Surgical Thoracoabdominal Aortic Aneurysm Repair in a Non-High-Volume Institution

Takashi Kunihara[1] Claudia Vukic[2] Fumihiro Sata[3] H. J. Schäfers[2]

[1] Department of Cardiac Surgery, The Jikei University School of Medicine, Minato-ku, Tokyo, Japan
[2] Department of Thoracic and Cardiovascular Surgery, Saarland University Medical Center, Homburg, Germany
[3] Health Center, Chuo University, Tokyo, Japan

Address for correspondence Takashi Kunihara, MD, PhD, Department of Cardiac Surgery, The Jikei University School of Medicine, 3-25-8 Nishishinbashi, Minato-ku, Tokyo 105-8461, Japan (e-mail: kuniharat@gmail.com).

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Abstract

Background Surgical thoracoabdominal aortic aneurysm (TAAA) repair remains challenging. Apart from mortality, spinal cord injury (SCI) is a dreaded complication. We analyzed our experience to identify predictors for SCI in a nonhigh-volume institution.

Patients and Methods All patients who underwent TAAA repair between February 1996 and November 2016 ($n = 182$) were enrolled. Most were male ($n = 121$; 66.4%), median age was 68 years (range: 21–84). Elective operations were performed in 153 instances (84.1%). Our approach to minimize SCI includes distal aortic perfusion, mild hypothermia, identification of the Adamkiewicz artery, and sequential aortic clamping. Cerebrospinal fluid drainage was introduced in 2001 and liberal use of selective visceral perfusion in 2006.

Results Early mortality was 12.1%; it was 8.5% after elective procedures. Reduced left ventricular function, nonelective setting, older age, and longer bypass time were identified as independent predictors for mortality in multivariable logistic regression model. Permanent SCI was observed in nine patients (4.9%), of whom seven (3.8%) developed paraplegia. In a multivariable logistic regression model for paraplegia, peripheral arterial disease (PAD), Crawford type II repair, smaller body surface area, and era before 2001 were identified as independent predictors, whereas only PAD was significant for SCI. The incidence of paraplegia was 13.8% in extensive repair out of the first 91 cases, whereas it was improved up to 2.7% thereafter.

Conclusion Using an integrated approach, acceptable outcome of TAAA repair can be achieved, even in a nonhigh-volume center. PAD and extensive involvement of the aorta are strong independent predictors for spinal cord deficit after TAAA repair.

Keywords
- aneurysm
- aorta/aortic
- neurological
- outcomes
- spinal cord
- surgery
- complications

Introduction

Thoracoabdominal aortic aneurysm (TAAA) repair still remains a challenging operation. It includes extensive exposure and reconstruction of the aorta involving multiple vital aortic branches. Subsequent ischemia and reperfusion injury of many organs may cause various postoperative complications. This is one of the reasons for a relatively high postoperative mortality compared with routine cardiac operation in the modern era. A recent meta-analysis showed that the pooled mortality rate in experienced centers is still 11.26% (including 5.5% rupture cases).[1]

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The most relevant morbidity after TAAA repair is spinal cord injury (SCI), which limits the quality of life of the patients and leads to increased late mortality. The incidence of SCI was more than 10 to 20% even in the experienced hands in early series.[2] With recent technical modifications based on better understanding of the pathophysiology and mechanisms of SCI, this incidence of SCI has improved up to 3 to 8% in experienced centers.[1] In the hands of experts in TAAA repair, permanent paraplegia and paraparesis have decreased up to 2.9 and 2.4%.[3] It is well known that the incidence of SCI depends on the extent of aortic involvement.[4] In the most extended type of TAAA (Crawford's classification type II), SCI still occurs in 8 to 10% in high-volume centers.[3] Thus, spinal cord protection during TAAA repair is still of central importance.

The relevant risk of morbidity and mortality has stimulated the increasing use of endovascular treatment of TAAA.[5] Consequently, the number of patients treated surgically has decreased over time.[3] Endovascular therapy with modifications has been shown to have a lower risk of morbidity and mortality.[5] On the contrary, there is the unpredictable probability of treatment failure through endoleaks, and chimney extensions into selected aortic branches may lead to separate complications.[6]

Thus, surgical repair of TAAA still seems to be a valid option if it can be performed with limited risk, and in particular, with low incidence of SCI. In the current study, we intended to analyze clinical results in a nonhigh-volume center. In addition, we investigated the role of technical modifications.

Patients and Methods

In this study, 182 patients who underwent replacement of the thoracoabdominal aorta in our institution between February 1996 and November 2016 were enrolled (►**Fig. 1**). The study was approved by the regional ethics committee for the analysis and publication in anonymized fashion.

