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ORIGINAL ARTICLE

Malperfusion in type A aortic dissection: results of emergency central aortic repair

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Abstract

Background Although outcomes of acute type A aortic dissection (ATAAD) have improved, malperfusion remains associated with high morbidity and mortality rates, and its optimal therapeutic treatment is unknown. Emergency central repair has been performed as our first-line approach for malperfusion. Here, we analyzed outcomes of ATAAD with malperfusion and reassessed emergency central repair.

Methods In total, 1026 ATAAD patients underwent emergency surgery within 48 h of symptom onset, of whom 318 (30.9%) patients complicated with any preoperative malperfusion were included. Pathophysiology of malperfusion and surgical outcomes were analyzed.

Results The in-hospital mortality rate was 12.9% for patients with malperfusion and 4.8% for patients without malperfusion ($p < 0.0001$). Coronary malperfusion was complicated in 7.5% of patients (% dead per group, 19.5%), mesenteric malperfusion in 3.6% (24.3%), renal malperfusion in 8.8% (14.4%), lower leg malperfusion in 12.6% (13.7%), brain malperfusion in 9.7% (12.0%), and spinal malperfusion in 1.1% (18.2%). Mortality rates varied substantially according to the number of affected organ systems (none, 4.8%; one system, 10.4%; two systems, 14.5%; three systems, 30.0%, and four systems; 30.3%; $p < 0.0001$). In malperfused patients, logistic regression analysis revealed that obesity (body mass index > 30 kg/m^2), preoperative shock (systolic blood pressure < 80 mmHg), and visceral ischemia were independent predictors for hospital death.

Conclusions Malperfusion of more organ systems and mesenteric malperfusion resulted in unfavorable prognosis, and effects of central repair were limited in such severe/complex malperfusion. To further improve outcomes of ATAAD with malperfusion, emergency reperfusion of affected organs followed by central repair might be considered.

Keywords Aortic dissection · Aortic operation · Aorta/aortic · Aortic arch · Ischemia · Revascularization

Introduction

Surgical outcomes of acute type A aortic dissection (ATAAD) have been recently improving. According to the annual survey by Japanese Association for Thoracic Surgery, 4953 patients with ATAAD underwent surgical procedures, with an overall hospital mortality of 10.6% in 2014 [1]. Physician awareness of aortic dissection and advances in diagnostic modalities has raised the survival rate of ATAAD. Furthermore, tear-oriented surgical strategies, vital organ protections such as a selective cerebral perfusion with hypothermia, and sealed vascular grafts aimed to improve surgical outcomes. Despite such major efforts, preoperative malperfusion significantly affects early and late survival and thus which needs to be urgently addressed [2–8]. Central aortic repair, direct revascularization of affected arteries, and fenestration have been considered therapeutic options for resolving malperfusion; however, their outcomes are unclear [2–8]. The restoration of blood flow into the true lumen by entry resection contributes to resolving malperfusion, and emergency central repair has been performed as our first-line approach for malperfusion. In this study, we examined outcomes of > 1000 consecutive surgical ATAAD patients, including 318 patients complicated with any preoperative

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malperfusion, and reassessed our emergency central repair strategy.

Patients and methods

Patients

This retrospective observational study was approved by the institutional review board, and informed consent was waived (approval No. A15-222, 12-87). First, data of 1260 consecutive patients who were emergently admitted to Jichi Medical University Hospital and Jichi Medical University, Saitama Medical Center between 1990 and 2016 and treated for TAAD were retrospectively reviewed. One hundred thirty-eight patients in whom onset of ATAAD occurred after 48 h were excluded. Of the remaining 1122 patients, 96 patients were medically treated and thus were excluded. Reasons for exclusion were rejection (10 cases), early thrombosed type (non-communicating aortic dissection/intramural hematoma and an ascending aorta with a diameter < 45 mm) (50 cases), unsuccessful cardiopulmonary resuscitation (15 cases), severe brain damage including comatose patients with mydriasis/no pupillary light reflex or severe brain edema with encephalocele (10 cases), severe dementia (2 cases), and other (9 cases).

The surgical results of 1026 consecutive patients who underwent emergency surgical repair within 48 h of onset of ATAAD were reviewed. Of 1026 consecutive patients, 318 (30.5%) with any type of preoperative malperfusion in one or more organ systems were finally included in the study. The selection process and number of selected patients are shown in Fig. 1.

