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ORIGINAL ARTICLE

# Prognostic factors associated with mortality in patients undergoing emergency surgery for abdominal aortic aneurysms

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**Abstract**
*Purpose* Surgical mortality rates following emergency surgery for ruptured abdominal aortic aneurysms (AAAs) remain high. This study investigated the mortality rate and identified prognostic factors affecting mortality in patients undergoing emergency repair of AAAs in our hospital.
*Methods* Between January 2005 and June 2010, a total of 42 patients underwent emergency surgery for AAAs and were included in this retrospective study. The following variables concerning each patient were collected by chart review and compared between survivors and nonsurvivors: age; gender; preoperative levels of hemoglobin (Hb), hematocrit (Ht), platelets (Plts), base excess (BE), and serum glucose and lactate; presence of preoperative shock defined as hypotension (systolic blood pressure of less than 80 mmHg); incidence of blood transfusion, whether AAA was ruptured or impending; interval from admission to the hospital or arrival in the operating room until aortic cross-clamping; surgical duration; and volume of intraoperative blood loss and transfusion, total fluid infusion, and urine output.
*Results* Nine patients died within 30 days postoperatively, a 30-day mortality rate of 21.4%. Among these nine nonsurvivors, eight had shown persistent preoperative shock ($P = 0.0004$ vs. survivors). Compared with the survivors, nonsurvivors were significantly older ($P = 0.0052$) and had lower preoperative levels of Hb/Ht ($P < 0.0001$), Plts ($P = 0.0003$), and BE ($P < 0.0001$), an elevated lactate level ($P = 0.0048$), shorter interval from admission ($P = 0.0459$) or arrival in the operating room ($P = 0.0288$) until aortic clamping, and intraoperatively more hemorrhage ($P = 0.0038$) associated with larger amounts of blood transfusion ($P = 0.0083$) and less urine output ($P = 0.0004$).
*Conclusions* The authors clarified that certain features such as age, persistent preoperative shock, and greater amounts of transfusion associated with greater blood loss and anemia were factors affecting the mortality in patients undergoing emergency surgery for AAAs. It might be of great importance to correct preoperative shock and anemia caused by massive bleeding before the onset of hemodynamic deterioration.

**Keywords** Abdominal aortic aneurysms · Mortality · Prognostic factors · Hypotension · Hemorrhagic shock

## Introduction

The authors have often anesthetized patients undergoing either elective or emergency surgery for abdominal aortic aneurysms (AAAs), probably in part because the Kurume University Hospital, to which the Advanced Emergency and Critical Care Medical Center is affiliated, is one of the key hospitals in our district. Concerning surgery of AAAs, the postoperative mortality rates following emergency surgery have been reported to remain as high as 20–70%, particularly in cases of ruptured AAAs; nevertheless, those in elective surgery are recently improving, to as low as 1–2% [1–10]. The individual institutional mortality rate for ruptured AAAs can vary, possibly depending upon referral pattern and specialization of the hospital. A large number of attempts have been made to clarify specific risk profiles and to infer predictive factors attributable to postoperative

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death in surgical patients with ruptured AAAs [11–21]. The aim of this retrospective study was to identify prognostic, preoperative, and intraoperative factors influencing mortality in patients undergoing emergency surgery for ruptured or imminent rupture of AAAs.

## Materials and methods

### Patients

The Institutional Research Committee of Kurume University Hospital approved the study and waived the need to obtain informed consent because this study was retrospective and observational. All consecutive patients who underwent emergency open repair of a ruptured AAA or impending rupture of the AAA between January 1, 2005 and June 30, 2010 at our hospital were included in this study. Subjects were divided into two groups, i.e., survivors and nonsurvivors. Nonsurvivors were defined as patients who died in hospital within 30 days of surgery, including intraoperative deaths.

