

Original Article

Open Surgical Decompression Is Useful for the Prevention and Treatment of Abdominal Compartment Syndrome after the Repair of Ruptured Abdominal Aortic and Iliac Artery Aneurysm

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Objective: This study was performed to determine whether open surgical decompression (OSD) decreased the mortality associated with abdominal compartment syndrome (ACS) following open repair (OR) of ruptured abdominal aortic aneurysm and iliac aneurysm (rAAA), and to investigate the risk factors associated with OSD.

Material and Methods: Total 113 consecutive patients with rAAA underwent OR in our institution. Ninety patients underwent primary abdominal closure; however, three of them developed ACS and required OSD. Prophylactic OSD was performed at the initial OR in 23 patients.

Results: The in-hospital mortality rate was higher in those who underwent OSD than in those who did not undergo OSD [27.0% (7/26) vs. 6.9% (6/87), respectively; $p=0.01$]. However, no ACS-related death occurred in the OSD group. Multivariate analyses revealed that a preoperative/intraoperative base excess (BE) <-11 [$p=0.045$; odds ratio (OR), 3.33; 95% confidence interval (CI), 1.021–10.850], performance of left thoracotomy ($p=0.038$; OR, 5.17; 95%CI, 1.098–24.357), and intraoperative blood transfusion $>1,800$ mL ($p=0.012$; OR, 4.30; 95%CI, 1.386–13.322) were associated with OSD.

Conclusion: The prevalence and mortality rates of ACS were low at our institution. OSD is considered to be useful for the prevention and treatment of ACS after repair of rAAA. OSD should be considered in patients with the above-mentioned factors.

Keywords: ruptured abdominal aortic artery aneurysm, ruptured abdominal iliac artery aneurysm, abdominal compartment syndrome, open surgical decompression, delayed abdominal wound closure

Introduction

The presence of massive intestinal edema and/or a large intra-abdominal hematoma following repair of a ruptured abdominal aortic and iliac artery aneurysm (rAAA) may increase the intra-abdominal pressure (IAP),^{1,2)} which may result in the development of abdominal compartment syndrome (ACS), a fatal complication of rAAA repair. Although the reported prevalence of ACS in open repair (OR) ranges from 4% to 20%, the mortality rate associated with ACS is 80%–100% when inappropriately treated.^{2–7)} Open surgical decompression (OSD) is considered to be an effective lifesaving procedure for preventing and treating this fatal complication.^{6–9)} We reviewed 113 patients who underwent OR for rAAA in our institute and assessed the usefulness of and the risk factors associated with OSD.


Patients and Methods

This retrospective observational study was approved by our institutional review board, and the need for informed patient consent was waived.

From January 2006 to December 2016, 113 patients with rAAA were managed in our institution (mean age, 75.1 ± 8.7 years; 84 men and 29 women). Abdominal pain and/or back pain were the main symptoms at onset (93/113; 82.3%). Preoperative shock (systolic blood pressure <90 mmHg) was observed in 55 (48.7%) patients, and three patients (2.7%) required cardiopulmonary resuscitation. Fifteen patients (13.3%) presented with syncope. Aneurysm rupture was confirmed using computed tomography in all patients, and emergency operations

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Received: September 19, 2017; Accepted: January 15, 2018
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were performed immediately following the diagnosis.

Surgical procedure

All patients underwent laparotomy. Twenty-six patients required suprarenal aortic cross-clamping. In addition, 11 of the 26 patients required descending aortic cross-clamping with left thoracotomy before the laparotomy owing to hemodynamic instability caused by massive bleeding. Fourteen of the 26 patients required suprarenal abdominal aortic cross-clamping with laparotomy without left thoracotomy due to a juxtarenal abdominal aortic aneurysm. One patient with hemodynamic instability required balloon occlusion before laparotomy owing to a shaggy descending aorta for which descending aortic cross-clamping with left thoracotomy could not be performed. The other 87 of 113 patients underwent infrarenal cross-clamping with laparotomy.

Measurement of IAP and diagnosis of ACS

Postoperatively, IAP was immediately and intermittently measured as the urinary bladder pressure through a Foley catheter. We diagnosed ACS as per the criteria established by the World Society of the Abdominal Compartment Syndrome¹; ACS was defined as a sustained IAP >20 mmHg associated with new organ dysfunction.