There were 121 male (66.4%) individuals, and the age ranged from 21 to 84 years (median: 68). The etiology was dissection in 54 (29.7%, acute: $n=1$, subacute: $n=4$, chronic: $n=49$) and Marfan's syndrome in 15 patients (8.2%). One hundred and fifty-three patients (84.1%) underwent elective surgery. The reasons for nonelective surgery ($n=29$, 15.9%) were impending rupture ($n=15$), symptomatic aneurysm ($n=7$), mycotic aneurysm ($n=3$), symptomatic mycotic aneurysm ($n=2$), and rupture ($n=2$). Similar to previous reports,[3] more than half of the octogenarians were operated on nonelective basis (five out of nine, 55.6%). According to Crawford/Safi's classification, type I was present in 56 (30.8%), type II in 34 (18.7%), type III in 43 (23.6%), type IV in 17 (9.3%), and type V in 32 (17.6%) patients. One hundred and three patients (56.6%) had undergone previous aortic surgery; the ascending aorta in 59 (32.4%), the aortic arch in 47 (25.8%), the descending thoracic aorta in 32 (17.6%), and the abdominal aorta in 54 (29.7%) patients. Fifty-seven patients (31.3%) had undergone previous cardiac surgery (valvular surgery in 31 [17.0%] and coronary artery bypass grafting in 34 [18.7%]). The baseline characteristics are listed in ►**Table 1**.

Technique of Operation

The majority of cases were operated by a corresponding author (H.J.S.), except for 31 cases operated by the first author (T.K.). All patients received the same protocol of anesthesia with neuromuscular blockade and continuous intravenous injection of fentanyl citrate. Separate bronchial

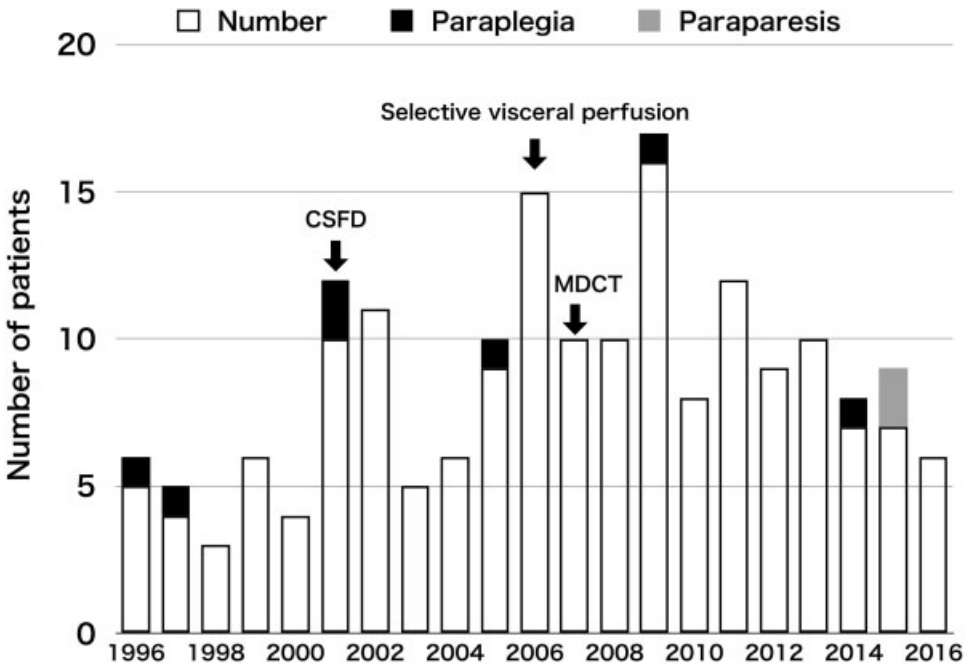

Fig. 1 Annual distribution of the number of cases undergoing thoracoabdominal aortic aneurysm repair. Black column means the case developing paraplegia and gray column means paraparesis. CSFD, cerebrospinal fluid drainage; MDCT, multidetector row computed tomography.