### Data collection

All charts and anesthetic records of the patients were reviewed to collect the following variable factors: age, gender, preoperative levels of hemoglobin (Hb), hematocrit (Ht), platelets (Plts), base excess (BE), and serum glucose and lactate measured just after the induction of anesthesia, presence of shock or hypotension defined as a systolic blood pressure <80 mmHg, whether AAA was ruptured including sealed ruptured or impending determined by the laparotomic findings, the incidence and volume of preoperative transfusion, interval from admission to the hospital or arrival in the operating room until aortic cross-clamping, surgical duration, and intraoperative volume of blood loss and transfusion, total fluid infusion, and urine output. With regard to transfusion, in addition to the total amount, data on blood components, i.e., red cell concentrates (RCC), fresh-frozen plasma (FFP), platelet concentrates (PC), and plasma protein fractions (PPF) were also collected. The differences in these factors were then compared between groups to determine the impact of these factors on mortality.

### Statistical analysis

Data were analyzed using the SAS statistical software package (SAS Institute, Cary, NC, USA). The Shapiro–Wilk test was used to examine whether continuous variables were normally distributed. Variables with a normal distribution were expressed as mean ± standard deviation and analyzed using Welch's or Student's $t$ test; variables not normally distributed were noted as median (interquartile range), and analyzed by exact Wilcoxon rank-sum test. Because only two patients received a preoperative transfusion, statistical comparison of the volume transfused preoperatively was not performed. Fisher's exact test was employed for the categorical data to compare differences in values between the groups. A value of $P < 0.05$ was considered significant.

## Results

A total of 244 patients underwent open repair of AAAs during the period studied: 202 were elective and the others emergent. Therefore, 42 patients were enrolled in this study. Of those, 9 patients died within 30 postoperative days, including 2 intraoperative deaths, whereas 33 survived and were discharged from hospital, indicating a 30-day mortality rate of 21.4%.

The preoperative characteristics of patients classified as survivors and nonsurvivors are presented in Table 1. Of the nine nonsurvivors, eight (88.9%) had shown persistent preoperative shock ($P = 0.0004$ vs. 21.2% of survivors). Of note, compared with survivors, nonsurvivors were significantly older ($P = 0.0052$), had lower levels of Hg/Ht ($P < 0.0001$), Plts ($P = 0.0003$), and BE ($P < 0.0001$), higher level of lactate ($P = 0.0048$), and a higher incidence of transfusion of RCC (55.6% vs. 6.1%, $P = 0.0026$) and PPF (77.8% vs. 24.2%, $P = 0.0055$). Correction of metabolic acidosis was attempted intraoperatively with intravenous sodium bicarbonate in both groups. There were no significant differences between the groups in gender distribution, level of serum glucose, and whether the AAA was ruptured or imminent.

Table 2 shows intraoperative variables in both groups, demonstrating that nonsurvivors compared with survivors had a shorter interval from admission to the hospital ($P = 0.0459$) or arrival in the operating room ($P = 0.0288$) until aortic cross-clamping occurred, greater blood loss ($P = 0.0038$), larger amounts of transfusion [total ($P = 0.0083$), RCC ($P = 0.0056$), and PPF ($P = 0.0038$)], and less urine output ($P = 0.0004$). There were no significant differences between the groups with regard to surgical duration and volumes of FFP and PC transfused.

In both patient groups, general anesthesia was induced with intravenous propofol, thiopental, midazolam, or ketamine, and maintained with inhalation of sevoflurane or total intravenous anesthesia using propofol with remifentanil and/or fentanyl, using muscle relaxants such as vecuronium and rocuronium. Perioperatively, the following inotropes were similarly used in both survivors and nonsurvivors as needed: dopamine, ephedrine, etilefrine,

