Title: Association between false lumen segmental arteries and spinal cord ischemia in type A acute aortic dissection

Hiroshi Kodama, *Shinya Takahashi, Takanobu Okazaki, Shohei Morita, Seimei Go, Masazumi Watanabe, Yoshitaka Yamane, Keijiro Katayama, Tatsuya Kurosaki, Taijiro Sueda

Department of Cardiovascular Surgery, Hiroshima University School of Medicine, Hiroshima, Japan

Corresponding author:

Shinya Takahashi

Department of Cardiovascular Surgery, Hiroshima University School of Medicine

1-2-3 Kasumi, Minami-ku, Hiroshima, 734-8551, Japan

E-mail: shiny@mte.biglobe.ne.jp

Phone: 082-257-5216

Fax: 082-257-5219

Abstract

Background

Spinal cord ischemia (SCI) and paraplegia are complications of surgery for type A acute aortic dissection (TAAAD). Since the segmental arteries play a key role in SCI, this study evaluated the association between SCI and false lumen segmental arteries (FLSAs: segmental arteries originating from the false lumen).

Methods

The study included 101 consecutive TAAAD patients (mean age, 66±13; range, 34-89 years) who underwent surgery from January 2011 to April 2017. The diagnosis of TAAAD and the number of FSLAs were determined by preoperative computed tomography (CT). Patients were divided into two groups according to the number of FLSAs at the Th9-L2 level: Group A (n=13), ≥8 FLSAs; and group B (n=88), ≤7 FLSAs. Preoperative, perioperative, and postoperative findings were compared between the groups, and risk factors for SCI were evaluated.

Results

The frequency of preoperative paralysis was significantly higher in Group A than Group B (P=.0070). The overall incidence of postoperative SCI was 8% (8/101) and significantly

higher in Group A than Group B (5/13 (45%) vs 3/88 (4%), P<.0001). Hospital mortality was 8% (8/101) and significantly higher in Group A than Group B (3/13 (23%) vs 5/88 (6%), P=.0302). Multivariate analysis showed that the independent risk factors for SCI were ≥8 FLSAs at Th9-L2 (odds ratio [OR], 20.4; 95% confidence interval [95% CI], 3.34-124.9, P=.0011) and diabetes mellitus (OR, 22.3; 95% CI, 1.69-294.5; P=.0184).

Conclusions

In patients who underwent surgery for TAAAD, ≥8 FLSAs at the Th9-L2 levels on preoperative CT was a risk factor for SCI.

(249 words)

Keywords: acute aortic dissection, spinal cord ischemia

Introduction

Spinal cord ischemia (SCI) resulting in paraplegia is a devastating complication after treatment for aortic pathology.[1] Over the past two decades, the incidence of new postoperative SCI in type A acute aortic dissection (TAAAD) ranged from 0-10%, and it may be increasing with the development of new surgical techniques, including the branched and frozen elephant trunk (FET) techniques.[2] There are many reports on SCI after surgical procedures to treat thoracic and thoracoabdominal aortic pathology; however, there are few reports on SCI risk factors after procedures to treat TAAAD.[3] Etz et al. reported that staged repair reduced the rate of paraplegia after extensive thoracoabdominal aortic surgery.[4] Despite the differences in pathology and phase, we hypothesized that acute occlusion of many segmental arteries (SAs) may be associated with SCI, and may also be related to the number of false lumen SAs (FLSAs: SAs originating from the false lumen) and false lumen thrombosis at the thoracoabdominal aortic level. Recent advances in the visualization of SAs on computed tomography (CT) have allowed the analysis of FLSAs in the field of endovascular aortic repair.[5] Even in emergency cases, clear visualization of SAs on 64- or 320-row multidetector CT allows prompt determination of the number of FLSAs. Therefore, this study aimed to determine the relationship between the number of FLSAs and

postoperative SCI, and the neurological deficit of the lower limbs compared with other risk factors in cases with TAAAD.

Patients and Methods

Patients

From January 2011 to April 2017, 104 consecutive patients underwent surgical treatment for TAAAD at the Hiroshima University Hospital. Three patients were excluded because they were treated medically due to advanced age (n=2) or traumatic type A dissection concomitant with subarachnoid hemorrhage and acute respiratory distress syndrome (n=1). The institutional review board approved the use of patient data in this retrospective study. Informed consent (permission to use medical records for medical research) was obtained from all patients before the operation.