Table 1 Baseline characteristics according to Crawford/Safi's classification

	All ($n=182$)	Crawford I ($n=56$)	Crawford II ($n=34$)	Crawford III ($n=43$)	Crawford IV ($n=17$)	Safi V ($n=32$)	*p*-Value
Age (y)	68 (21–84)	69 (21–83)	64 (29–78)	63 (27–79)	73 (32–84)	71 (52–84)	<0.01
Male	121 (66.4%)	29 (51.8%)	25 (73.5%)	32 (74.4%)	11 (64.7%)	24 (75.0%)	0.08
Body height (m)	1.73 (1.49–2.00)	1.68 [1.55–1.92]	1.77 (1.56–1.97)	1.75 (1.49–2.00)	1.74 (1.63–1.99)	1.72 (1.54–1.96)	0.01
Body weight (kg)	77.5 (42.7–127)	73.0 (46.0–108.0)	80.0 (47.0–127.0)	79.0 (51.0–105.0)	82.0 (63.0–102.0)	79.5 (42.7–103.0)	0.05
Body surface area (m^2)	1.91 (1.36–2.56)	1.82 (1.45–2.30)	1.95 (1.48–2.56)	1.90 (1.55–2.29)	1.94 (1.68–2.34)	1.93 (1.36–2.24)	0.02
Marfan's syndrome	15 (8.2%)	1 (1.8%)	5 (14.7%)	7 (16.3%)	2 (11.8%)	0	0.02
Aortic dissection	54 (29.7%)	17 (30.3%)	18 (52.9%)	14 (32.6%)	1 (5.9%)	4 (12.5%)	<0.01
Maximum distal aortic diameter (cm)	6.5 (3.0–12.0)	6.5 (5.0–11.0)	7.0 (5.0–10.0)	6.6 (4.5–11.0)	5.5 (3.0–12.0)	5.8 (3.7–9.0)	0.02
Hypertension	161 (88.5%)	50 (89.3%)	30 (88.2%)	34 (79.1%)	16 (94.1%)	31 (96.9%)	0.16
Diabetes mellitus	19 (10.4%)	5 (8.9%)	2 (5.9%)	3 (7.0%)	3 (17.6%)	6 (18.8%)	0.31
Coronary artery disease	85 (46.7%)	25 (44.6%)	13 (38.2%)	21 (48.8%)	10 (58.8%)	16 (50.0%)	0.68
Left ventricular ejection fraction < 50%	12 (6.6%)	4 (7.1%)	2 (5.9%)	2 (4.7%)	0	4 (12.5%)	0.64
Peripheral arterial disease	48 (26.4%)	12 (21.4%)	1 (20.6%)	8 (18.6%)	8 (47.1%)	13 (40.6%)	0.05
Atrial fibrillation	16 (8.8%)	6 (10.8%)	1 (2.9%)	0	3 (17.6%)	6 (18.8%)	0.05
Prior stroke	20 (10.9%)	2 (3.6%)	2 (5.9%)	6 (14.0%)	3 (17.6%)	7 (21.9%)	0.06
Chronic kidney disease	50 (27.5%)	11 (19.6%)	8 (23.5%)	15 (34.9%)	5 (29.4%)	11 (34.4%)	0.41
Serum creatinine level (mg/dL)	1.0 (0.5–8.3)	1.0 (0.5–3.3)	1.1 (0.7–4.8)	1.0 (0.6–8.3)	1.0 (0.8–5.2)	1.1 (0.6–2.3)	0.54
Chronic obstructive pulmonary disease	60 (33.0%)	16 (28.6%)	8 (23.5%)	15 (34.9%)	6 (35.3%)	15 (46.9%)	0.31
Past or current tobacco use	94 (51.6%)	32 (57.1%)	9 (26.5%)	24 (55.8%)	11 (64.7%)	18 (56.3%)	0.03
Symptomatic	32 (17.6%)	7 (12.5%)	7 (20.6%)	4 (9.3%)	6 (35.3%)	8 (25.0%)	0.09
Impending rupture	24 (13.2%)	7 (12.5%)	7 (20.6%)	3 (7.0%)	4 (23.5%)	3 (9.4%)	0.28
Previous aortic surgery	103 (56.6%)	30 (53.6%)	25 (73.5%)	23 (53.5%)	7 (41.2%)	18 (56.3%)	0.2
Previous cardiac operation	57 (31.3%)	15 (26.8%)	11 (32.4%)	17 (39.5%)	6 (35.3%)	8 (25.0%)	0.62
Evaluation of the Adamkiewicz artery	129 (70.9%)	42 (75.0%)	25 (73.5%)	33 (76.7%)	6 (35.3%)	23 (71.9%)	0.02
Identification of the Adamkiewicz artery	51 (28.0%)	17 (30.4%)	8 (23.5%)	14 (32.6%)	2 (11.8%)	10 (31.3%)	0.51

ventilation was employed and the left lung was collapsed during the operative procedure. All operations were performed through a left thoracoabdominal incision, with the level of the thoracic incision depending on the proximal extent of surgery. All patients underwent prosthetic replacement of the diseased aorta with or without reimplantation of the intercostal/lumbar arteries (ICAs/LAs) using sequential clamping as far as possible. Left heart bypass was used in most instances. After 1 mg/kg of heparin was given, left heart bypass was initiated between the left lower pulmonary vein and the left femoral artery with mild hypothermia (the lowest nasopharyngeal temperature: 32 ± 3°C). The extracorporeal circuit was constructed as a closed system with a soft cardiotomy reservoir and active suction. Steroids were not added to the priming solution.

Cardiopulmonary bypass between the left femoral artery and vein (plus additional pulmonary artery if necessary) was used if deep hypothermic circulatory arrest was planned. This approach was employed if proximal control of the descending aorta or arch was difficult ($n = 24$, 13.0%). In these instances, the patient was cooled to a nasopharyngeal temperature of 23°C and 3 mg/kg of heparin was given.