**Table 1** Preoperative characteristics of survivors versus nonsurvivors

| Variable | Survivors ($n = 33$) | Nonsurvivors ($n = 9$) | $P$ value |
|---|---|---|---|
| Age (years) | 74.0 ± 9.0 | 81.0 ± 5.0 | 0.0052 |
| Gender (male/female) | 28/5 | 6/3 | 0.3364 |
| Shock (%) | 21.2 | 88.9 | 0.0004 |
| Hemoglobin (g/dl) | 9.3 ± 2.0 | 5.8 ± 2.5 | <0.0001 |
| Hematocrit (%) | 27.6 ± 6.1 | 17.1 ± 7.0 | <0.0001 |
| Platelets ($10^3$/μl) | 156 [128–182] | 36 [13–43] | 0.0003 |
| Base excess (mmol/l) | −1.1 [−4.4 to −0.2] | −15.9 [−21.0 to −12.7] | <0.0001 |
| Glucose (mg/dl) | 160 [130–203] ($n = 32$) | 122 [87–253] | 0.2453 |
| Lactate (mmol/l) | 1.8 [1.2–3.7] ($n = 31$) | 7.3 [3.9–10.1] | 0.0048 |
| Ruptured AAA (%) | 75.8 | 88.9 | 0.6551 |
| RCC transfused (%) | 6.1 | 55.6 | 0.0026 |
| FFP transfused (%) | 0 | 11.1 | 0.2143 |
| PC transfused (%) | 0 | 0 | – |
| PPF transfused (%) | 24.2 | 77.8 | 0.0055 |
| Total volume of transfusion (ml) | 560 [250–750] ($n = 9$) | 1250 [750–2,650] ($n = 7$) | 0.0690 |
| Volume of PPF transfused (ml) | 688 ± 477 ($n = 8$) | 964 ± 488 ($n = 7$) | 0.2875 |

Data are presented as mean ± standard deviation, number, or median [interquartile range]

*AAA* abdominal aortic aneurysm, *RCC* red cell concentrates, *FFP* fresh-frozen plasma, *PC* platelet concentrates, *PPF* plasma protein fractions

**Table 2** Intraoperative variables in survivors versus nonsurvivors

| Variable | Survivors ($n = 33$) | Nonsurvivors ($n = 9$) | $P$ value |
|---|---|---|---|
| Time from admission to aortic clamping (min) | 222 [181–269] | 141 [117–242] ($n = 8$) | 0.0459 |
| Time from arrival in operating room to aortic clamping (min) | 116 [82–152] | 40 [34–94] ($n = 8$) | 0.0288 |
| Surgical duration (min) | 287 ± 68 | 236 ± 101 | 0.1868 |
| Blood loss (g) | 2,120 [1,240–3,270] | 6,310 [3,315–9,810] | 0.0038 |
| Total volume of transfusion (ml) | 3,170 [2,100–6,220] | 7,160 [6,580–13,980] | 0.0083 |
| RCC transfused (%) | 93.9 | 88.9 | 0.5247 |
| Volume of RCC transfused (ml) | 1,680 [1,120–3,080] ($n = 31$) | 4,060 [3,640–5,600] ($n = 8$) | 0.0056 |
| FFP transfused (%) | 72.7 | 88.9 | 0.4164 |
| Volume of FFP transfused (ml) | 880 [480–1,800] ($n = 24$) | 2,280 [1,200–4,200] ($n = 8$) | 0.0582 |
| PC transfused (%) | 27.3 | 55.6 | 0.1326 |
| Volume of PC transfused (ml) | 380 [200–400] ($n = 9$) | 400 [200–800] ($n = 5$) | 0.4630 |
| PPF transfused (%) | 90.9 | 88.9 | 1 |
| Volume of PPF transfused (ml) | 1,000 [750–1,750] ($n = 30$) | 3,000 [1,500–3,875] ($n = 8$) | 0.0038 |
| Volume of total fluid infusion (ml) | 2,522 ± 880 | 2,011 ± 1,048 | 0.1457 |
| Volume of urine output (ml) | 615 [310–900] | 10 [2–20] | 0.0004 |

Data are presented as median [interquartile range], mean ± standard deviation, or number

*RCC* red cell concentrates, *FFP* fresh-frozen plasma, *PC* platelet concentrates, *PPF* plasma protein fractions

adrenaline, and noradrenaline. Differences in the use and doses of these agents between the groups were not compared. Surgical procedure was Y-graft replacement by midline laparotomy in all patients, excluding the 2 intraoperative deaths. The properties of aneurysms were degenerative or atherosclerotic in 38 patients and inflammatory in 4 patients.