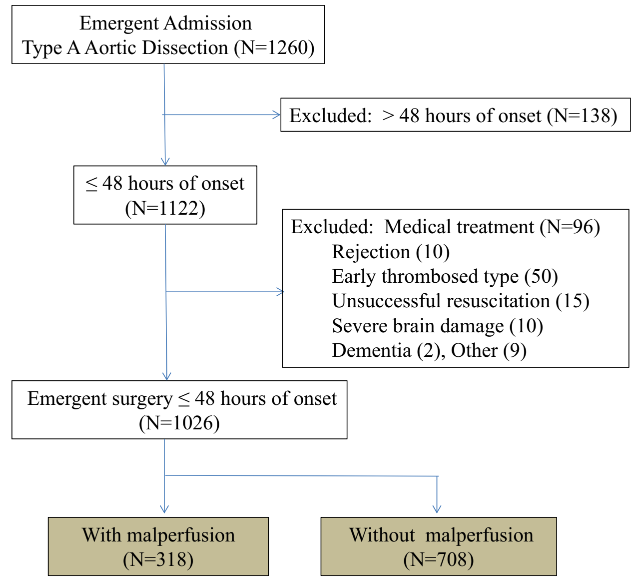

Fig. 1 Selection process and numbers of patients

Methods

Contrast-enhanced computed tomography (CT) and echocardiography were primarily performed for diagnosis. Malperfusion was diagnosed in patients with clinical symptoms and occlusion of the corresponding arteries on diagnostic imaging. Coronary malperfusion was defined based on CT findings, new ST-segment elevation > 0.1 mV, abnormal left ventricular wall motion on echocardiogram, and/or significant elevation in serum creatine kinase levels before surgery (male >248 IU/L, female >153 IU/L). Cerebral malperfusion was defined based on newly developed focal neurological signs with/without CT findings and disorder of consciousness with carotid artery dissection demonstrated by ultrasound. Consciousness disorder caused by shock was diagnosed clinically and excluded. Irreversible deep coma, comatose patients with mydriasis/no pupillary light reflex, or severe brain edema with encephalocele evaluated by CT were considered contraindications to surgery. Regarding spinal cord malperfusion, patients with newly developed bilateral paraplegia and paraparesis not caused by lower leg ischemia were considered to have spinal cord malperfusion. Mesenteric malperfusion was determined based on clinical, radiographic factors, and elevation of biochemical markers, including radiographic evidence of flow obstruction with clinical evidence of ischemia to the abdominal viscera (melena, abdominal pain with distention, etc.). Renal malperfusion was defined based on CT evidence of delayed enhancement, poor contrast enhancement of the entire unilateral kidney, or renal artery dissection together with an increase in creatinine levels. Patients with increased serum creatinine levels only were not included. Lower leg malperfusion was defined based on clinical findings, namely CT evidence of poor contrast enhancement of lower leg arteries, laboratory findings indicating an increase in creatine kinase levels, and clinical findings (loss of femoral pulse and sensation/motor function of the limbs).

After the diagnosis was confirmed, the patient was immediately transferred to the operating room. All patients underwent surgery within 48 h after onset of symptoms. Variables were collected from medical records and preoperative examination records. Surgical variables, in-hospital outcomes, and long-term outcomes were analyzed. In-hospital mortality was defined as death in the hospital. We either examined patients at our outpatient clinic or contacted them by letter or telephone. The mean follow-up period for the entire cohort was 5.0 ± 4.7 years; the follow-up rate was 98.1%.