Since August 2006, selective visceral perfusion was utilized whenever visceral arteries had to be reattached ($n = 63$, 34.6%). The main intention was preservation of the visceral organs; in addition, we hoped to improve collateral blood flow to the spinal cord. Each vessel including the renal arteries was cannulated with a balloon catheter and perfused from a side branch of the arterial return circuit. They were perfused at a rate of 500 to 800 mL/min until they were reattached to the vascular graft. A centrifugal cell-saving device was used in all patients during the operation. Postoperative shed mediastinal blood was discarded.

Adjuncts for Spinal Cord Protection

In elective cases, angiography was performed in most instances to identify the arteria radicularis magna (Adamkiewicz artery, AKA) preoperatively ($n = 129$, 72.1%). Since December 2007, multidetector row computed tomography (MD-CT) was also used for this purpose. When identified and involved in the diseased aortic segments, the corresponding ICA/LA was reattached to the vascular graft as the first step after creating the proximal aortic anastomosis. When the AKA could not be identified, ICAs with poor back bleeding, predominantly situated at left side between T8 and L1 were reattached.[7]

Since October 2001, cerebrospinal fluid drainage (CSFD) was routinely used in 113 patients (62.1%) irrespective of the use of hypothermic circulatory arrest. Lumbar CSFD was performed through a 16-gauge indwelling catheter, which was inserted into the L4/5 intervertebral space before induction of the anesthesia with careful monitoring of neurological disturbance. CSF was allowed to freely drain if CSF pressure exceeded 13 cmH_2O (or 10 mm Hg). Throughout its use, CSF pressure was monitored intermittently. CSFD was started just after the induction of anesthesia and was continued until intact spinal cord function was confirmed. The catheter was generally removed on the second or third postoperative day.

Postoperatively, mean systemic blood pressure was maintained above 80 mm Hg. Low-molecular-weight heparin was given for anticoagulation (1 mg/kg/d). All patients received aspirin 100 mg/d.

Statistical Analysis

The statistical analysis was performed by a consultant statistician (F.S.) using the "Statistical Program for Social Sciences" for windows version 23.0 (IBM Corp, Armonk, New York, United States). All continuous values are expressed as median (range) or mean $\pm$ standard deviation, as appropriate. Nonparametric test using either Mann–Whitney *U*-test or Kruskal–Wallis' one-way analysis of variance, as appropriate, was used for comparison of the continuous variables between the groups. Categorical variables are presented as number and percentage. Pearson chi-square test was used for comparison of frequencies between the groups. A *p*-value of less than 0.05 was considered statistically significant. For multivariable logistic regression model of mortality and spinal cord deficit, pre- and intraoperative variables showed in ►**Appendix 1** were used and analyzed separately. Clinically relevant factors associated with the outcomes univariately with a *p*-value of <0.02 were entered into multivariable logistic regression models using likelihood ratio forward selection.

Results

Intraoperative Outcomes

Median aortic cross-clamping, extracorporeal circulation, and operating times were 53 (15–165), 81 (29–234), and 230 (110–660) minutes, respectively. Seven patients were operated by simple clamping: one patient with a type I, another with a type III, and five patients with a type IV repair. In the 24 patients who underwent hypothermic circulatory arrest, median circulatory arrest time was 12 (5–36) minutes. Sixty-five patients (35.7%) required blood transfusion of packed red blood cells with a median of 2.4 (1–22) units, but only half of them ($n = 31$, 17.0%) needed more than 2 units. Packed red blood cells were significantly more given to patients who underwent deep hypothermic circulatory arrest (2.3 ± 2.8 units) than those did not (0.9 ± 2.2 units, $p < 0.01$). The detail of intraoperative parameters according to Crawford/Safi's classification is illustrated in ►**Table 2**.

Postoperative Mortality

Twenty-two patients died within 30 days after the surgery resulting in an early mortality of 12% (►**Table 3**). Early mortality was 8.5% after elective procedures, whereas it was 31.0% after nonelective setting ($p < 0.01$). The patients who underwent deep hypothermic circulatory arrest died more frequently (25.0%) than those did not (10.1%, $p = 0.05$). We could not find any learning curve in mortality; early mortality was 11.0% among the first 91 cases and it was 13.2% thereafter. The causes of early death were multiorgan