The diagnosis of TAAAD was confirmed by CT. Enhanced, thin-slice CT clearly revealed the extent of the dissection, the localization of the primary entry tear, the neck vessel, visceral and lower limb malperfusion, and the location of SAs among the intercostal and lumbar arteries. Preoperative CT was to classify the origin of each SA as the false lumen or

true lumen. The SAs from Th9 to L2 were investigated, and this range of SAs was determined from previous findings described by Takahashi et al. and Katayama et al.[6,7] We analyzed 12 SAs and divided patients into two groups according to the number of FLSAs: a group with ≥ 8 FLSAs at Th9-L2 (Group A, n=13) and a group with ≤ 7 FLSAs at Th9-L2 (Group B, n=88). Figure 1 shows the typical schema of TAAAD in Groups A and B. In Group A, the dissected false lumen passed from the left to right side of the descending aorta through the dorsal side of the aorta (Figure 1 (A)-(G)) in a clockwise rotation. In contrast, in Group B, the dissected false lumen passed the right side of the descending aorta (Figure 1 (H)-(M)) without rotation. Eight of 13 cases in Group A had a similar clockwise rotating pattern around the diaphragm to the level of the superior mesenteric artery.

Our principal strategy for the treatment of TAAAD consisted of resection of the primary entry. Replacement of the ascending aorta or total arch was chosen to resect the primary entry. FET implantation was added to total arch replacement when the primary entry was in the distal aortic arch. When the primary entry was located in the descending aorta, total arch replacement with or without FET implantation and secondary thoracic endovascular repair were considered, if necessary.

Based on the report of Scali et al.,[8] SCI was defined as any new lower extremity motor

and/or sensory deficit including stroke and peripheral neuropathy not attributable to any intracranial or peripheral nerve disorders. Preoperative, perioperative, and postoperative findings were compared between the two groups, and the risk factors for SCI were evaluated.

Surgical procedures

All patients underwent emergent surgery and did not receive cerebrospinal fluid drainage preoperatively. The surgical procedure for total arch replacement with FET for TAAAD was as follows. Under general anesthesia, a median sternotomy was performed. Cardiopulmonary bypass was established via right axillary arterial perfusion and double venous cannulation, and was initiated with general cooling to reach a rectal temperature of 28°C. When the rectal temperature decreased to 30°C, the left common carotid and left subclavian arteries were transected and selectively perfused at rates of 200 ml/min. When the rectal temperature reached 28°C, brief circulatory arrest and retrograde cardioplegia were performed. The brachiocephalic artery was clamped, and perfusion to the right axillary artery was decreased to a rate of 400 ml/min. The ascending aorta was transected. The size and length of the stent graft were determined during intraoperative measurement using a ball-shaped sizer that was

inserted into the true lumen of the descending aorta through a transverse incision in the aortic arch. The stent graft was positioned higher than the level of Th8 under the guidance of real-time transesophageal echocardiography. A four-branched graft was anastomosed end-to-end to the distal aortic arch with continuous 4/0 polypropylene sutures. Antegrade systemic perfusion from the four-branched graft and rewarming were started. The left subclavian artery and left common carotid artery were anastomosed to a respective branch of the prosthesis using the four-branched graft. A straight 3-cm graft was partly inserted into the sinotubular junction (STJ) and fixed to each of three commissures that were suspended using a 4/0 polypropylene suture with a felt strip. Subsequently, the tube graft was anastomosed to the STJ line using 4/0 horizontal mattress sutures. Finally, the brachiocephalic artery was reconstructed.

Although several procedures are performed at our institute, including ascending aorta replacement and total arch replacement with or without FET / conventional elephant trunk (ET) implantation, we use the same style of cannulation, extracorporeal circulation system (including antegrade selective cerebral perfusion), cooling protocol, and proximal anastomosis procedures. The distal anastomosis with ascending aorta replacement was performed using 4/0 prolene running sutures with internal and external felt reinforcements.

