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Original Article

Spinal Cord Injury Risk in Open Repair for Descending Thoracic and Thoracoabdominal Aneurysm

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Objective: In open repair for descending thoracic aortic aneurysm (DTAA) or thoracoabdominal aortic aneurysm (TAAA), the influence of re-interventions on spinal cord injury (SCI) remains unclear. This study evaluated the relationships between re-interventions, atherosclerosis, and SCI.
Methods: We retrospectively reviewed 78 patients who underwent open surgical repair for DTAA or TAAA between April 2011 and May 2023. The associations of SCI with (i) re-interventions with a history of endovascular therapy and graft replacement and (ii) atherosclerotic factors, including monocyte count, triglyceride levels (TG), and intra-aortic plaques, were examined.
Results: The rates of SCI complications and 30-day mortality were both 3.8% (3/78). There was no significant difference between the incidence of SCI in the re-intervention and first-time intervention groups (p >0.90). However, patients with protruding plaque on computed tomography (CT) were more affected by SCI than those without (13.3% vs. 1.6%, p = 0.034). Univariate analysis revealed that SCI was associated with increased monocyte count, TG, protruding plaques on CT, and intraoperative blood loss.
Conclusion: Re-interventions for DTAA and TAAA showed no association with the development of SCI under appropriate protective measures. The implicated risk factors may be atherosclerosis factors such as elevated monocyte count, TG, and protruding plaques on CT.

Keywords: atherosclerosis, monocytes, plaques, spinal cord injury, triglycerides

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Introduction

Open surgical repair for descending thoracic aortic aneurysm (DTAA) or thoracoabdominal aortic aneurysm (TAAA) is necessary for some patients, even in the era when thoracic endovascular aortic repair (TEVAR) is actively performed.[1,2] Although spinal cord injury (SCI) is a serious and devastating complication of these surgical interventions, strategies to prevent SCI after open surgical repair are not completely established.[3,4] Awad et al. categorize post-TEVAR risk factors into "surgical" and "patient."[1] The surgical risk factors include extensive aortic aneurysm and extensive aortic coverage, prior abdominal aortic aneurysm repair, urgent procedures, and excessive blood loss. Since these factors are similar to those seen in open surgical repair, various preventive measures such as hypothermia, segmental aortic clamping, distal aortic perfusion, reconstruction of intercostal arteries (ICAs), cerebrospinal fluid drainage (CSFD), and neuromonitoring have been employed.[2,5,6] In cases involving previous aortic interventions such as TEVAR, the frozen elephant trunk (FET) technique, endovascular aortic repair (EVAR), and graft replacement for abdominal aortic aneurysms (AAAs), the blood supply to the spinal cord is often decreased. However, recent studies have shown that previous interventions were not related to an increased probability of developing SCI.[7,8] Therefore, the relationship between re-interventions and the risk of developing SCI is controversial in an era when stent grafts are widely used as a minimally invasive treatment.[1,2,9]

On the other hand, patient risk factors for SCI include advanced age, renal insufficiency, and degenerative aneurysms. In particular, atherosclerosis is a potential risk factor for SCI, as low-density plaques have been potentially correlated with a shaggy aorta.[10–12]

Therefore, this study aimed to investigate the influence of re-interventions and atherosclerotic factors on SCI after open surgical repair for DTAA or TAAA.

Materials and Methods

Study population

This study protocol conformed to the ethical guidelines of the Declaration of Helsinki and was approved by the appropriate ethics committee (approval number: U22-07-006). The need for informed consent was waived because of the retrospective nature of the study. This observational study retrospectively analyzed patients who underwent open surgery for DTAA or TAAA at our university hospital between April 2011 and May 2023. Preoperative information, on-admission blood test results, surgical records, and findings of preoperative imaging, including enhanced computed tomography (CT), were reviewed, and their relationship with SCI was examined. SCI was defined as permanent paraparesis or paraplegia after surgery.[3] We divided the included patients into two groups: with or without previous aortic intervention. The participants in the previous aortic intervention group were defined as having undergone surgical intervention involving the distal arch aneurysm to the terminal aorta. Accordingly, the re-intervention group had a history of FET accompanied by total arch replacement (TAR), TEVAR, EVAR, or graft replacement in this range. As for FET, cases were included in which the distal end of the FET was inserted below Th6. The re-intervention group was compared with the first-time intervention group in terms of sacrificed ranges and SCI.