Our protocol for OSD

In our institute, we perform OSD at the time of the initial rAAA repair to prevent ACS when abdominal wall closure is challenging due to excessive bowel edema and/or excessive retroperitoneal hematoma formation. In cases where the abdominal wall can be closed at the time of initial repair, the IAP is carefully monitored postoperatively, and once the IAP is >20 mmHg with organ dysfunction, such

as renal failure or circulatory insufficiency,³ OSD is immediately performed. This provides considerable potential for further intra-abdominal organ edema without clinically relevant IAP elevation. During OSD or after delayed abdominal wound closure, the IAP was closely monitored to ensure that all incidences of recurrent ACS were detected.

After abdominal decompression, we used a vacuum-assisted wound closure system (V.A.C. Abdominal Dressing System; KCI, San Antonio, TX, USA) for delayed abdominal closure. In our institute, we regularly suture only a vinyl sheet to the abdominal skin and cover the wound using the V.A.C. system. Further, we usually exchange the sheet every 3–5 days to prevent infectious complications (Figs. 1A and 1B).

Delayed abdominal wound closure

We performed delayed abdominal wound closure when the patient's hemodynamic status stabilized and the IAP was <20 mmHg and not expected to increase following delayed abdominal wound closure. After delayed abdominal wound closure, the hemodynamic status and IAP were closely evaluated to appropriately detect ACS recurrence.

Statistical analyses

Continuous variables of patients who did (OSD group) and did not (non-OSD group) undergo OSD were compared using the Mann–Whitney U test. Categorical variables are presented as counts; between-group differences were assessed using Fisher's exact test. Multiple regression analyses were performed for variables that were statistically significant in the univariate analysis. A p-value <0.05 was considered statistically significant. Statistical analyses were performed with SPSS version 22 (IBM Corp., Armonk, NY, USA).

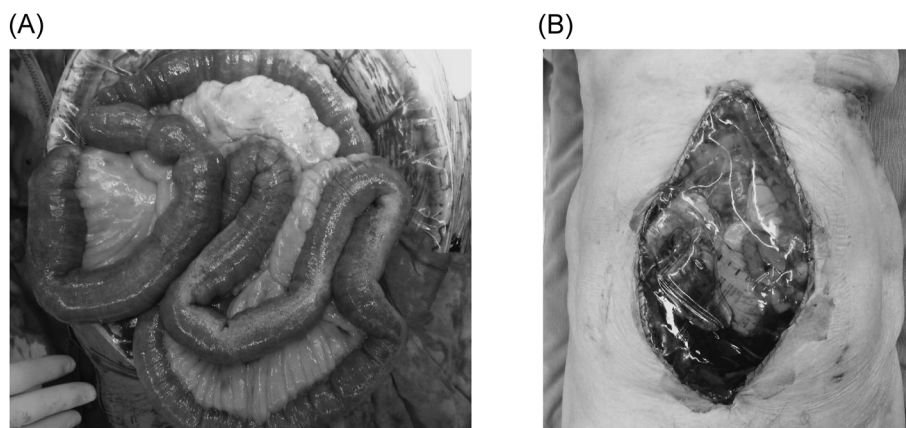
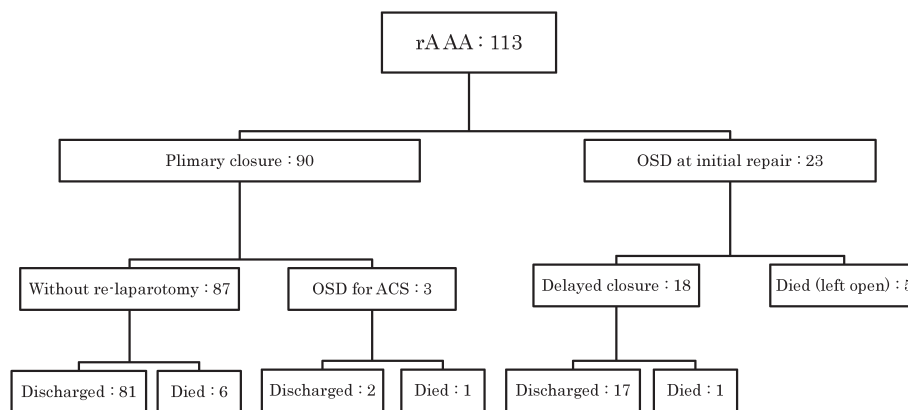


Fig. 1 An 80-year-old woman with open surgical decompression after open repair for ruptured abdominal aortic aneurysm. **(A)** Open surgical decompression was performed at the time of initial repair owing to excessive bowel edema. **(B)** Postoperative day 6. Patient's hemodynamic status was stable and bowel edema reduced. Delayed abdominal closure was performed, and she was discharged without recurrence of ACS.