Statistical analysis

We first counted the number of FLSAs at the Th9-L2 level on preoperative CT and then performed receiver operating characteristic (ROC) curve analysis to estimate the optimal cutoff value of preoperative FLSAs to predict SCI. Our previous report showed that most of Adamkievicz arteries responsible for SCI are located below Th9, and graft deposition below Th9 is one of the risk factors for SCI in thoracic aortic surgery.[6,7] The results of our ROC curve analysis showed that the optimal cutoff value was 8, since this cutoff value gave the highest sensitivity and specificity (the area under the ROC curve was 0.81) (Supplementary Fig). We then divided all patients into two groups according to this optimal cutoff value: group A (preoperative FLSAs $\geq$ 8) and group B (preoperative FLSAs $\leq$ 7). Data are expressed as the mean ± standard deviation. Statistical comparisons were conducted using an unpaired Student's t-test for continuous variables and chi-square test for categorical variables. Univariate and multivariate logistic regression analysis were conducted to identify risk factors for SCI. In logistic regression analysis, the number of FLSAs was entered as both a continuous variable and dichotomized categorical variable. Multivariate logistic regression analysis included variables with a P value <0.1 in univariate analysis. A

P-value of <0.05 was considered statistically significant.

Results

Preoperative patient characteristics

Table 1 shows the preoperative patient characteristics. The mean age was 67.3±13.1 years, and 51 patients (51%) were male. There were no significant differences in the frequency of preoperative comorbidities. All cases in Group A, and 74 cases in Group B (84%) had dissection of the descending aorta; however, there was no significant difference between the two groups (P=.1213). Twenty-two patients (22%) were in shock. Forty-nine patients (49%) had pericardial effusion. There were no significant differences in the prevalence of the following conditions between the groups: consciousness disturbance; cerebral, coronary, and visceral malperfusion; and limb ischemia. The prevalence of pericardial bloody effusion in Group B was higher than that in Group A (P=.0016). The prevalence of limb paralysis for any reason including cerebral, spinal cord, and peripheral nerve dysfunction and muscle weakness was higher in Group A than in Group B (P=.0070).

Perioperative findings and SCI

Table 2 shows the perioperative findings. Replacement of the ascending aorta was performed in 59 cases (58%), and total arch replacement was performed in 42 cases (42%). Total arch replacement and FET implantation was more frequent in Group A than in Group B (P<.01). Eleven cases (11%) underwent concomitant cardiac procedures that included aortic valve replacement (n=1), Bentall procedure (n=4), and coronary artery bypass grafting (n=6). Hospital mortality was 8% (8 cases). Hospital mortality in Group A was significantly higher than that in Group B (3/13(23%) vs. 5/88(6%), P=.0303). In 7 cases, SCI was not evaluated because of early death. The overall incidence of SCI was 8% (8/101). The incidence of SCI in Group A was higher than that in Group B (5/13 (45%) vs. 3/88 (4%), P<.0001).

Characteristics of cases with ≥ 8 FLSAs at Th9-L2

Table 3 shows the characteristics of 13 cases in Group A with ≥ 8 FLSAs at the Th9-L2 level. In 8 cases, postoperative CT showed that the false lumen was patent. Two of the 8 cases died postoperatively. In case 7, there was malposition of the FET inserted into the false lumen, resulting in visceral ischemia and death on the 3rd post-operative day. Two

cases suffered from paraparesis. In case 6, the non-stented prosthesis inserted into the distal aorta resulted in stenosis. The patient had paraparesis with a distal arterial pressure of 55/30 mmHg. Additional thoracic endovascular aortic repair restored distal pressure, and the patient recovered to start walking with a slight palsy. In 5 cases, postoperative CT showed that the false lumen was occluded. In case 9, the patient underwent total arch replacement with FET implantation. Postoperative CT showed that the false lumen at the thoracic descending aorta completely disappeared, and communications between aortic true lumen and intercostal arteries were restored. However, the other 4 cases suffered from paraplegia and did not recover. In case 13, the patient had a descending aortic aneurysm and developed a retrograde type A acute aortic dissection, and there were also 12 FLSAs at Th9-L2 and complete false lumen thrombosis resulting in paraplegia preoperatively. Although the patient underwent replacement of the ascending aorta and cerebrospinal fluid drainage postoperatively, the paraplegia continued.