Table 2 Intraoperative parameters according to Crawford/Safi's classification

	All ($n=182$)	Crawford I ($n=56$)	Crawford II ($n=34$)	Crawford III ($n=43$)	Crawford IV ($n=17$)	Safi V ($n=32$)	*p*-Value
Elective setting	153 (84.1%)	50 (89.3%)	27 (79.4%)	39 (90.7%)	13 (76.5%)	24 (75.0%)	0.22
Era since October 2001 (CSFD)	149 (81.9%)	40 (71.4%)	30 (88.2%)	34 (79.1%)	14 (82.4%)	31 (96.9%)	0.04
Era since August 2006 (visceral perfusion)	105 (57.7%)	31 (55.4%)	18 (52.9%)	23 (53.5%)	10 (58.8%)	23 (71.9%)	0.49
Era since December 2007 (MDCT)	91 (50.0%)	27 (48.2%)	14 (41.2%)	18 (41.9%)	9 (52.9%)	23 (71.9%)	0.08
Bypass grafting to the mesenteric arteries	56 (30.8%)	8 (14.3%)	13 (38.2%)	14 (32.6%)	9 (52.9%)	12 (37.5%)	0.01
Revascularization of the intercostal arteries	118 (64.8%)	46 (82.1%)	31 (91.2%)	27 (62.8%)	1 (5.9%)	13 (40.6%)	<0.01
Cerebrospinal fluid drainage	113 (62.1%)	33 (58.9%)	24 (70.6%)	25 (58.1%)	6 (35.3%)	25 (78.1%)	0.04
Hypothermic circulatory arrest	24 (13.2%)	12 (21.4%)	7 (20.6%)	2 (4.7%)	0	3 (9.4%)	0.03
Selective visceral perfusion	63 (34.6%)	7 (12.5%)	15 (44.1%)	20 (46.5%)	5 (29.4%)	13 (40.6%)	<0.01
Aortic cross-clamping time (min)	53 (15–165)	47 (19–120)	73 (15–165)	61 (20–123)	42 (28–78)	38 (16–116)	<0.01
Extracorporeal circulation time (min)	81 (29–234)	66 (35–166)	121 (72–234)	89 (34–184)	65 (32–137)	76 (29–147)	<0.01
Circulatory arrest time (min)	12 (5–36)	13 (6–20)	14 (9–36)	12 (7–17)		10 (5–11)	0.37
Operation time (min)	230 (110–660)	225 (150–425)	300 (195–450)	230 (137–660)	205 (110–288)	205 (120–343)	<0.01
Transfusion of packed red blood cells	65 (35.3%)	26 (46.4%)	14 (41.2%)	13 (30.2%)	4 (23.5%)	8 (25.0%)	0.21
Number of packed red blood cells transfused	2.4 (1–22)	2.6 (1–6)	2.5 (1–12)	2.1 (1–22)	2.0 (2–2)	3.0 (1–6)	0.17
Transfusion of packed red blood cells >2 units	31 (17.0%)	13 (23.2%)	6 (17.6%)	7 (16.3%)	0	5 (15.6%)	0.28

Abbreviations: CSFD, cerebrospinal fluid drainage; MDCT, multidetector row computed tomography.

failure mainly caused by nonocclusive mesenteric ischemia in 17 patients and cardiac failure in 5 patients.

Fifteen baseline parameters were significant predictors for early mortality in univariate analysis, of which reduced left ventricular function and nonelective procedure were identified as independent predictors in multivariable logistic regression model (►**Table 4**). Mortality was 6.8% in patients with preserved left ventricular function (ejection fraction >50%), whereas it was 33.3% in those with reduced function. In intraoperative variables, age and extracorporeal circulation time were identified as independent predictors in multivariate logistic regression model. The mean age of operative survivors and nonsurvivors was 64 ± 12 and 70 ± 10 years, respectively. Mean extracorporeal circulation time of operative survivors and nonsurvivors was 81 ± 34 and 118 ± 47 minutes.

Spinal Cord Deficit

Permanent SCI was observed in nine patients (4.9%); seven patients (3.8%) developed permanent paraplegia and two patients (1.1%) paraparesis (►**Table 3**). The assumed cause of permanent SCI was failure to identify and reimplant AKA

