Some reports indicate that aneurysm size is related to perioperative mortality [2,5]; however, on the contrary in our study, the aneurysm size in the survivors was larger than in the patients who died, at 7.77 cm and 7.28 cm, respectively. Concerning the type of rupture, 22 patients had retroperitoneal ruptures, four patients had peritoneal ruptures and four patients showed character-

Table 2. The aneurysmal rupture status

	Survivor	Death	p-value
Aneurysm type			
Suprarenal	2	0	
Infrarenal	23	5	0.69
Aneurysm size (cm)	7.77	7.28	0.69
Rupture site			
Peritoneal	4	0	
Retroperitoneal	18	4	
Peritoneal and retroperitoneal	3	1	0.35
Approach method			
Surgical approach	23	4	
Transabdominal	6	0	
Retroperitoneal	17	4	0.298
Endovascular repair	2	1	

istics of both of retroperitoneal and peritoneal rupture. In the retroperitoneal rupture group, the mortality rate (73.3%) was higher than in the other groups, but no statistically significant difference was seen (Table 2).

3) Operative factors

Considering perioperative aspects, the longer the elapsed time in the emergency room (ER), the higher the mortality was after surgery. The mean interval time between arrival at the ER and entry to the operating room or angiography room was 254.5 minutes in the survival group and 503.0 minutes in the mortality group, and the difference was statistically significant ($p<0.001$). The operating time was longer in patients who died than survivors, 400 minutes and 295 minutes, respectively, but it was not statistically significant ($p=0.80$). During preparation for operation, patients who showed unstable vital signs, such as systolic BP <80 mmHg, heart rate >100 /min, and confusion were given packed cell transfusions. The mean preoperative transfusion amount of the mortality group was higher than for the survival group at 2,240 mL and 448 mL, respectively, and this difference was statistically significant ($p=0.005$). With regard to the amount of preoperative fresh frozen plasma transfused, the mortality group received more than the survivors, at 1,440 mL and 384 mL, respectively: this difference was also statistically significant ($p=0.004$) (Table 3).

Table 3. The operative factors and the amount of preoperative transfusion

	Survivor	Death	p-value
Interval time between ER to OR (min)	254.5	503.0	<0.001
Duration of operation (min)	295.3	400.0	0.80
Preoperative packed cell transfusion (mL)	448	2,240	0.05
Preoperative fresh frozen plasma transfusion (mL)	384	1,446	0.04
Preoperative platelet transfusion (mL)	256	640	0.347

Values are presented as mean or number.
ER, emergency room; OR, operating room.

4) Postoperative mortality and follow-up

Of five patients who died, four patients had hypovolemic shock due to coagulopathy in early postoperative day (<30 days) and one contracted pneumonia at a later postoperative point (>30 days). The survivors were regularly followed up in the outpatient clinic until April, 2011, with no further complications reported.

DISCUSSION

The etiology of AAA is intimately related with the degradation of the elastic media of the atheromatous aorta [2]. There are many associated risk factors, such as old age, lower preoperative BP, hemoglobin <10 g/dL, and creatinine >1.5 mg/dL, postoperative mortality became higher [4,5]. To develop AAA, the involved biochemistry and molecular regulation of matrix macromolecules were reported, such as over-expressed stromelysin-1, tissue inhibitor of metalloproteinase (TIMP)-3 and lower TIMP-2 and plasminogen activator inhibitor-1 messenger ribonucleic acid levels [6-8].

With the advancement of radiology, the diagnosis of arterial disease becomes less difficult. However many people experience few symptoms of AAA, such as abdominal pain, claudication, and loss of consciousness, until the event of rupture [2]. After rupture, the mortality of AAA usually becomes high. The statistics reported RAAA mortality at between 65% and 85%, with the elective operations occurring at a rate of only 1% to 3% [2,4]. In elective operations, pa-

tients were evaluated for cardiac and pulmonary function, and underlying diseases could be treated before the operation to reduce the risk of postoperative complications. However, during emergent operations, the patients could have concomitant embolic events, such as myocardial infarction and CVA, and could have more complications arising from hypovolemia and coagulopathy due to the need for massive transfusion to keep vital signs stable [9-11]. In our study, preoperative diastolic BP, preoperative transfusion amount, and the interval time between the arrival at ER and at the operating room are statistically significantly associated with postoperative mortality (Tables 1-3).

There are multiple variables reflecting active blood loss and hypovolemic status. In particular, the systolic and diastolic BP could decrease and heart rate (HR) could increase in acute hemorrhage. Although the difference of BP and HR between the survival and mortality groups was not statistically significant in this study (p=0.36 vs. 0.99), the mean systolic BP was higher and the mean HR was slightly lower in survivors. The lower diastolic BP was associated with a significantly higher mortality (p=0.04). However just one factor—preoperative mean diastolic BP—could not rationally explain the connection to postoperative mortality. Overall, various parameters showed mortality group had lower systolic and diastolic and higher HR as reflecting active volume loss. And the active volume loss induced lots of transfusion or volume infusion, which caused the dilutional coagulopathy.