False lumen thrombosis after ET or FET implantation

All FET (n=29) or ET (n=13) implantation was performed in patients with descending aortic dissection. There was no significant difference in the rate of false lumen thrombosis in the

thoracic aorta at Th5-8 between the cases with FET (17/29 (59%)) and those with ET (5/13 (38%), p=0.2265). The rate of aortic false lumen thrombosis of the thoracic aorta at Th9-12 and the abdominal aorta at L1-2 in cases with FET was relatively higher than that in cases with ET (Th9-12: FET, 15/29 (52%) vs ET, 3/13 (23%), p=0.0829; L1-2: FET, 7/29 (24%) vs ET, 0/13 (0), p=0.0523).

Risk factors for SCI

Because 7 cases died postoperatively before evaluation of SCI, 94 cases were included in the analysis. Univariate logistic analysis revealed that diabetes mellitus, preoperative limb ischemia, the number of FLSAs at Th9-L2, ≥ 8 FLSAs at Th9-L2, FET implantation, and false lumen thrombosis of the thoracic aorta at the Th5-8 level were significant risk factors for SCI (P<.05, Table 4). Multivariate logistic regression analysis revealed that ≥ 8 FLSAs at Th9-L2 (odds ratio [OR], 27.6; 95% confidence interval [95% CI], 4.30-176.5; P=.0005) and diabetes mellitus (OR, 22.1; 95% CI, 1.61-302.8; P=.0205) were independent risk factors for SCI (area under the ROC curve, 0.84).

Discussion

This study demonstrated that, in patients with TAAAD, the segmental communicating artery with a dorsal false lumen and false lumen thrombosis may be associated with SCI and impairment of lower limb function. Recently, several reports have described paraplegia and paraparesis after TAAAD surgery; however, there are few reports on SCI risk factors.[2] Our study may indicate a new concept related to surgery for TAAAD.

Incidence of SCI

Postoperative SCI is one of the most serious complications of thoracic and/or abdominal aortic surgery, with an incidence of up to 25%.[9] SCI during the postoperative period following acute aortic dissection is less common. There were no descriptions of SCI in most patients undergoing replacement of ascending aorta and hemiarch repair published before 2000.[10-12] Conzelmann et al. reported that paraparesis and/or paraplegia occurred in 55 of 2137 patients (2.6%) who received an aortic operation, including 69% who had ascending aorta replacement.[13] A recent report and a meta-analysis in patients undergoing extended arch repair, which included data published between 2001 and 2016, showed that the incidence of SCI ranged from 0-10%, and a pooled analysis demonstrated an SCI incidence of 3.5%.[2,12,14] These reports suggest that SCI after TAAAD surgery may occur in any type

of surgery and occurs more frequently in extended aortic arch repair.

Mechanisms of SCI

SCI in thoracic and thoracoabdominal aortic surgery and endovascular repair can be caused by a variety of mechanisms.[3,6] These include the following: prolonged aortic cross-clamping; decrease in mean arterial pressure; increase in cerebrospinal fluid drainage pressure; loss of an essential SA; covering of the subclavian, visceral, and hypogastric arteries; and insufficient distal perfusion pressure during aortic clamping. There are few reports on the pathophysiology of SCI after surgery for acute aortic dissection. Martens et al. reported no independent risk factors for SCI in patients who underwent total arch repair, including patients with dissection and/or aneurysm.[15] Our previous study showed that for the distal position of the stent graft below Th9, a mean pressure <70 mmHg during the postoperative ICU stay and diabetes were significant predictors of SCI after FET implantation for thoracic aortic pathology, including dissection and aneurysm.[7]

FET and SCI

The reported an incidence of SCI after FET implantation of 8-9%, and this appears to be significantly higher than that observed after ET implantation (0.4-2.8%).[16-19] Martens et al. reported an SCI incidence of 5% (8/160) in FET cases, and FET length was a risk factor for