Table 3 Early clinical outcomes according to Crawford/Safi's classification

	All ($n=182$)	Crawford I ($n=56$)	Crawford II ($n=34$)	Crawford III ($n=43$)	Crawford IV ($n=17$)	Safi V ($n=32$)	*p*-Value
Death within 30 d	20 (11.0%)	5 (8.9%)	5 (14.7%)	3 (7.0%)	2 (11.8%)	5 (15.6%)	0.63
Stroke	6 (3.3%)	3 (5.3%)	1 (2.9%)	2 (4.7%)	0	0	0.61
Permanent spinal cord injury	9 (4.9%)	2 (3.6%)	3 (8.8%)	1 (2.3%)	0	3 (9.4%)	0.39
Permanent paraplegia	7 (3.8%)	2 (3.6%)	3 (8.8%)	0	0	2 (6.3%)	0.27
New onset hemodialysis	22 (12.1%)	4 (7.1%)	7 (20.6%)	5 (11.6%)	2 (11.8%)	4 (12.5%)	0.46
New onset atrial fibrillation	38 (20.9%)	15 (26.8%)	6 (17.6%)	8 (18.6%)	3 (17.6%)	6 (18.8%)	0.78
Low cardiac output syndrome	11 (6.0%)	3 (5.3%)	3 (8.8%)	0	1 (5.9%)	4 (12.5%)	0.23
Respiratory failure	49 (27.0%)	13 (23.2%)	11 (32.3%)	10 (23.2%)	7 (41.2%)	8 (25.0%)	0.56
Reintubation	12 (6.6%)	2 (3.6%)	2 (5.9%)	3 (7.0%)	1 (5.9%)	4 (12.5%)	0.61
Necessitating tracheostomy	14 (7.7%)	5 (8.9%)	4 (11.8%)	1 (2.3%)	1 (5.9%)	3 (9.4%)	0.58
New-onset recurrent nerve palsy	3 (1.6%)	2 (3.6%)	1 (2.9%)	0	0	0	0.98
Re-exploration for bleeding	12 (6.6%)	4 (7.1%)	4 (11.8%)	2 (4.7%)	0	2 (6.3%)	0.65
Gastrointestinal complications	10 (5.5%)	3 (5.3%)	3 (8.8%)	1 (2.3%)	1 (5.9%)	2 (6.3%)	0.81
Nonocclusive mesenteric ischemia	19 (10.4%)	6 (10.7%)	4 (11.8%)	3 (7.0%)	3 (17.6%)	3 (9.4%)	0.81
Length of ventilation (d)	1 (0–33)	1 (1–33)	1 (0–22)	1 (0–21)	1 (0–17)	1 (0–24)	0.88
Length of intensive care unit stay (d)	2 (0–34)	2 (1–34)	2 (0–22)	2 (1–28)	2 (1–17)	2 (1–23)	0.98
Length of hospital stay (d)	16 (2–59)	16 (6–48)	17 (2–53)	16 (2–59)	15 (6–32)	15 (4–41)	0.98

in eight cases, lack of visceral organ perfusion in six, extended aortic aneurysm in six ($n=3$ each in types II and V), peripheral arterial disease (PAD) including carotid artery stenosis in five, no CSFD in four, and prolonged aortic cross-clamping time in three (165, 89, and 62 minutes). The incidence of SCI became slightly better from the first 91 cases (both SCI and paraplegia: 5.5%) to the recent 91 cases (SCI: 4.4%, paraplegia: 2.2%). This learning curve became more evident in the high-risk subgroup for SCI. The incidence of both SCI and paraplegia was 13.8% in 37 cases undergoing either Crawford type II or Safi type V repair out of the first 91 cases, whereas they were dramatically improved up to 5.4 and 2.7% in the recent 27 cases ($p=0.39$ and $p=0.16$).

Thirteen preoperative parameters were significant predictors for paraplegia in univariate analysis, of which PAD was identified as the strongest independent predictors, followed by Crawford type II repair and smaller body surface area in multivariable logistic regression model (►**Table 4**). The incidence of paraplegia was 10.4 or 1.5% in patients with or without PAD, respectively. Crawford type II repair resulted in 8.8% paraplegia risk, whereas it was 2.2% in others. Mean body surface area of patients with or without paraplegia was 1.76 ± 0.29 and 1.92 ± 0.19 m^2, respectively. In intraoperative variables, era of CSFD was identified as independent predictor in a multivariable logistic regression model. The incidence of paraplegia was 11.8% before October 2001 and it dramatically decreased up to 2.0% thereafter.

Thirteen preoperative parameters proved to be significant predictors for SCI in univariate analysis, of which only PAD was identified as an independent predictor in multivariable logistic regression model (►**Table 4**). Incidence of paraplegia was 12.5 or 2.2% in patients with or without PAD, respectively. No independent predictor for SCI was identified among intraoperative variables. The use of deep hypothermic circulatory arrest or not did not affect both the incidence of paraplegia (4.2 vs. 3.8%, respectively, $p=1.0$) and SCI (4.2 vs. 5.1%, respectively, $p=1.0$) at all.

Table 4 Multivariable logistic regression model of mortality and spinal cord deficit

	Mortality	*p*-Value	OR	95% CI	Paraplegia	*p*-Value	OR	95%CI	SCI	*p*-Value	OR	95% CI
Preoperative variables	LVEF < 50%	<0.01	7.59	1.785–32.273	PAD	0.02	9.679	1.566–59.829	PAD	0.01	6.19	1.483–25.840
	Nonelective setting	0.03	4.347	1.189–15.888	Crawford type II	0.04	6.584	1.115–38.883				
					Body surface area	0.04	0.015	0–0.773				
Intraoperative variables	Age	0.02	1.082	1.014–1.154	Era of CSFD	0.03	0.099	0.013–0.769				
	Bypass time	<0.01	1.028	1.012–1.044								

Abbreviations: CI, confidence interval; CSFD, cerebrospinal fluid drainage; LVEF, left ventricular ejection fraction; OR, odds ratio; PAD, peripheral arterial disease; SCI, spinal cord injury.