Surgical procedures

The first-line approach for malperfusion was emergency central repair to eliminate rupture, followed by revascularization of the unresolved malperfused area if necessary. The surgical procedure comprised median sternotomy with standard cardiopulmonary bypass. Surgery was performed under hypothermic circulatory arrest (bladder temperature 20–26 °C). The left subclavian artery, left ventricular apex, and femoral artery were used for arterial cannulation. Myocardial protection was performed with antegrade or retrograde infusion of cold blood cardioplegic solution. All patients underwent circulatory arrest with open distal anastomosis. From 1990 to 2007, deep hypothermic circulatory arrest was used at a bladder temperature of 20 °C for distal procedures for simple ascending aortic replacement. Since 2008, antegrade selective cerebral perfusion or retrograde cerebral perfusion begun to be performed for cerebral protection during circulatory arrest with a bladder temperature of 26 °C. A tear-oriented surgical strategy [9] was employed, and limited ascending/hemiarch replacement was performed for patients in whom the entry site was located in the ascending aorta or DeBakey IIIb retrograde dissection. For patients whose entry site extended to or was located in the aortic arch, total/partial arch replacement was performed. The rate of primary entry resection was 78% (798/1026 cases). Although aortic valves were preserved whenever possible, aortic root replacement was performed when the intimal tear extended to the sinus of Valsalva or when root dilatation associated with annuloaortic ectasia was observed.

Statistical analysis

Continuous variables were compared using the unpaired two-tailed *t* test and were expressed as mean ± standard deviation. Categorical variables were presented as counts, and differences between the groups were assessed by Fisher's exact test. Overall survival was defined as the time from surgery to death from any cause. Time-related survival was estimated using the Kaplan–Meier method and compared by a log-rank test. Multivariate logistic regression modeling was used to determine independent perioperative predictors of in-hospital death. Predictors were entered into a univariate analysis, and any variable with $p < 0.05$ was entered into the multiple logistic regression model. Statistical analysis was performed with Stat View 5.0 (Abacus Concepts Inc., Berkeley, CA), and p values < 0.05 were considered statistically significant.

Results

Patient characteristics

Of 1026 consecutive patients, 318 (30.5%) patients had any type of preoperative malperfusion. Clinical characteristics of the patients with or without malperfusion are presented in Table 1. Patients with malperfusion were younger than those without malperfusion (62 ± 12 vs. 65 ± 13 years, $p < 0.01$). Significantly more patients were men in the malperfusion group than in the no malperfusion group (57% vs. 50%, $p = 0.02$). Patients with preoperative malperfusion had more serious clinical conditions than those without malperfusion. Patients with malperfusion were more likely to have shock, cardiopulmonary resuscitation, and consciousness disorder than those without malperfusion.

The distribution of organs affected by preoperative malperfusion and mortality per group is shown in Table 2. Mortality rates varied substantially according to the number of organ systems affected by malperfusion [none 34/708 (4.8%); one system 24/230 (10.4%); two systems 9/62 (14.5%); three systems 6/20 (30.0%), and four systems 2/6 (30.3%); $p < 0.0001$]. The surgical results of complex malperfusion were poorer than those of simple malperfusion.

Operative details for malperfused patients

Central aortic repair was first attempted, and revascularization for malperfused area was performed if malperfusion persisted after central repair. Regarding proximal reconstruction, modified Bentall/re-implantation was performed in 22 cases, and concomitant aortic valve replacement in eight. Regarding the distal extent of replacement, partial or total arch replacement was performed in 64 patients, and open stent grafting in three.

Of 77 coronary malperfusion, 24 (31%) were resolved by central repair alone. However, 53 cases (69%) required coronary artery bypass grafting after central repair because of the inability to wean from cardiopulmonary bypass. Regarding lower leg ischemia, 110 of 124 patients (89%) were re-vascularized by central repair alone; however, 14 patients (11%) required bypass surgery for lower extremities (aortic bifemoral bypass 3; axillofemoral bypass 5; and femoral–femoral bypass 6). Regarding mesenteric ischemia, iliac–superior mesenteric artery bypass was performed in 3/37 cases (8%), bowel resection in two cases (5%), and exploratory laparotomy in three cases (8%) after central repair.

Table 1 Preoperative characteristics of patients with or without malperfusion before surgery