Atherosclerotic parameters

We investigated the following atherosclerosis-related factors: serum triglyceride levels and monocyte count on admission.[13,14] A protruding plaque on enhanced CT (**Fig. 1**) was defined as reflecting severe atheroma when a plaque near the replacement area had a thickness of >5 mm.[10,15] The relationship between protruding plaque on CT and SCI was examined.

Operative strategy

Prophylactic CSFD was attempted for extensive aneurysms (Crawford I, II, and III) or high-risk cases with a history of TEVAR, EVAR, or abdominal aortic graft replacement. ICA reconstruction was performed for high-risk cases, especially when motor-evoked potential (MEP) changes were observed during surgery.

The target aortic aneurysm was exposed through a fifth, sixth, or seventh intercostal thoracotomy, with or without laparotomy. After heparinization, left heart bypass was established through common femoral or central artery (i.e., descending aorta) cannulation and left atrial drainage through the left lower pulmonary vein.

Under moderate hypothermia (32–33°C), graft replacement was performed at the proximal aorta by segmental aortic clamping. If the distal aorta was unsuitable for clamping because of the risk of plaque scattering, distal anastomosis was performed under circulatory arrest in the lower limbs. Once the graft was clamped, distal perfusion was restarted. As a rule, in all patients with Crawford type II, ICA reconstruction was attempted. However, in some patients with other Crawford types, reconstruction was not performed if MEPs were satisfactory. After the opening of the aneurysm, prompt ligation of ICAs and temporary occlusion using a catheter was performed to reduce blood loss. For visceral perfusion, the celiac and superior mesenteric arteries were perfused through additional catheters. Each kidney was intermittently perfused with cooled lactated Ringer's solution.[16]

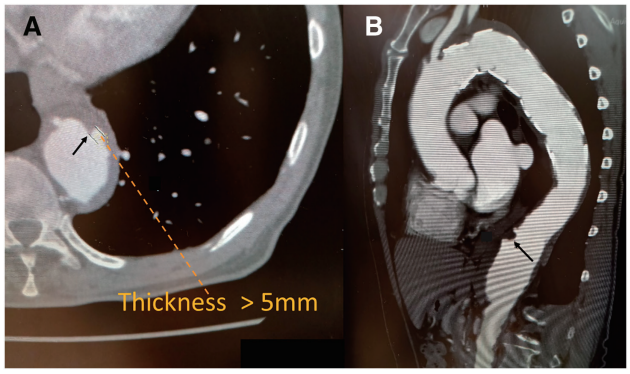

Fig. 1 Preoperative enhanced computed tomography. (**A**) Axial view of a protruding plaque larger than 5 mm (black arrow) in the descending aorta. (**B**) Sagittal view of the same plaque (black arrow).

Statistical analysis

Categorical variables are expressed as numbers and proportions, whereas continuous variables are presented as median and interquartile range or mean ± standard deviation. Statistical analysis of basic characteristics was performed using Student's *t*-test for continuous variables with normal distribution, the Mann–Whitney *U* test for all other continuous variables, and the chi-squared test for categorical variables. In analyses including the monocyte count, patients with infected aneurysms were excluded because inflammatory parameters were usually increased; therefore, the white blood cell count became elevated, leading to elevated monocyte counts. Risk factors associated with SCI, including intraoperative blood loss and atherosclerosis, were identified by univariate analysis. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated. Owing to the small sample size, multivariate analysis was not performed. The Kaplan–Meier method was used to calculate the long-term survival rate. The survival rates of the re-intervention and first-time intervention groups were compared using the log-rank method. All statistical analyses were performed using JMP version 14.0 (SAS Institute Inc., Cary, NC, USA). A p-value <0.05 was considered statistically significant.