Details of the nonsurvivors are summarized in Table 3. Most patients with ruptured AAAs were classified into Rutherford levels 3 and 4, indicating that they were in severe shock [22]. Nonsurvivors had comorbidities such as bronchial asthma, hypertension, hyperlipidemia, hyperuricemia, disseminated intravascular coagulation, gastrointestinal hemorrhage, chronic kidney disease, coronary

**Table 3** Details of nonsurvivors

| Number | Age (years) | Sex | Rutherford level | Comorbidity | POD of death | Cause of death |
|---|---|---|---|---|---|---|
| 1 | 83 | M | 4 | Unknown | 0 | Hemorrhagic shock |
| 2 | 76 | M | 4 | Bronchial asthma | 0 | Hemorrhagic shock |
| 3 | 73 | M | Unruptured | HT, hyperlipidemia, hyperuricemia | 2 | Hemorrhagic shock |
| 4 | 82 | F | 4 | Unknown | 0 | Hemorrhagic shock |
| 5 | 75 | F | 3 | Bronchial asthma, DIC, GI hemorrhage | 11 | Sepsis, multiple organ failures |
| 6 | 83 | M | 3 | HT, hyperuricemia, CKD | 15 | Sepsis, multiple organ failures |
| 7 | 86 | F | 4 | None | 13 | Acute renal failure |
| 8 | 86 | M | 4 | HT, DIC | 0 | Hemorrhagic shock, sepsis |
| 9 | 85 | M | Undefined | HT, coronary spasms, DIC, acidosis | 0 | Hemorrhagic shock |

*POD* postoperative day, *M* male, *F* female, *HT* hypertension, *DIC* disseminated intravascular coagulation, *GI* gastrointestinal, *CKD* chronic kidney disease

spasms, and metabolic acidosis. The suggested causes of deaths included hemorrhagic shock, sepsis, multiple organ failure, and acute renal failure.

## Discussion

The overall mortality for ruptured AAAs has been reported to vary between 85% and 95%, showing that the majority of patients die without ever reaching hospital; of those who reach the hospital alive and undergo emergency surgery, between 20% and 70% die [1–10, 23, 24]. In our series, the 30-day mortality rate was 21.4%. These persistently poor results, in the face of low mortality rates associated with elective surgery, have prompted many authors, including us, to predict the risk factors influencing surgical outcome of ruptured AAAs.

The main finding of our study is that predictors for perioperative death in patients undergoing emergency open repair of AAAs are older age, more severe anemia and thrombocytopenia, a higher incidence of persistent shock with metabolic acidosis and an elevated level of serum lactate, greater necessity to transfuse RCC and PPF, and greater intraoperative hemorrhage, inevitably associated with larger amounts of total, RCC, and PPF transfusions and reduced urine output. Many other authors have reported that the factors predicting nonsurvival were older age, female gender, persistent hypotension, massive bleeding and transfusion, anemia, acidosis, increased creatinine level, hypothermia, pacemaker implanted, long duration of surgery and aortic clamping, and prolonged interval from admission to hospital until surgery [11–21]. Our observations agree in part with those of other investigations. Although this was a retrospective study notwithstanding, the authors suggest that obstinate shock or hypotension caused by massive hemorrhage with consequent serious anemia, metabolic acidosis, and diminution in urine output might be the particularly crucial prognostic factor increasing the mortality rate for patients with ruptured AAAs. It should be kept in mind that emergency AAA presenting without hemodynamic shock could be associated with misdiagnosis and delay in appropriate management, leading to an increased risk of significant rupture, hemodynamic decompensation, and poor outcome [25]. Correct diagnosis and treatment before the development of shock may be decisively important to reduce mortality.