Postoperative Morbidities

Twelve patients (6.6%) underwent re-exploration for bleeding. One-hundred and twenty-five patients (68.7%) could be extubated within 24 hours postoperatively and 30 patients (16.7%) required mechanical ventilation for more than 3 days. Reintubation was necessary in 12 patients (6.6%) and tracheostomy was performed in 14 patients (7.7%). Postoperative new-onset hemodialysis was necessary in 22 patients (12.1%), of whom renal function recovered to normal level at discharge in 7 cases (3.8%).

Twelve out of 26 patients suffering from renal failure died within 30 days after aortic repair, so the early postoperative mortality rate in this subgroup was 46.2%. Postoperative new-onset atrial fibrillation occurred in 38 patients (20.9%). Recurrent nerve palsy was observed in two patients with Crawford type I and one with Crawford type II repairs. Six patients (3.3%) developed cerebral symptoms, of whom only two had a permanent stroke (1.1%). Nineteen patients (10.4%) developed nonocclusive mesenteric ischemia and underwent consecutive continuous infusion of papaverine into the superior mesenteric artery. Median length of intensive care unit stay was 2 (0–34) days and almost a quarter of patients after Crawford type II repair stayed in intensive care unit longer than 1 week ($n = 8$, 23.5%). Median length of hospital stay was 16 (2–59) days and a quarter of overall patients was discharged at later than 3 weeks after the operation ($n = 47$, 25.8%) (►**Table 3**).

Discussion

This is a single-center, retrospective cohort study to evaluate early outcomes after TAAA repair. With 182 cases over a 20-year period, our center is clearly a nonhigh-volume center. Despite the limited volume, our results, that is, 12.1% mortality and 4.9% permanent SCI, seem comparable with high-volume centers (7.5 and 5.4%, respectively).[3] This is even more relevant after elective procedures. Mortality was 8.5% and permanent SCI was 4.0% after elective operation in our cases, compared with 6.2 and 4.8% in a high-volume center, respectively.[3] Probably, our favorable results could be achieved through our integrated approach toward minimizing SCI. Namely, we have started TAAA repair with principal strategy using distal aortic perfusion, mild hypothermia, and sequential aortic clamping. Then, we have sequentially introduced additional maneuvers such as CSFD since October 2001, selective visceral perfusion since August 2006, and aggressive revascularization of the AKA by identification of AKA through MD-CT since December 2007 (►**Fig. 1**). The era since incorporation of CSFD was protective for paraplegia in the current study. In addition, our multidisciplinary aortic team with cardiologists, anesthesiologists, radiologists, intensivists, perfusionists, and nurses might play an important role to achieve our outcomes.

Multifactorial mechanisms of SCI after TAAA repair can be classified into the following three key processes: longer duration and higher degree of ischemia, failure in re-establishment of blood flow to the spinal cord, and a biochemically mediated ischemia and reperfusion injury.

To minimize the duration and degree of spinal cord ischemia, sequential aortic clamping with mild hypothermic distal aortic perfusion has been propagated.[8] It has been shown that distal perfusion can minimize SCI especially in extended type of TAAA (i.e., type II).[9,10] Recent researchers have found that blood flow of the anterior spinal artery is supplied by a rich collateral network.[11] In the current study, advanced atherosclerosis (PAD) was identified as predictor of both postoperative SCI and paraplegia, probably related to jeopardized collateral blood flow to the spinal cord.[3,12] Therefore, it is important to maintain this collateral blood flow during reattachment of the ICAs/LAs, especially in patients with degenerative aortic aneurysm with occlusion or stenosis of the part of ICAs/LAs.

Temperature plays an important role in attenuating the degree of spinal cord ischemia. Experimental studies suggest that cooling the spinal cord by 3°C or 5°C prolongs ischemic tolerance 2-fold or 2.5-fold, respectively.[13,14] Deep hypothermia can thus reduce the incidence of SCI; on the contrary, increasing morbidity and mortality rates have emerged as its drawback.[15] Therefore, this adjunct should be considered in balance with advantages (e.g., huge or extensive aneurysm, friable aortic tissue, high risk of embolization, complex repair, and redo cases) and disadvantages (e.g., coagulopathy, pulmonary dysfunction, and massive fluid shift).

To secure spinal cord protection, we have tried to identify AKA preoperatively as far as possible. This is a technically demanding by angiography, so we also used MD-CT since 2007. Of the nine patients with permanent SCI, the AKA could be identified preoperatively in only one at the height of TH11, out of replaced aortic segment. In the remaining eight patients, the AKA could not radiologically be identified; this was due to nonelective operation without preoperative angiography in four.