Table 1 Basic characteristics of patients who underwent surgery for descending or thoracoabdominal aneurysms, with or without previous aortic intervention

Characteristics	All (n = 78)	Re-intervention (n = 34)	First-time intervention (n = 44)	p-value
Age (years), median (IQR)	68.0 (60.0, 72.0)	69.0 (66.0, 73.0)	66.5 (58.8, 70.5)	0.090
Male sex, n (%)	58 (74.4)	30 (88.2)	28 (63.6)	0.014*
History of smoking, n (%)	44 (56.4)	23 (67.6)	21 (47.7)	0.079
Current smoker, n (%)	8 (10.3)	6 (17.6)	2 (4.5)	0.073
Hypertension	75 (96.2)	31 (91.2)	44 (100)	0.079
Diabetes	17 (21.8)	8 (23.5)	9 (20.5)	0.700
Dyslipidemia	44 (56.4)	21 (61.7)	23 (52.3)	0.400
CKD (creatinine >1.5 mg/dL)	22 (28.2)	12 (35.3)	10 (22.7)	0.200
Hemodialysis	3 (3.8)	1 (2.9)	2 (4.5)	>0.90
Cerebral infarction	11 (14.1)	5 (14.7)	6 (13.6)	>0.90
COPD	7 (9.0)	3 (8.8)	4 (19.1)	>0.90
Marfan syndrome	2 (2.6)	1 (2.9)	1 (4.5)	>0.90
Protruding plaque on enhanced CT	15 (19.2)	8 (23.5)	7 (15.9)	0.397
Previous intervention, n (%)				
TAR + FET	3 (3.8)	3[a] (8.8)	–	–
TEVAR	15 (19.2)	15[a] (42.4)	–	–
EVAR or abdominal graft replacement	19 (24.4)	19[a] (55.9)	–	–
DAA or TAAA replacement	4 (5.1)	4[a] (11.8)	–	–
Aortic aneurysm, n (%)				
Emergency	9 (11.5)	6 (17.7)	3 (6.8)	0.138
Type of etiology				
Degenerative	33 (42.3)	16 (47.1)	17 (38.6)	0.022*
Dissecting[b]	34 (43.6)	11 (35.3)	23 (52.3)	
Infected[b]	13 (16.7)	8 (23.5)	5 (11.4)	
Classification				
Descending	20 (25.6)	7 (20.6)	13 (29.5)	0.430
Crawford I	4 (5.1)	1 (3.2)	3 (7.0)	
Crawford II	19 (24.4)	7 (20.6)	12 (27.3)	
Crawford III	10 (12.8)	4 (11.8)	6 (13.6)	
Crawford IV	23 (29.5)	11 (38.2)	10 (22.7)	
Crawford V/Safi	2 (2.6)	2 (5.9)	0 (0)	
Prophylactic CSFD	39 (50.0)	15 (44.1)	24 (54.5)	0.361
Intra- and postoperative data				
ICA reconstruction, n (%)	15 (19.2)	5 (14.7)	10 (22.7)	0.373
Bleeding (mL), median (IQR)	915.0 (632.8, 1,550.0)	996.5 (575.0, 1,633.5)	887.0 (638.8, 1,489.8)	0.800
SCI, n (%)	3 (3.8)	1 (2.9)	2 (4.5)	>0.90
30-day mortality, n (%)	3 (3.8)	2 (5.9)	1 (2.3)	0.600
In-hospital mortality, n (%)	7 (9.0)	4 (11.8)	3 (6.8)	0.700

[a]The previous aortic intervention group included seven overlapping cases in each procedure combination.

[b]Two overlapping cases were included.