Table 1 Comparison of the patient characteristics before and after the operation

	OSD (n=26)	Non-OSD (n=87)	p-value
Age (years, median)	77 (range, 46–92)	76 (range, 35–89)	0.54
Male	22 (84.6%)	62 (71.3%)	0.13
BMI (kg/m ² , median)	23.4 (range, 19.5–31.1)	23.3 (range, 15.6–32)	0.75
Preoperative shock (systolic blood pressure of <90 mmHg)	19 (73.1%)	36 (41.4%)	0.004
Preoperative syncope	9 (34.6%)	24 (27.6%)	0.32
Preoperative intubation	4 (15.3%)	4 (4.6%)	0.08
Preoperative CPR	1 (3.8%)	2 (2.3%)	0.55
Pre/intra-operative minimum Hb (g/dL, median)	6.5 (range, 3.1 to 10.2)	7.6 (range, 1.7–13.3)	0.06
Pre/intra-operative minimum BE (median)	-13.5 (range, -4.4 to -29)	-8.6 (range, -1.5 to -24.5)	<0.01
Left thoracotomy	6 (23.1%)	5 (5.7%)	0.017
Supra renal aortic cross clamping	10 (38.5%)	16 (18.4%)	0.98
Aortic occluding balloon	1 (3.8%)	0	0.24
Intra-operative blood transfusion (mL, median)	2,540 (range, 980–13,440)	1,400 (range, 0–6,440)	<0.01
Concomitant operation	1 (3.8%)	4 (4.6%)	0.68
Omentopexy	0	2 (2.3%)	
Thrombectomy in the lower extremities	1 (3.8%)	1 (1.1%)	
Femoro-femoral bypass	0	1 (1.1%)	
Operation time (min, median)	254 (range, 178–600)	222 (range, 69–438)	0.043

OSD: open surgical decompression; BMI: body mass index; CPR: cardiopulmonary resuscitation; Hb: hemoglobin; BE: base excess



rAAA: ruptured abdominal aortic aneurysm and iliac artery aneurysm, ACS: Abdominal compartment syndrome, OSD: Open surgical decompression

Fig. 2 Diagram of the study patients.

Results

Total 110 patients underwent prosthesis graft replacement, and two patients with a ruptured internal iliac artery aneurysm underwent only aneurysm resection. One patient who died on postoperative day 1 underwent only closure of the ruptured tear due to intraoperative cardiopulmonary arrest and the inability to resuscitate the patient. Concomitant operations were performed in five patients. Omentopexy was performed in two patients (one with a ruptured infectious abdominal aortic aneurysm and another with a duodenal perforation owing to the rupture of an abdominal aortic aneurysm into the duodenum). Thrombectomy in the lower extremities was

performed for two patients. Femorofemoral bypass was performed for one patient. The patients' preoperative and intraoperative clinical profiles are presented in **Table 1**. Preoperative shock (systolic blood pressure <90 mmHg), low base excess (BE), performance of left thoracotomy, and higher intraoperative blood transfusion volume were significantly more common in the OSD group than in the non-OSD group; moreover, the operation time was significantly longer in the OSD group. A flow diagram of the study patients is displayed **Fig. 2**.

Primary abdominal closure was performed at the time of rAAA repair in 90 of the 113 patients (79.6%), whereas three of these 90 patients (3.3%) developed ACS and underwent OSD. The cause of ACS was bleeding and

Table 2 Mortality and morbidity

	OSD (n=26)	Non-OSD (n=87)	p-value
Hospital mortality	7 (26.9%)	6 (6.9%)	0.01
Sepsis due to graft infection	1	0	
Cerebral infarction	1	0	
Pneumonia	1	0	
Bowel ischemia	2	2	
MOF	2	4	
Morbidity			
Graft infection	1	0	
Spinal cord injury	1	2	
Hypoxic encephalopathy	1	0	
Bleeding (redo surgery requiring)	4	1	
Wound infection	2	3	
Bowel ischemia	2	0	
MOF	2	3	
Cerebral infarction	1	0	
Ileus	1	3	
Respiratory failure	3	3	
Leg artery embolism	0	1	
Acute renal failure	0	3	
Wound herniation	0	1	
Peritonitis due to appendicitis	0	1	
Angina pectoris	0	1	
Duodenal ulcer	0	1	
Hospital stay (day, median)	26.5 (range, 0–62)	15 (range, 0–88)	<0.01