CSFD had already been advocated since more than a half century ago.[16] Its usefulness has recently been documented through a prospective randomized clinical trial[17] and a meta-analysis.[18] Its clinical role has been believed to enhance spinal cord perfusion pressure by decreasing CSF pressure. Removal of some negative neurotrophic substances mediated through ischemia-reperfusion injury of the spinal cord has been proposed as another role of CSFD.[19] Currently, CSFD has been reserved for extensive disease or high-risk cases for SCI alone because CSFD-associated complications are rare (0.2%) but miserable.[18,20] In the current study, extensive aneurysm (Crawford type II and Safi type V) had a higher incidence of SCI (9.1%) than others (2.6%) ($p = 0.07$), which was attenuated by CSFD. In this subgroup (types II and V), SCI was observed in 6.1% (3/49) or 17.6% (3/17) of cases with or without CSFD, respectively ($p = 0.08$), whereas they were 3.1 and 2.0% in the other subgroup (Crawford type I, III, and IV), respectively.

Other preoperative independent predictors for postoperative SCI have previously been reported by others, such as an emergent setting,[21,22] aortic dissection,[22,23] age >75 years old,[23] and renal dysfunction.[24] Lack of preoperative detailed examination, unstable hemodynamics, or injury of critical ICAs/LAs may contribute to the negative impact of urgent/emergent setting on postoperative SCI. In our cohort, aortic dissection was not associated with increased risk of SCI (SCI was 3.7% with dissection vs. 5.5% without dissection). A difference became obvious in type II extent aneurysm (SCI was 33.3% with dissection vs. 12.5% without dissection, $p = 0.59$), which is generally regarded as high-risk subset for SCI. Patients with SCI were older than those without (71 ± 4 vs. 65 ± 12 years old) without statistical power ($p = 0.13$). Older patients have generally more atherosclerotic disease than younger. Indeed, subgroup with PAD was significantly older than others (71 ± 7 vs. 63 ± 13 years old, $p < 0.01$) and identified as an independent predictor for paraplegia and SCI. In our study, preoperative serum creatinine level was quite similar between patients with or without SCI (1.2 ± 0.3 and 1.3 ± 0.8 mg/dL, respectively, $p = 0.16$) and five patients with preoperative chronic hemodialysis did not develop postoperative SCI.

Study Limitations

Relevant limitations are present in this study. First, this is not a randomized study but a retrospective study with consecutive patients without a control group. The use of operative adjuncts has evolved in detail over time. However, the consistent participation of a single surgeon (H.J.S.) may minimize the drawback of a retrospective nature of this study. Second, the number of patients was still small and SCI occurred very infrequently to assess real effect of patients' background and operative maneuvers. Nevertheless, we believe we could improve the operative outcomes of TAAA repair by integrated approach, although our institution is a nonhigh-volume center. We also believe independent predictors for SCI could be identified using a sophisticated statistical method.

Conclusion

In conclusion, our integrated approach could improve the operative outcomes of TAAA repair even in a nonhigh-volume center. PAD and extensive involvement of the aorta were independent predictors for spinal cord deficit after TAAA repair.

Disclosure

All the authors have nothing to disclose with regard to commercial support.

Conflict of Interest

All the authors have declared no competing interest.

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Appendix 1 Pre- and intraoperative variables used for multivariable logistic regression model of mortality and spinal cord deficit

Preoperative variables
Age
Gender
Body height
Body weight
Body surface area
Aortic dissection
Crawford types I–IV
Safi type V
Crawford type II or Safi type V
Impending rupture
Mycotic aneurysm
Rupture or mycotic aneurysm
Previous aortic surgery
Previous cardiac surgery
Previous valve surgery
Previous coronary artery bypass grafting
Previous surgery for the ascending aorta
Previous surgery for the aortic arch
Previous surgery for the descending aorta
Previous surgery for the abdominal aorta
Ischemic heart disease
Sinus rhythm
Peripheral arterial disease including carotid artery stenosis
Hypertension
Chronic obstructive pulmonary disease
Diabetes mellitus
Current smoker
Previous smoker
Left ventricular ejection fraction $\leq$ 50%
Stroke or transient ischemic attack
Chronic kidney disease
Serum creatinine level
Hemodialysis
Liver disease
Adamkiewicz artery identified
Maximum aorta diameter
Emergent setting
Intraoperative variables
Number of the intercostal/lumbar arteries reimplanted
Revascularization of the intercostal/lumbar arteries
Selective visceral perfusion
Cerebrospinal fluid drainage
Era since introduction of cerebrospinal fluid drainage (October 2001)
Era since aggressive use of selective visceral perfusion (August 2006)
Minimum temperature
Extracorporeal circulation time
Aortic cross-clamping time
Circulatory arrest time
Use of circulatory arrest
Operation time
Number of packed red blood cell transfused
Transfusion of packed red blood cell