*A p-value <0.05 was considered to be statistically significant. Data were compared using the Student's *t*-test for age, the Mann–Whitney *U* test for all other continuous variables, and the chi-squared test for categorical variables.

CKD: chronic kidney disease; COPD: chronic obstructive pulmonary disease; CT: computed tomography; CSFD: cerebrospinal fluid drainage; DAA: descending aortic aneurysm; EVAR: endovascular aortic repair; FET: frozen elephant trunk; ICA: intercostal artery; IQR: interquartile range; SCI: spinal cord injury; TAAA: thoracoabdominal aortic aneurysm; TAR: total arch replacement; TEVAR: thoracic endovascular aortic repair

Results

Basic characteristics of the study population

The characteristics of the study population are shown in **Table 1**. The re-intervention group included FET accompanied by TAR (8.8%, 3/34), TEVAR (42.4%, 15/34), EVAR/abdominal aortic replacement (55.9%, 19/34), and DTAA or TAAA replacement (11.8%, 4/34). We classified the cases according to their etiology as follows: 33 degenerative aneurysms (including atherosclerotic

changes), 34 dissecting aneurysms, and 13 infected aneurysms with 2 overlapping cases. Compared with the first-time intervention group, there was no significant difference in the basic characteristics of the re-intervention group, except for male sex, hypertension, and type of etiology. Prophylactic CSFD was performed preoperatively in 50% (39/78) of patients and reconstruction of ICAs in 19.2% (15/78). The rates of SCI complications and 30-day mortality were both 3.8% (3/78). The three patients with SCI included two with paraplegia and one with paraparesis. The etiology in all patients with SCI was degenerative aneurysm. No delayed SCI was observed during the follow-up period. There was no significant difference in SCI between the re-intervention and first-time intervention groups (p = 0.715). The causes of in-hospital mortality included (i) intestinal necrosis, rupture at the anastomosis site due to infection, and ventricular fibrillation within 30 days (one case each); (ii) sepsis/disseminated intravascular coagulation (two cases); (iii) sepsis due to intestinal perforation (one case); and (iv) cerebral hemorrhage in long-term hospitalization (one case).

Covered or sacrificed segmental arteries

In the re-intervention group, the distributions of the covered segmental arterial range in the previous intervention are shown in **Fig. 2A**, and their mean range was 6.7 ± 3.3 (range, 2–13) segmental arteries. The interval until the second aortic intervention was 61 ± 57 (range, 0.25–185) months. **Figure 2B** presents the sacrificed segmental arteries in the current open surgical repair for DTAA or TAAA in each group. The re-intervention group had a significantly shorter range than the first-time intervention group (3.8 ± 2.7 vs. 6.3 ± 2.2, p = 0.0003) and included five cases with a range of zero, that is, without additional sacrificed segmental arteries, because the replacement range was similar to the covered range in the former TEVAR. Notably, their causes were one pseudoaneurysm and four aneurysmal enlargements by endoleak.

Relationships of SCI with previous intervention and protruding plaque

Regarding SCI occurrence, 4.5% (2/44) of those in the first-time intervention group developed SCI, whereas 2.9% (1/34) in the re-intervention group did. There was no significant difference in the incidence of SCI in the re- or first-time intervention groups (p = 0.715, **Fig. 3A**). However, patients with protruding plaque on CT had a significantly higher SCI occurrence than those without it (13.3%, 2/15 vs. 1.6%, 1/63, p = 0.034; **Fig. 3B**).

Risk factors for postoperative SCI

In the univariate analysis, the presence of protruding plaques (OR: 12.889, 95% CI: 1.057–157.184, p = 0.045), triglyceride levels (OR: 1.020, 95% CI: 1.005–1.036, p = 0.006), monocyte counts (OR: 1.011, 95% CI: 1.004–1.025, p = 0.001), and intraoperative blood loss (OR: 1.002, 95% CI: 1.000–1.004, p = 0.018) were risk factors for postoperative SCI (**Table 2**).