It is certainly established that the serum lactate concentration increases during hemorrhagic shock with peripheral hypoperfusion. Singhal et al. [26] demonstrated that an immediate postoperative serum lactate $\geq$4.0 mmol/l and base deficit $\leq -7$ mmol/l were good predictors of outcome after ruptured AAA repair. Considering our observation that preoperatively increased serum lactate discriminated between survivors and nonsurvivors, continuously elevated levels of lactate might be critical in emergency surgery for ruptured AAAs.

As shown in Table 2, we demonstrated a shorter interval between admission to the hospital or arrival in the operating room and aortic clamping in nonsurvivors, which might indicate that nonsurvivors were in a higher state of emergency. On the other hand, in our cases, the time from admission to aortic clamping for ruptured AAAs seems to be quite long, suggesting that most of the AAAs in our patients would have been sealed or impending rupture on admission and then developed into rupture during the course from admission to laparotomy. We enrolled in this study only patients who would be scheduled to undergo emergency surgery for AAAs.

To save patients with ruptured AAAs, it is absolutely imperative to perform surgery as soon as possible. Of further interest, Dueck et al. noted that high-volume surgeons with subspecialty training conferred a significant

survival benefit for patients undergoing ruptured AAA repair [18]. Each hospital needs further improvement to streamline the system for accepting such emergency patients and to establish a closer network of affiliated institutions. Our goal is to achieve expeditious admission of patients to improve therapeutic outcomes.

The relationship of perioperative mortality of ruptured AAAs to medical comorbidities such as cardiac arrest, low cardiac function, arrhythmia, disturbance of consciousness, acute renal failure, coagulopathy, intestinal ischemia, multiple organ dysfunction, and chronic obstructive pulmonary disease has been reported [2–9, 11–15, 17–20]. To our regret, we did not analyze the preexisting complications and preoperative organ dysfunctions in our patients because the sample number seemed to be too small to compare these data between the two groups, and the descriptions of those features on the charts may have been insufficient.

As concerns blood transfusion, it has been reported that intraoperative erythrocyte transfusion is associated with a higher risk of mortality and morbidity of pulmonary, septic, wound, or thromboembolic complications in surgical patients with severe anemia [27]. Whether this association results from the adverse effects of transfusion or is the result of increased blood loss in the patients receiving blood remains to be elucidated. However, some nonsurvivors in our study died of sepsis, multiple organ failures, or acute renal failure (see Table 3).

Recently, certain issues related to treatment of ruptured AAAs have been discussed: one is abdominal compartment syndrome (ACS) and another is endovascular aneurysm repair (EVAR). Abdominal compartment syndrome is defined as an intraabdominal pressure >12 mmHg or an abdominal perfusion pressure <60 mmHg in the presence of end-organ dysfunction, ultimately leading to multiorgan failure [28]. Pertinent postoperative wound closure may have an important role in prevention of ACS, which would otherwise be treated by decompression such as negative pressure dressings. Makar et al. [29] demonstrated that in patients with ruptured AAAs, emergency EVAR compared with emergency conventional open repair is less stressful, reduces blood loss, requires less transfused and total intraoperative intravenous fluid infusion, and has a lower systemic inflammatory response syndrome score and lower lung injury score, in association with less intraabdominal hypertension. Within the limitations of the data published to date, EVAR as the primary treatment for ruptured AAA is achievable and seems to be associated with favorable mortality over open repair when cases are appropriately selected [30–32]. In our hospital, EVAR for AAAs was started in July 2006, and 91 patients had received EVAR by June 30, 2010. Further investigations into the advantage of EVAR over open repair to achieve a better outcome in patients undergoing emergency surgery for ruptured AAAs may be needed.

Finally, to improve the surgical mortality of ruptured AAAs, it might be of great importance to correct preoperative shock and anemia caused by massive bleeding before the onset of hemodynamic deterioration.

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