	Malperfusion no. (%), $N=318$	No malperfusion no. (%), $N=708$	p
Age (years)	62 ± 12	65 ± 13	< 0.01
Male	183 (57)	351 (50)	0.02
Comorbidity			
Obesity (BMI > 30 kg/m^2)	20 (6)	49 (7)	0.8
Marfan syndrome	10 (3)	18 (3)	0.7
Diabetes	16 (5)	50 (7)	0.3
Current smoking	119 (37)	210 (30)	0.02
Ischemic heart disease	8 (3)	30 (4)	0.2
Cerebrovascular disease	22 (7)	64 (9)	0.3
COPD	7 (2)	19 (3)	0.8
Hemodialysis	5 (2)	15 (2)	0.6
Creatinine (mg/dL)	1.2 ± 1.2	1.1 ± 1.2	0.3
Preoperative status			
Shock (SBp < 80 mmHg)	100 (32)	136 (19)	< 0.01
Cardiopulmonary resuscitation	29 (9)	21 (3)	< 0.01
Preoperative ECMO	5 (2)	4 (1)	0.2
Consciousness disorder	86 (27)	70 (10)	< 0.01
Cardiac tamponade	80 (25)	156 (22)	0.3
Severe aortic regurgitation	48 (15)	97 (14)	0.6
Location of the entry tear			
Ascending aorta/Valsalva	199 (63)	445 (63)	0.9
Aortic arch	56 (18)	95 (13)	0.1
Descending aorta or unidentified	63 (19)	168 (23)	0.2

BMI body mass index, *COPD* chronic obstructive pulmonary disease, *SBP* systolic blood pressure, *ECMO* extra-corporeal membrane oxygenation

Table 2 Type of preoperative malperfusion and mortality

Involved organ system	Number ($n=1026$)	Mortality (% dead per group)
Overall[a]	318 (30.5%)	41/318 (12.9%)
Coronary	77 (7.5%)	15/77 (19.5%)
Cerebral	100 (9.7%)	12 /100 (12.0%)
Spinal cord	11 (1.1%)	2/11 (18.2%)
Mesenteric	37 (3.6%)	9/37 (24.3%)
Renal	90 (8.8%)	13/90 (14.4%)
Lower leg	124 (12.6%)	17/124 (13.7%)

[a]One patient frequently had multiorgan malperfusion

In-hospital mortality and complications

The surgical outcomes are presented in Table 3. Of 318 patients with malperfusion of any type, in-hospital mortality occurred in 12.9% of cases (41/318). In contrast, only 4.8% (34/708) of patients who did not have malperfusion died ($p < 0.01$). The mean length of stay in the intensive care unit (10 ± 10 days for malperfusion vs. 8 ± 7 days for no malperfusion; $p = 0.01$) and the total duration of hospitalizations (30 ± 32 days vs. 25 ± 16 days; $p = 0.01$) were significantly different.

The overall complication rate was significantly worse in the malperfusion group than in the no malperfusion group [38.1% (121/318) vs. 16.5% (117/708); $p < 0.01$]. Stroke ($p < 0.01$), paraplegia ($p < 0.01$), postoperative extra-corporeal membrane oxygenation (< 0.01), postoperative hemodialysis/ hemodiafiltration ($p < 0.01$), and mesenteric ischemia ($p < 0.01$) were more frequent in the malperfusion group than in the no malperfusion group.

Causes of hospital mortality of total 75 deaths included heart failure (15), stroke (15), intestinal ischemia (13), multiple organ failure (9), rupture of the downstream aorta (9), bleeding (4), myocardial infarction (3), pneumonia (3), renal/ liver failure (2), lower leg ischemia (1), and unknown (1). No statistically significant differences were found between the groups with respect to causes of hospital mortality.

Risk factors for hospital mortality in ATAAD with malperfusion

The univariate analysis revealed that hospital mortality was negatively affected by obesity [body mass index

Table 3 In-hospital mortality and postoperative complications

	Malperfusion no. (%), $N=318$	No malperfusion no. (%), $N=708$	p
Hospital mortality (%)	41 (12.9%)	34 (4.8%)	< 0.01
ICU stay (days)	10 ± 10	8 ± 7	< 0.01
Hospitalization (days)	30 ± 32	25 ± 16	< 0.01
Complications (postoperative)			
Stroke	50 (15.7)	51 (7.2)	< 0.01
Paraplegia	13 (4.1)	4 (0.6)	< 0.01
Re-exploration	19 (6.0)	28 (4.0)	0.2
Deep sternal wound infection	4 (1.3)	7 (1.0)	0.8
ECMO	20 (6.3)	12 (1.7)	< 0.01
Hemodialysis/hemodiafiltration	39 (12.3)	35 (4.9)	< 0.01
Pneumonia	5 (1.6)	17 (2.4)	0.5
Mesenteric ischemia	16 (5.0)	4 (0.6)	< 0.01
Overall[a]	121(38.1)	117 (16.5)	< 0.01