Respiratory failure includes pneumonia, aspiration, and tracheotomy. Spinal cord injury includes paraplegia, urinary, and rectal incontinence. MOF: multiple organ failure

massive intra-abdominal hematoma formation in two patients and excessive bowel edema in one patient. All three patients underwent OSD 1 day following rAAA repair. Prior to the OSD, all three of these patients had presented with hypotension, oliguria, and an intrabdominal pressure >20 mmHg. All three patients underwent delayed abdominal wound closure after a median duration of 9 days (range, 8–17 days) without ACS recurrence. While two patients achieved wound closure without complication, one patient required 17 days to achieve wound closure in two stages owing to insufficient reduction in the bowel edema. Two patients were discharged without other complications, whereas one patient died of sepsis caused by a prosthetic vascular graft infection. In 23 of 113 patients (20.4%), prophylactic OSD was performed at the time of the initial rAAA repair owing to excessive bowel edema and/or the development of a huge retroperitoneal hematoma. Five of these 23 patients (21.7%) died before abdominal wound closure due to extensive bowel necrosis (n=2), multiple organ failure (n=2), and extensive cerebral infarction (n=1). Eighteen of the 23 patients (78.3%) achieved wound closure after a median duration of 6 days (range, 4–8 days). Of these 18 patients, 1 (5.6%) died of pneumonia on postoperative day 42; this patient

had developed hypoxic encephalopathy postoperatively and required a tracheotomy. Seventeen patients were discharged or transferred to another hospital without ACS recurrence. The in-hospital mortality rate was 27.0% (7/26) in the OSD group and 6.9% (6/87) in the non-OSD group (p=0.01). The in-hospital mortality and morbidities are presented in Table 2.

Risk factors for OSD

The following parameters were analyzed as the possible risk factors of OSD: age, sex, body mass index, preoperative syncope, preoperative endotracheal intubation, vital signs of shock (systolic blood pressure <90 mmHg) at presentation, low preoperative/intraoperative hemoglobin, low preoperative/intraoperative BE, performance of left thoracotomy, suprarenal abdominal aortic cross-clamping, high intraoperative blood transfusion volume, and long operation time. The univariate analyses showed that preoperative shock (systolic blood pressure <90 mmHg) (73.1% vs. 41.4%, p<0.01), preoperative/intraoperative BE ≤ -11 (73.1% vs. 32.2%, p<0.01), left thoracotomy (23.1% vs. 5.7%, p<0.01), operation time >210 min (80.8% vs. 59.8%, p=0.049), and intraoperative blood transfusion >1,800 mL (76.9% vs. 33.3%,

Table 3 Multivariate analysis of the risk factors for OSD

	Univariate analysis			Multiple regression analysis		
	Number of patients	Number of OSD patients	p-value	p-value	OR	95%CI
Preoperative shock: yes	55	19	0.004	0.75	1.22	0.364–4.071
Pre/intra-operative minimum BE<-11	47	19	p<0.01	0.045	3.33	1.021–10.850
Left thoracotomy: yes	11	6	0.017	0.038	5.17	1.098–24.357
Intra-operative blood transfusion >1,800mL	49	20	p<0.01	0.012	4.3	1.386–13.322
Operation time >210 min	73	21	0.039	0.21	2.2	0.657–7.345

OSD: open surgical decompression; OR: odds ratio; CI: confidence interval; BE : base excess

$p < 0.01$) had a significantly stronger association with the OSD group than with the non-OSD group.

A receiver operating characteristic curve was generated, and the area under the curve (AUC) and its 95% confidence interval (CI) were calculated to determine the best discriminating level of the preoperative/intraoperative BE, intraoperative blood transfusion, and operation time obtained on admission for predicting the need for performing OSD. The receiver operating characteristic curve analysis confirmed that a BE of -11 (AUC, 0.737; 95%CI, 0.631–0.844), intraoperative blood transfusion of 1,800 mL (AUC, 0.718; 95%CI, 0.607–0.829), and operation time of 210 min (AUC, 0.605; 95%CI, 0.487–0.723) were the best diagnostic cut-off values that indicated the need for OSD.

A multiple regression analysis was performed, including the variables that were significantly related to OSD in the univariate analysis. Further analysis showed that a preoperative/intraoperative $BE \leq -11$ [$p = 0.045$; odds ratio (OR), 3.33; 95%CI, 1.021–10.850], performance of left thoracotomy ($p = 0.038$; OR, 5.17; 95%CI, 1.098–24.357), and intraoperative blood transfusion $> 1,800$ mL ($p = 0.012$; OR, 4.30; 95%CI, 1.386–13.322) were independently correlated with OSD performance. The results of statistical analyses are shown in Table 3.