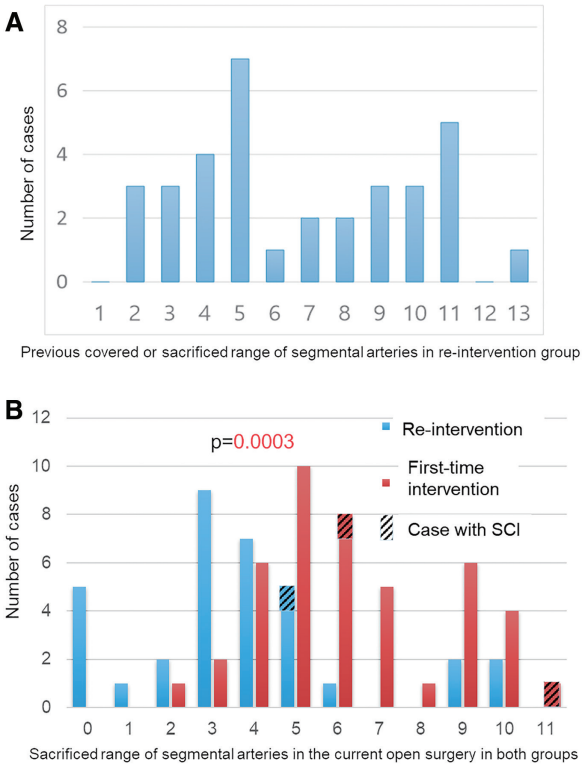

Fig. 2 Number of cases in each covered or sacrificed range of vertebrae. (**A**) Number of previously covered or sacrificed segmental arteries in the re-intervention group. (**B**) Number of sacrificed segmental arteries in the current open surgical repair for DTAA or TAAA in both re-intervention and first-time intervention groups. Cases complicated by SCI are shaded. DTAA: descending thoracic aortic aneurysm; SCI: spinal cord injury; TAAA: thoracoabdominal aortic aneurysm

Discussion

Stent grafts have become widely used, and more patients who previously underwent aortic interventions will account for candidates of open DTAA and TAAA repairs. Therefore, we aimed to evaluate the relationships between re-interventions, atherosclerotic factors, and SCI. Our results suggest that re-intervention and a more extensive range of sacrificed segmental arteries do not increase the risk of SCI complications. However, atherosclerotic factors such as protruding plaque, monocyte counts, and triglyceride levels may affect the incidence of SCI (**Table 2**, **Figs. 2B** and **3A**).

Research has shown that the incidence of atherosclerosis, characterized by intra-aortic plaques on CT, is

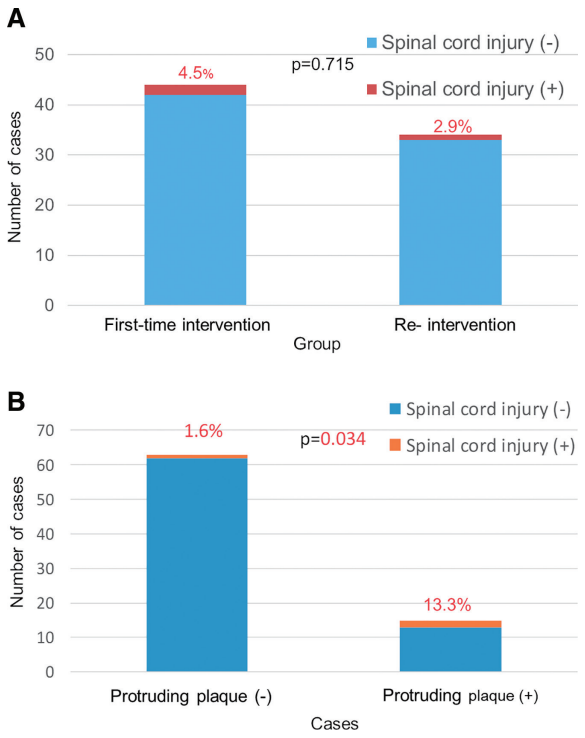

Fig. 3 Association of postoperative spinal cord injury with different factors. (**A**) Relationship with re-intervention or first-time intervention. (**B**) Relationship with the presence or absence of a protruding plaque on CT. CT: computed tomography

increasing because of population aging and Westernized dietary habits. In particular, some previous studies have pointed out the involvement of atherosclerosis in postoperative SCI.[10–12] However, the relationship between monocytes, which differentiate into macrophages in plaque formation, and SCI after the open aortic repair has rarely been discussed. Therefore, our findings would help provide valuable insights into the prevention of SCI.