SCI that reached borderline significance (P=0.053).[15] The authors also mentioned that SCI predominantly occurred with FET implantation in acute aortic dissection without cerebrospinal fluid drainage. However, there was no significant difference in the incidence of SCI after FET implantation compared with conventional arch procedures for TAAAD in several single-center studies and a meta-analysis.[2,20] Pochettino et al. reported that the incidence of SCI after the FET procedure for TAAAD was 8.3% and not significantly different compared with conventional arch procedures (2.4%).[21] In the current study, we used the FET procedure in selected patients (i.e., narrow descending aorta with malperfusion), with a short FET length reaching above the Th6 level (the level of the aortic valve evaluated by transesophageal echocardiography). Furthermore, FET implantation was associated with a higher incidence of SCI in univariate analysis (with FET, 5/26 (19%); without FET 3/68 (4%), P=0.0213).

False lumen thrombosis and SCI

False lumen thrombosis of the descending aorta at the initial surgery for TAAAD prevents a secondary procedure for a descending dilated aorta.[22,23] Jakob et al. reported that false lumen thrombosis of the descending aorta with the FET technique was more frequent (90%) than with conventional hemiarch or total arch replacement (11%, P<0.01), and there were

no cases of SCI in either group.[24] Martens et al. reported that SCI predominantly occurred in chronic dissection with false lumen thrombosis.[15] These data suggest that false lumen thrombosis would not be required to enlarge the dissected aorta in patients with a false lumen; however, the false lumen should be kept patent in some cases. In the present study, FET implantation and postoperative false lumen thrombosis ~~at the middle~~ of the thoracic aorta at the Th5-8 level were associated with a higher incidence of SCI in univariate analysis. The false lumen of the descending aorta should be thrombosed to improve long-term prognosis; however, the interaction between FLSAs and false lumen thrombosis should be considered preoperatively.

Our current strategies to prevent SCI are as follows.

1) If preoperative CT shows a patent false lumen of the descending aorta and there are ≥ 8 FLSAs at Th9-L2, FET implantation should be avoided, and replacement of the ascending aorta or total arch replacement with a short, non-frozen ET should be considered. We intend to maintain the patent dorsal false lumen at the Th9-L2 level.

2) In cases other than the above, any procedures including replacement of the ascending aorta, aortic arch with or without FET or non-frozen ET implantation should be selected in accordance with the dissecting lesion; however, the FET should be inserted above the Th6

level.

Our previous study showed that the distal position of the stent graft below Th9 was a significant independent risk factor for SCI, and the FET was inserted above Th6 to maintain a margin of safety.[5]

Limitations

There are several limitations in this study. First, this was a retrospective study, and there were a small number of cases. There were only 8 SCI cases in our study, and this was too few for multivariate analysis to have sufficient statistical power; however, the calculated statistical power for ≥ 8 FSLAs to predict SCI was 0.905. This indicated that there was a sufficient number of patients, and the power was strengthened by the goodness-of-fit test in multivariate analysis. Second, in the present study, most cases were evaluated by 64-row multidetector CT in our hospital; however, in some cases, CT was performed in a different hospital using less than 64 rows, and the resolution was not satisfactory.

Conclusions

In patients undergoing surgery for TAAAD, techniques have been developed for treating

false lumen thrombosis of the distal arch and descending aorta to reduce the possibility of reoperation and/or the need for additional procedures to dilate the dissected aorta. In the present study, patients with ≥ 8 FLSAs at the Th9-L2 level were likely to undergo total arch replacement with FET implantation, and operative procedure should be selected carefully in such patients. However, postoperative SCI occurred in patients with any procedure. Our study showed that ≥ 8 FLSAs at the Th9-L2 level on preoperative CT was a risk factor for SCI, and all patients with this condition should be carefully managed for a better outcome.

References

1 Safi HJ, Miller CC 3rd, Huynh TT, Estrera AL, Porat EE, Winnerkvist AN, et al. Distal aortic perfusion and cerebrospinal fluid drainage for thoracoabdominal and descending thoracic aortic repair: ten years of organ protection. Ann Surg. 2003;238:372-80.

2 Takagi H, Umemoto T; ALICE Group. A Meta-Analysis of Total Arch Replacement With Frozen Elephant Trunk in Acute Type A Aortic Dissection. Vasc Endovascular Surg. 2016;50:33-46.