The hemoglobin level usually cannot reflect the hypovolemic status in short timeframes. In this study, the hemoglobin and hematocrit were lower in the patients who died but were not related with a significantly higher mortality. However, the preoperative transfusion amount was significantly associated with higher mortality. The high transfusion amount reflects extreme blood loss. Massive transfusion with a volume expander causes dilutional coagulopathy [9-11]. Furthermore, changes in the hemostatic system and hypoxia due to hypotension cause extravasation of tissue factor, which leads to a consumptive coagulopathy [9,10]. During operation, heparin injection and manipulation of tissue could aggravate the platelet dysfunction and the coagulopathy [9]. Acute hemorrhage demands massive transfusion, which causes coagulopathy, and the long duration of hypotension

causes tissue anoxia, which leads to disseminated intravascular coagulopathy [9-11]. This cycle may be the cause of death for four patients who died just after surgery in our study. Some surgeons suggest that to prevent coagulopathy following by massive transfusions of packed cells, the transfusion with platelet concentration and fresh frozen plasma should follow [9-11].

Longer interval time to operation or intervention was strongly associated with high mortality. Most patients had a diagnosis from the previously visited local hospital, which would contact us to prepare to operate before departing. However, a few patients complaining of abdominal pain, loss of consciousness and dizziness would be wrongly admitted to other departments. Their general condition and vital signs became worse during the evaluation. The long duration for an evaluation caused delayed operations and resulted in a poor outcome. Whether by open surgical repair or by endovascular repair, preventing of bleeding is important.

There could be a possibility of bias as the amount of packed cell transfusion increase in a time-dependent manner. However, the relationship between them was not statistically significant ($p=0.594$).

According to some reports, endovascular aneurysm repair can be successfully performed unless the proximal lack of aneurysm is shorter than 15 mm, the angulation of proximal neck is greater than 120° , or the iliac arteries are tortuous and occlusive [12-14]. Sometimes, in old patients with high risk comorbidities under general anesthesia, endovascular repair is preferred to open repair.

Even in the elective AAA operations, patients who have high risk factors, such as previous myocardial infarction, CVA, or chronic obstructive pulmonary disease, usually undergo cardiac and pulmonary evaluation to prevent postoperative complication. In our study, the comorbidities did not impact mortality. It seems that a severe hypovolemic state may overwhelm the impact of comorbid factors on the perioperative mortality.

CONCLUSION

In our study, the preoperative diastolic pressure, the amount of preoperative packed cell transfusion, and the inter-

val time to operating room were significantly associated with postoperative mortality. It is better to prevent blood loss as quickly as possible by open repair or by endovascular repair. If the patient is not suitable for open repair, endovascular repair should be considered.

REFERENCES

1. Kniemeyer HW, Kessler T, Reber PU, Ris HB, Hakki H, Widmer MK. *Treatment of ruptured abdominal aortic aneurysm, a permanent challenge or a waste of resources? Prediction of outcome using a multi-organ-dysfunction score.* Eur J Vasc Endovasc Surg 2000;19:190-6.
2. Sakalihan N, Limet R, Defawe OD. *Abdominal aortic aneurysm.* Lancet 2005;365:1577-89.
3. Dominguez Fernandez E, Erhard J, Stracke A, Albrecht KH. *Surgical management of ruptured abdominal aortic aneurysm--diagnosis, risk factors and prognosis.* Zentralbl Chir 1998;123:42-5.
4. Halpern VJ, Kline RG, D'Angelo AJ, Cohen JR. *Factors that affect the survival rate of patients with ruptured abdominal aortic aneurysms.* J Vasc Surg 1997;26:939-45.
5. Alonso-Perez M, Segura RJ, Sanchez J, et al. *Factors increasing the mortality rate for patients with ruptured abdominal aortic aneurysms.* Ann Vasc Surg 2001;15:601-7.
6. Carrell TW, Burnand KG, Wells GM, Clements JM, Smith A. *Stromelysin-1 (matrix metalloproteinase-3) and tissue inhibitor of metalloproteinase-3 are overexpressed in the wall of abdominal aortic aneurysms.* Circulation 2002;105:477-82.
7. Defawe OD, Colige A, Lambert CA, et al. *TIMP-2 and PAI-1 mRNA levels are lower in aneurysmal as compared to athero-occlusive abdominal aortas.* Cardiovasc Res 2003; 60:205-13.
8. Ghorpade A, Baxter BT. *Biochemistry and molecular regulation of matrix macromolecules in abdominal aortic aneurysms.* Ann N Y Acad Sci 1996;800:138-50.
9. Hardy JF, de Moerloose P, Samama CM; Members of the Groupe d'Interet en Hemostase Perioperatoire. *Massive transfusion and coagulopathy: pathophysiology and implications for clinical management.* Can J Anaesth 2006;53(6 Suppl):S40-58.
10. Murray DJ, Olson J, Strauss R, Tinker JH. *Coagulation changes during packed red cell replacement of major blood loss.* Anesthesiology 1988;69:839-45.
11. Murray DJ, Pennell BJ, Weinstein SL, Olson JD. *Packed red cells in acute blood loss: dilutional coagulopathy as a cause of surgical bleeding.* Anesth Analg 1995;80:336-42.
12. Buskens E. *Endovascular repair of abdominal aortic aneurysm.* Lancet 2005;366:890-1.
13. Greenhalgh RM, Brown LC, Kwong GP, Powell JT,

Thompson SG; EVAR trial participants. *Comparison of endovascular aneurysm repair with open repair in patients with abdominal aortic aneurysm (EVAR trial 1), 30-day operative mortality results: randomised controlled trial.* Lancet

2004;364:843-8.

14. Lindholt JS. *Endovascular aneurysm repair.* Lancet 2004; 364:818-20.