In this study, the re-intervention group had a bimodal distribution of range covered by a stent graft or sacrificed by graft replacement (**Fig. 2A**). It was considered that the peak at shorter values reflects the intervention for short DTAA, Crawford type IV or V, or AAA, whereas the peak at longer values reflects the intervention for extensive DTAA or Crawford I, II, or III.

Generally, collateral flow to the spinal cord through the vertebral, internal iliac, residual intercostal, lumbar, or iliolumbar arteries develops after spinal cord ischemia or if compensatory changes occur to maintain spinal blood flow over time.[17–19] Therefore, re-interventions might not lead to a higher SCI risk due to long intervals and collateral flow. Besides, the recommendation of staged surgical intervention is based on the expectation of collateral development.[19,20] Although previous aortic interventions can cause a decrease in blood supply to the spinal cord, our finding that SCI was not associated may be a positive effect of staged surgical intervention. In our population, the re-intervention group had a remarkably shorter range of sacrificed segmental arteries. As such, these treatments were not always planned as secondary interventions (**Fig. 2B**). Among 34 patients in the group with previous aortic surgery, only three (8.8%) dared to undergo segmental surgery to avoid extensive replacement. Of these three patients, one was affected by SCI even after 2 months of follow-up.

In terms of the sacrificed range in the open repair and sacrificed total range in re-interventions with open repairs, extensive coverage can be managed with adequate protective measures, as mentioned in the Methods section. Even if the abovementioned factors increased the risk of SCI, effective measures such as CSFD, MEP monitoring, ICA reconstruction, segmental aortic clamping, and avoiding hypotension might have offset this risk.

On the other hand, atheromatous plaque sometimes scatters and can cause embolization of segmental arteries, including non-sacrificed arteries in the preserved range, during open surgery for shaggy aortas, no matter how carefully the surgery was performed. **Figure 3B** suggests that the presence of protruding plaque, which leads to a shaggy aorta, is related to SCI risk. Moreover, atherosclerosis factors, including monocyte counts and triglyceride levels, were also identified as SCI risk factors. Generally, macrophages are induced in patients with plaques, which can explain the increase in monocyte count.[13,14] In addition, macrophage deposition of low-density lipoprotein cholesterol and triglyceride deposition in the aortic wall via remnant lipoproteins were possibly observed as protruding plaques. Triglycerides are well-known risk factors for atherosclerosis. Protruding plaque on CT is closely related to monocyte counts and triglyceride levels, even though plaque formation depends on various factors, including lipid and blood sugar levels, blood pressure, stress, and smoking. Monocytes are susceptible to inflammatory, immune, and other factors; thus, their proliferation is nonspecific, and we should pay attention to monocyte counts as one of the risk factors for atherosclerosis to prevent SCI.[21]

We assume that the ease of creating and dislodging plaques leading to SCI is inconsistent as they may have a wide range of hardness, and soft and hard plaques differ in ease of scattering. Therefore, these plaques need to be qualitatively assessed.[22,23] Although the mechanisms underlying SCI were not specified, we hypothesize that microemboli from plaque scattering are involved in SCI after open surgery. Thus, there may be little benefit in reconstructing ICAs once microemboli in ICAs occur peripherally. Moreover, aortic procedures such as clamping might not be recommended for patients with rich atherosclerotic plaques, given the risk of multiple microemboli.[10,11]