3 Etz CD, Weigang E, Hartert M, Lonn L, Mestres CA, Di Bartolomeo R, et al. Contemporary spinal cord protection during thoracic and thoracoabdominal aortic surgery and endovascular aortic repair: a position paper of the vascular domain of the European Association for Cardio-Thoracic Surgery†. Eur J Cardiothorac Surg. 2015;47:943-57.

4 Etz CD, Zoli S, Mueller CS, Bodian CA, Di Luozzo G, Lazala R, et al. Staged

repair significantly reduces paraplegia rate after extensive thoracoabdominal aortic aneurysm repair. J Thorac Cardiovasc Surg. 2010;139:1464-72.

5 Ge YY, Guo W, Cheshire N, Liu XP, Jia X, Xiong J, et al. Preoperative thoracic false lumen branches relate to aortic remodeling after thoracic endovascular aortic repair for DeBakey IIIb aortic dissection. J Vasc Surg. 2017;65:659-68.

6 Takahashi S, Orihashi K, Imai K, Mizukami T, Takasaki T, Sueda T. Cold blood spinoplegia under motor-evoked potential monitoring during thoracic aortic surgery. J Thorac Cardiovasc Surg. 2011;141:755-61.

7 Katayama K, Uchida N, Katayama A, Takahashi S, Takasaki T, Kurosaki T, et al. Multiple factors predict the risk of spinal cord injury after the frozen elephant trunk technique for extended thoracic aortic disease. Eur J Cardiothorac Surg. 2015;47:616-20.

8 Scali ST, Wang SK, Feezor RJ, Huber TS, Martin TD, Klodell CT, et al. Preoperative prediction of spinal cord ischemia after thoracic endovascular aortic repair. J

Vasc Surg. 2014;60:1481-90.

9 Jacobs MJ, Mommertz G, Koeppel TA, Langer S, Nijenhuis RJ, Mess WH, et al. Surgical repair of thoracoabdominal aortic aneurysms. J Cardiovasc Surg (Torino). 2007;48:49-58.

10 Ehrlich MP, Ergin MA, McCullough JN, Lansman SL, Galla JD, Bodian CA, et al. Results of immediate surgical treatment of all acute type A dissections. Circulation. 2000;102(19 Suppl 3):III248-52.

11 Pugliese P, Pessotto R, Santini F, Montalbano G, Luciani GB, Mazzucco A. Risk of late reoperations in patients with acute type A aortic dissection: impact of a more radical surgical approach. Eur J Cardiothorac Surg. 1998;13:576-80.

12 Bachet J, Goudot B, Dreyfus GD, Brodaty D, Dubois C, Delentdecker P, et al. Surgery for acute type A aortic dissection: the Hopital Foch experience (1977-1998). Ann Thorac Surg. 1999;67:2006-9.

13 Conzelmann LO, Weigang E, Mehlhorn U, Abugameh A, Hoffmann I, Blettner M, et al; GERAADA Investigators. Mortality in patients with acute aortic dissection type A: analysis of pre- and intraoperative risk factors from the German Registry for Acute Aortic Dissection Type A (GERAADA). Eur J Cardiothorac Surg. 2016;49:e44-52.

14 Smith HN, Boodhwani M, Ouzounian M, Saczkowski R, Gregory AJ, Herget EJ, et al. Classification and outcomes of extended arch repair for acute Type A aortic dissection: a systematic review and meta-analysis. Interact Cardiovasc Thorac Surg. 2017;24:450-459.

15 Martens A, Beckmann E, Kaufeld T, Umminger J, Fleissner F, Koigeldiyev N, et al. Total aortic arch repair: risk factor analysis and follow-up in 199 patients. Eur J Cardiothorac Surg. 2016;50:940-8.

16 Jakob H, Tsagakis K, Pacini D, Di Bartolomeo R, Mestres C, Mohr F, et al. The International E-vita Open Registry: data sets of 274 patients. J Cardiovasc Surg (Torino) 2011;52:717–23.

17 Pacini D, Tsagakis K, Jakob H, Mestres CA, Armaro A, Weiss G, et al. The frozen elephant trunk for the treatment of chronic dissection of the thoracic aorta: a multicenter experience. Ann Thorac Surg 2011;92:1663–70.