ICU intensive care unit, *ECMO* extra-corporeal membrane oxygenation

[a]Excluding duplicates

(BMI) > 30 kg/m^2], preoperative shock (systolic blood pressure < 80 mmHg), mesenteric malperfusion, number of organ system affected by malperfusion > 2, concomitant CABG, and cardiopulmonary bypass time > 4 h (Table 4). The multivariable risk analysis for hospital mortality revealed that shock status [$p<0.0001$; odds ratio (OR) 6.124; 95% confidence interval (CI) 2.817–13.316], BMI > 30 kg/m^2 ($p=0.0040$; OR 5.746; 95% CI 1.746–18.909), and mesenteric malperfusion ($p=0.0202$; OR 3.720; 95% CI 1.228–11.2725) were predictors of hospital death (Table 5).

Long-term survival

Forty-nine and 113 remote deaths were observed in the malperfusion and no malperfusion groups, respectively, during follow-up. Causes of 49 remote deaths in the malperfusion group included stroke (9), malignancy (8), dissection (5), myocardial infarction (4), pneumonia (4), heart failure (3), rupture of abdominal aneurysm (2), renal failure (2), intraoperative bleeding during urological surgery (1), aspiration (1), mediastinitis (1), gastrointestinal bleeding (1), decrepitude (1), and unknown (7). While, causes of 113 remote deaths in the no malperfusion group included pneumonia (20), malignancy (20), stroke (14), heart failure (11), rupture of thoracic/abdominal aneurysm (11), decrepitude (9), myocardial infarction (5), renal failure (5), dissection (3), sepsis (3), others (7), and unknown (5).

Actual postoperative survival rates at 1, 3, and 5 years were 93.7% (95% CI 90.8–96.6%), 89.1% (85.0–93.2%), and 84.8% (79.9–89.7%), respectively, in the malperfusion group and 96.7% (95.3–98.1%), 91.9% (89.5–94.3%), and 87.2% (84.1–90.3%), respectively, in the no malperfusion group ($p=0.89$) (Fig. 2). Preoperative malperfusion did not affect the long-term outcomes of operative survivors (Fig. 2).

Discussion

Salvage of patients with malperfusion is essential for further improvement of surgical outcomes of ATAAD. Central aortic repair can resolve malperfusion in most cases of dynamic-type obstruction by restitution of adequate blood flow into the true lumen [6, 10, 11]. During the past 26 years, our baseline strategy for ATAAD has been immediate central repair to eliminate the risk of rupture and improved surgical results of ATAAD in most cases [9]. Malperfusion is reported to be complicating 20–30% of ATAAD cases, and the mortality rate of patients with malperfusion was 17–44% [2–5, 8]. In the current study, 30.5% of patients had any type of preoperative malperfusion, and there was a significant difference in hospital mortality depending on the presence or absence of malperfusion (12.9% vs. 4.8%, $p<0.01$). Especially, mesenteric malperfusion and/or malperfusion of more organ systems resulted in unfavorable prognosis and limited effects of central repair were observed.

Mesenteric malperfusion was observed relatively infrequently, accounting for 6% in German Registry for Acute Aortic Dissection Type A [5, 12] and 3.6% in the current study; however, it is a devastating complication, resulting in a mortality rate of 70–100% [5, 12]. It is reported that immediate central aortic repair is associated with extremely poor prognosis for mesenteric malperfusion [12, 13]. Similarly, in our study, the multivariate analysis identified mesenteric malperfusion as a strong predictor of hospital death, suggesting that central repair alone was

Table 4 Risk factors for hospital mortality of overall malperfusion ($N = 318$) (univariate analysis)