Table 2 Univariate analysis of risk factors for postoperative spinal cord injury

Risk factors	Odds ratio	95% CI	p-value
Re-intervention	0.636	0.029–6.919	0.712
Prophylactic CSFD	0.412	0.035–4.781	0.464
ICA reconstruction	1.692	0.142–20.186	0.686
Sacrificed segmental arteries in open surgical repair in the current operation	1.352	0.862–2.121	0.189
Total sacrificed segmental arteries	1.205	0.894–1.626	0.221
Presence of protruding plaque on enhanced CT	12.889	1.057–157.184	0.045*
Triglyceride (per 1 mg/dL)	1.020	1.005–1.036	0.006*
Monocyte count/L (per 1 count)	1.011	1.004–1.025	0.001*
Intraoperative blood loss (per mL)	1.002	1.000–1.004	0.018*

*A p-value <0.05 was considered to be statistically significant.
CI: confidence interval; CSFD: cerebrospinal fluid drainage; CT: computed tomography; ICA: intercostal artery

As seen in previous studies, intraoperative blood loss is also a risk factor for SCI (**Table 2**).[1,2] SCI can occur because of the arterial steal phenomenon, wherein massive blood loss causes ischemia, even if efforts are made to maintain the blood pressure. Therefore, prompt ligation of ICAs and temporary occlusion with a catheter is important to reduce blood loss due to backflow after an aortotomy. Furthermore, meticulous hemostasis before reaching the aorta and administering heparin is an even more important step because bleeding increases after heparin administration.

Regarding outcome, in-hospital mortality (9.0%), SCI complication rate (3.8%), and 30-day mortality (3.8%) were comparable to those of previous studies.[3,4,10] The rate of mortality in infected aneurysms was 33.3% (4/12), whereas mortality in other combined degenerative and dissecting aneurysms was 4.8% (3/62). Therefore, infected aneurysms contributed to an increase in in-hospital mortality.

Finally, as the clinical impacts, increasing protective factors and decreasing risk factors, including those we pose, are necessary to avoid SCI because multiple factors affect SCI. Preoperative preparation for elective cases, medication, diet, and exercise therapy are as important as smoking cessation to reduce and stabilize aortic plaques.[2,4,5] Moreover, as an operative procedure, the no-clamp technique under circulatory arrest and hypothermic protection can help avoid plaque dislodgement in cases of severe atherosclerosis.

Limitations

This study has several limitations. First, this was a single-center, retrospective observational study with a limited sample size. Although the aortic interventional groups we analyzed appear heterogeneous, they all have a reduced inflow as a common feature due to segmental arteries being covered by stent grafts or sacrificed by graft replacement. Larger cohorts are desired which would also enable multivariate analysis. Second, the presence of protruding plaques does not accurately reflect the severity of atherosclerosis because a qualitative assessment of plaques on enhanced CT was not performed. Third, we analyzed each item regardless of whether the patient had received preoperative medical therapy. Thus, further stratified analysis under standardized conditions for drug therapy, meals, and preoperative examinations is necessary to better assess the risk factors for SCI.

Conclusion

Re-interventions were not related to developing SCI after open surgical repair for DTAA and TAAA. Instead, atherosclerotic factors such as protruding plaque on CT, monocyte counts, and triglyceride levels may potentially influence SCI more than re-interventions or extensive coverage. To the best of our knowledge, this study is the first to demonstrate that monocytes may be involved in SCI development after DTAA and TAAA repair. Based on our findings, a more cautious and prophylactic strategy may be necessary for patients with atherosclerotic factors to decrease the risk of SCI in an era of changing patient backgrounds.

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Data Availability Statement

The datasets analyzed during the current study are available from the corresponding author upon reasonable request.

Disclosure Statement

None.

Author Contributions

Study conception: MaF, HW
Data collection: GK, MiF, MS, YM, CI, MH, KW
Analysis: MaF, HM, YH
Manuscript preparation: MaF
Supervision: HW
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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