18 Safi HJ, Miller CC III, Estrera AL, Villa MA, Goodrick JS, Porat E, et al. Optimization of aortic arch replacement: two-stage approach. Ann Thorac Surg 2007;83:S815–8.

19 Etz CD, Plestis KA, Kari FA, Luehr M, Bodian CA, Spielvogel D, et al. Staged repair of thoracic and thoracoabdominal aortic aneurysms using the elephant trunk technique: a consecutive series of 215 first stage and 120 complete repairs. Eur J Cardiothorac Surg 2008;34:605–14.

20 Shrestha M, Beckmann E, Krueger H, Fleissner F, Kaufeld T, Koigeldiyev N, et al. The elephant trunk is freezing: The Hannover experience. J Thorac Cardiovasc Surg. 2015;149:1286-93.

21 Pochettino A, Brinkman WT, Moeller P, Szeto WY, Moser W, Cornelius K, et al. Antegrade thoracic stent grafting during repair of acute DeBakey I dissection prevents development of thoracoabdominal aortic aneurysms. Ann Thorac Surg. 2009;88:482-9.

22 Tsai MT, Wu HY, Roan JN, Tsai YS, Hsieh PC, Yang YJ, et al. Effect of false lumen partial thrombosis on repaired acute type A aortic dissection. J Thorac Cardiovasc Surg. 2014;148:2140-6.e3.

23 Fattouch K, Sampognaro R, Navarra E, Caruso M, Pisano C, Coppola G, et al. Long-term results after repair of type a acute aortic dissection according to false lumen patency. Ann Thorac Surg. 2009;88:1244-50.

24 Jakob H, Tsagakis K, Tossios P, Massoudy P, Thielmann M, Buck T, et al. Combining classic surgery with descending stent grafting for acute DeBakey type I dissection. Ann Thorac Surg. 2008;86:95-101.

Notes

Conflicts of interest: None declared.

Table 1. Patient preoperative characteristics

Variables	All (n=101)	Group A FLSAs at Th9-L2 ≥8 (n=13)	Group B FLSAs at Th9-L2 ≤ 7 (n=88)	P-value
Age (years, mean±SD)	67.3±13.1	61.4±15.1	68.1±12.7	.0836
Male (n, %)	51 (51)	8 (62)	43 (49)	.3936
Body mass index (kg/m^2)	23.3±3.3	24.8±4.4	23.0±3.0	.0607
Body mass index ≥ 30 kg/m^2 (n, %)	2 (2)	2 (15)	0	.0002
Preoperative comorbidities				
Hypertension (n, %)	76 (75)	8 (62)	68 (78)	.2198
Hyperlipidemia (n, %)	14 (14)	2 (15)	12 (14)	.8648
Diabetes mellitus (n, %)	4 (4)	1 (8)	3 (3)	.4598
COPD (n, %)	3 (3)	1 (8)	2 (2)	.2826
Smoking (n, %)	28 (28)	3 (23)	25 (28)	.6885
Renal insufficiency (Cr>1.5 mg/dL) (n,%)	16 (16)	3 (23)	13 (15)	.4440
Hemodialysis (n, %)	5 (5)	0	5 (6)	.3780
Cerebrovascular disease (n, %)	10 (10)	1 (8)	9 (10)	.7752
Marfan syndrome (n, %)	4 (4)	1 (8)	3 (3)	.4598
Dissection of descending aorta (n, %)	87 (86)	13 (100)	74 (84)	.1213

Preoperative status				
Shock status (BP<60 mmHg) (n, %)	22 (22)	3 (23)	19 (22)	.9036
Pericardial bloody effusion (n, %)	49 (49)	1 (8)	48 (55)	.0016
Consciousness disturbance	15 (15)	2 (15)	13 (15)	.9668
Cerebral malperfusion (n, %)	6 (6)	1 (8)	5 (6)	.7747
Coronary malperfusion (n, %)	4 (4)	0	4 (5)	.2886
Visceral ischemia (n, %)	2 (2)	1 (8)	1 (1)	.1133
Limb ischemia (n, %)	9 (9)	2 (15)	6 (7)	.2857
Paralysis (n, %)	10 (10)	4 (31)	6 (7)	.0070