	Hospital death $N = 41$	Survivor $N = 277$	p
Age < 65 years	22 (53.7%)	153 (55.2%)	0.9
Male	26 (63.4%)	157 (56.7%)	0.5
Obesity (BMI > 30 kg/m^2)	6 (14.6%)	14 (5.1%)	0.03
Surgery before 2008	22 (53.7)	162 (58.5)	0.6
Comorbidity			
Marfan syndrome	2 (4.9%)	8 (2.9%)	0.6
Diabetes	4 (9.8%)	12 (4.3%)	0.1
Current smoking	16 (39.0%)	103 (37.2%)	0.9
Ischemic heart disease	2 (4.9%)	6 (2.2%)	0.3
Cerebrovascular disease	2 (4.9%)	20 (7.2%)	0.8
COPD	1 (2.4%)	6 (2.2%)	> 0.99
Hemodialysis	0 (0%)	5 (1.8%)	> 0.99
Serum creatinine, mg/dl	1.1 ± 0.5	1.2 ± 1.3	0.8
Preoperative status			
Shock (SBp < 80 mmHg)	26 (63.4%)	74 (26.7%)	< 0.01
Tamponade	14 (34.1%)	66 (23.8%)	0.2
Neurological deficit	16 (39.0%)	70 (25.3%)	0.1
Severe aortic regurgitation	5 (12.2%)	43 (15.5%)	0.8
DeBakey type I	29 (70.7)	214 (77.3)	0.4
II	1 (2.4)	12 (4.3)	> 0.99
IIIb retrograde	11 (26.8)	51 (18.4)	0.2
Malperfusion			
Coronary	15 (36.6%)	62 (22.4%)	0.053
Mesenteric	9 (22.0%)	28 (10.1%)	0.04
Renal	13 (31.7%)	77 (27.8%)	0.6
Lower leg	17 (41.5%)	107 (38.6%)	0.7
Brain	12 (29.3%)	88 (31.2%)	0.9
Supine	2 (4.9%)	9 (3.2%)	0.6
Number of organ system affected by malperfusion ≥ 2	17 (41.5%)	70 (25.3%)	0.04
Proximal procedures			
Modified Bentall/re-implantation	5 (12.2%)	17 (6.1%)	0.2
Concomitant AVR	2 (4.9%)	6 (2.2%)	0.3
Distal extent of replacement			
Total/partial arch replacement	11 (26.8%)	53 (19.1%)	0.3
Concomitant CABG	13 (31.7%)	41 (14.8%)	0.01
Location of the entry tear			
Ascending aorta/Valsalva	21 (51.2%)	178 (64.2%)	0.1
Aortic arch	9 (22.0%)	47 (17.0%)	0.5
Descending aorta or unidentified	10 (24.4%)	52 (18.8%)	0.4
Cardiopulmonary bypass time > 4 h	14 (34.1)	45 (16.2%)	< 0.01

BMI body mass index, *COPD* chronic obstructive pulmonary disease, *SBP* systolic blood pressure, *ECMO* extra-corporeal membrane oxygenation, *AVR* aortic valve replacement, *CABG* coronary artery bypass grafting

insufficient to salvage these critically ill patients. Peripheral revascularization, including fenestration, extra-anatomical bypass, or active reperfusion, should be prioritized according to the severity and location of ischemia. Recent studies have reported the potential of percutaneous interventional endovascular treatment of the malperfusion followed by central aortic repair using hybrid operation rooms [12, 14, 15].

However, mesenteric ischemia often lacks clear preoperative symptoms and thus can be frequently overlooked, possibly exacerbating occultly during operation [16, 17]. Although severe acidosis and/or hyperlactatemia were

Table 5 Risk factors for hospital mortality of overall malperfusion ($N = 318$) (multivariate analysis)

	Odds ratio	95% confidence interval	p
Shock (SBp < 80 mmHg)	6.124	2.817–13.316	< 0.0001
Obesity (BMI > 30 kg/m^2)	5.746	1.746–18.909	0.0040
Mesenteric malperfusion	3.720	1.228–11.272	0.0202
CPB time > 4 h	2.229	0.985–5.045	0.0545
Concomitant CABG	2.043	0.874–4.776	0.0992
Number of organ system affected by malperfusion ≥ 2	1.147	0.488–2.696	0.7532