Discussion

Owing to the advancements in the diagnostic and surgical techniques as well as perioperative care systems, the 30-day mortality rate associated with the OR of rAAA was lower than that reported previously. However, the mortality of both open and endovascular rAAA repair remains high (30%–50%).^{2,4,10–12} Multiple organ failure is a significant cause of mortality, and ACS is considered a cause of multiple organ failure in patients undergoing rAAA repair.^{2,3,5,7} Although the reported ACS prevalence in OR ranges from 4% to 20%, the mortality rate of inappropriately treated ACS ranges from 80% to 100%, and delayed treatment is associated with increased morbidity and mortality.^{2–7,13} Therefore, the treatment or prevention

of ACS will contribute to lower rAAA mortality.^{6–9} Decompressive laparotomy (i.e., OSD) is considered effective for preventing and treating the fatal complications of ACS. Several surgeons have reported that OSD enabled a reduction in the early mortality and overall mortality among patients undergoing rAAA repair.^{4,6–8,14} According to our treatment protocol, the ACS prevalence was 2.7% (3/113) in the present study, lower than that reported in previous trials (4%–20%).^{2–7,13} Further, no ACS-related deaths occurred in our study. Therefore, we believe that OSD contributes to reduced mortality in patients with ACS and helps prevent ACS in those undergoing rAAA repair. In fact, in the present study, the mortality rate in the OSD group was 27.0% (7/26), significantly higher than that in the non-OSD group (6.9%, 6/87; $p = 0.01$). We attribute this result to the more distressing preoperative status and the higher expected operative risk in the OSD group than in the non-OSD group (see Table 1).

Sörelis et al.⁴ reported that the in-hospital mortality rate among patients who underwent decompressive laparotomy after ACS development was significantly higher in those who required OSD immediately following the primary surgery (62% vs. 22%, respectively). Therefore, we believe that it is important to perform prophylactic treatment and identify the patients who require OSD. Several studies have revealed the OSD risk factors. Carr et al. reported that the independent risk factors for ACS development include massive fluid resuscitation, multiple transfusions, hypothermia, base deficit/acidosis, and a high body mass index.⁸ In the present study, the risk factors leading to the requirement of OSD were a preoperative/intraoperative $BE < -11$, the performance of left thoracotomy, and intraoperative blood transfusion $> 1,800$ mL. Patients fulfilling these conditions are considered to be in a distressing condition, often requiring massive fluid or blood administration. These factors can lead to bowel edema and/or intra-abdominal (retroperitoneal) hematoma formation that further increases the IAP and ACS development.¹² Therefore, for ACS prevention, we recommend that surgeons consider OSD following rAAA repair in patients with these conditions.

Another consideration is the optimal timing for performing delayed abdominal wound closure after OSD. Several surgeons have reported that a longer OSD duration is associated with a greater risk of developing infectious complications, including graft infection and enteroatmospheric fistula.^{4,14} Abdominal wall closure was performed at a median duration of 6 days (range, 4–17 days) after initial OSD, similar to that in other reports (range, 4–10.5 days). Moreover, no patients developed ACS recurrence, and the frequency of infectious complications was low. Only one patient died because of sepsis caused by prosthetic vascular graft infection. Based on these results, we believe that our timing for delayed abdominal wound closure was appropriate.

This study has certain limitations. First, this was a non-randomized and retrospective study that did not include a control group. The decision to perform OSD depended on several surgeons; therefore, there may have been a bias in the selection of patients for OSD performance. Thus, we could not draw a concrete conclusion regarding the appropriateness of OSD. Second, this study involved a small group of patients treated with OSD in a single institution. A large-scale multicenter study is warranted to obtain a full understanding of the effectiveness of OSD in the treatment and prevention of ACS. Third, this study includes data collected over a 10-year period. During this time, there may have been advancements in the surgical techniques and perioperative management protocols that may have influenced the outcomes.

Conclusion

OSD is a safe procedure for patients with ACS due to rAAA. Patients who had preoperative/intraoperative BE < -11, left thoracotomy, and intraoperative blood transfusion > 1,800 mL required OSD more frequently.

Disclosure Statement

The authors have no conflicts of interest to disclose.

Author Contributions

Study conception: AK

Data collection: AK

Analysis: AK

Investigation: AK

Writing: AK, MY

Funding acquisition: none

Critical review and revision: all authors

Final approval of the article: all authors

Accountability for all aspects of the work: all authors

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