SBP systolic blood pressure, *BMI* body mass index, *CPB* cardiopulmonary bypass

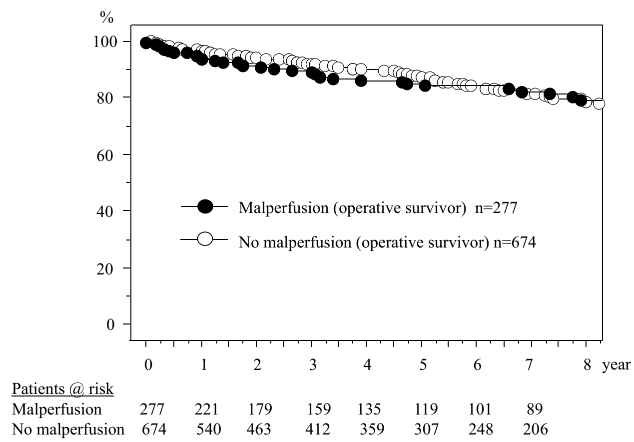

Fig. 2 Kaplan–Meier curves for long-term survival of patients undergoing surgery for acute type A dissection

important signs of mesenteric ischemia, prognosis of such progressed conditions was extremely poor [17, 18]. If the degree of mesenteric damage or complication of malperfusion is unclear, probe laparotomy should be performed first to determine the strategy. In the absence of ongoing or exacerbating severe mesenteric ischemia with bowel necrosis, central aortic repair should be prioritized. When mesenteric ischemia is observed, mesenteric or celiac bypass, temporary active perfusion, or bowel resection should be considered based on each patient's condition [15]. However, if extensive irreversible organ damage is observed, discontinuation of further aggressive treatment should be considered [8].

As with mesenteric malperfusion, coronary malperfusion was recognized as a strong predictor for hospital death [2–8]. In our univariate analysis, coronary malperfusion was close to statistical significance ($p = 0.053$). It occurs not only before surgery but also during operation, and requires flexible treatment in a case-by-case basis. Regarding coronary malperfusion in perioperative period, we assessed mechanism, physiology, and our surgical strategy in a previous report [19].

Pacini et al. [4] and Czerny et al. [5] reported that multiorgan malperfusion amplified the risk of hospital mortality. Similarly, results of our study indicated that the number of involved organs substantially affected outcomes. The univariate analysis also indicated that malperfusion in two or more organs was one of the predictors of hospital death. These patients frequently had serious conditions such as shock, coma, and acidosis, making it difficult to survive. As the mortality from end-organ damage seems to exceed the risk of rupture in ATAAD with complex malperfusion, immediate end-organ reperfusion before central repair should be considered in such cases. Uchida et al. reported the effectiveness of early reperfusion strategy comprising percutaneous coronary intervention for coronary malperfusion, direct surgical fenestration for carotid artery occlusion, active perfusion of the superior mesenteric artery for mesenteric malperfusion, and external shunting from brachial artery to the femoral artery for lower leg ischemia. They performed central repair immediately after reperfusion therapy and achieved outstanding improvement in surgical results of patients with malperfusion (3.6% of hospital mortality) [8]. Thus, immediate aggressive reperfusion for malperfused organs might improve surgical outcomes of ATAAD patients with serious malperfusion.

The long-term clinical course of ATAAD with malperfusion before surgery is unknown, and therefore, the effect of preoperative malperfusion on long-term outcomes remains unclear. Chu et al. reported that mid-term mortality after immediate open surgical repair in patients with ATAAD complicated by malperfusion was no different from that in patients without malperfusion [20]. The current study demonstrated that the long-term results of operative survivors were favorable even in the malperfusion group, suggesting that favorable long-term outcomes were expected for operative survivors, although malperfusion was associated before surgery.

This study had several limitations. A principal limitation was patient selection bias. Most ATAAD patients were referred from other hospitals for operative indications; this cohort may not have included some high-risk patients who were considered by other hospitals to be non-candidates for surgery or who were deemed to be unstable for transfer. This study was conducted as a retrospective analysis of clinical experience and encompassed data of 26 years. During this time, variability in factors, such as surgical technique and perioperative management, could have influenced outcomes. Although extent and status of false lumen impact long-term outcomes of operative survivors, we did not assess this issue in this report. As for fate of false lumen, surgical results, and downstream effect in remote period, we reported a patent false lumen decreased survival and increased distal aortic events after surgery in previous reports [21, 22].

Conclusions

Malperfusion in patients with ATAAD increased the risk of hospital mortality, especially malperfusion of more organ systems and/or mesenteric malperfusion, which made prognosis unfavorable. The effects of central aortic repair were limited in this patient cohort; thus, emergency reperfusion of the affected vascular bed followed by central repair might have to be considered for such severe cases.

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Compliance with ethical standards

Conflict of interest Koji Kawahito and the other authors have no conflict of interest.

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