FLSA, false lumen segmental artery; COPD, chronic obstructive pulmonary disease; Cr, Creatine

Table 2. Perioperative factors associated with neurological deficit

Variables	All (n=101)	Group A FLSAs at Th9-L2 ≥8 (n=13)	Group B FLSAs at Th9-L2 ≤ 7 (n=88)	P-value
Procedures				
Total arch replacement (n, %)	42(42)	10 (77)	32 (36)	.0056
Frozen elephant trunk (n, %)	29(29)	10 (77)	19 (22)	<.0001
Concomitant procedures				
Aortic valve replacement (n, %)	1(1)	0	1(1)	.6993
Bentall procedure (n, %)	4(4)	1(8)	3(3)	.4598
CABG (n, %)	6(6)	0	6(7)	.3317
Operation time (min)	372.6±142.2	391.1±103.2	369.9±147.3	.6182
Cardiopulmonary bypass time (min)	213.1±104.5	223.0±79.4	211.8±107.8	.7295
Aortic cross clamp time (min)	112.0±39.4	117.5±48.7	111.3±38.2	.6114
Circulatory arrest time (min)	40.4±16.8	38.2±7.4	40.7±17.8	.6158
Postoperative findings				
Hospital death (n, %)	8(8)	3(23)	5(6)	.0302
SCI (n, %)	8(8)	5(38)	3(4)	<.0001
Thrombosis of the false lumen				
~~Middle t~~Thoracic ~~descending~~ aorta at the Th5-8 level (n, %)	45(45)	6(46)	39(44)	.9012

~~Distal t~~Thoracic ~~descending~~ aorta at the Th9-12 level (n, %)	41(41)	5(38)	36(41)	.8668
Abdominal aorta at L1-2~~SMA~~ level (n, %)	24(24)	4(31)	20(23)	.5248

CABG, coronary artery bypass grafting; SCI, spinal cord ischemia; SMA, superior mesenteric artery.

Table 3. Cases with > 8 SAs (Th9-L2) directly communicating with the false lumen

			Preoperative status		Number of					
Case	Age	Entry	shock	Malperfusion	FLSAs	Preop SCI	Procedure	FL	Postop SCI	Outcome
1	63	Asc	N	N	12	N	Asc	Patent	N	Alive
2	62	Des	N	N	10	N	TAR, FET	Patent	N	Alive
3	62	Arch	N	Vis, LL	8	N	TAR, FET	Patent	N	Alive
4	81	Asc	N	N	11	N	Asc	Patent	N	Alive
5	62	Des	N	N	12	N	TAR, FET	Patent	N	Alive
6	63	Arch	N	N	8	N	TAR, FET	Patent	Y, paraparesis, trans	Alive, add TEVAR
7	34	Asc	N	N	12	N	Bentall, TAR, FET	Patent	-	Died (MOF, 3POD)
8	56	Arch	N	RCCA, LCCA, LL	12	Y	TAR, FET	Patent	-	Died (CH, 5POD)
9	50	Asc	N	RCCA	9	N	TAR, FET	Occluded	N	Alive
10	39	Des	N	Vis, LL	10	Y	TAR, FET	Occluded	Y, paraplegia	Alive
11	62	Arch	N	N	12	N	TAR, FET	Occluded	Y, paraplegia	Alive
12	89	Des	N	N	9	N	Asc, FET	Occluded	Y, paraplegia	Alive
13	75	Des	N	N	12	Y	Asc	Occluded	Y, paraplegia	Died(ruptured DA)

Des, descending; Asc, ascending; N, no; Y, yes; Vis, visceral; LL, lower limb; RCCA, right common carotid artery; LCCA, left common carotid artery; TAR, total arch replacement; TEVAR, thoracic endovascular repair; FET, frozen elephant trunk; ET, elephant trunk; DA, descending aorta; CH, cerebral hemorrhage; POD, postoperative day; trans, transient; MOF, multiple organ failure; Preop, preoperative; Postop, postoperative

Table 4. Logistic regression analysis for SCI after the procedure for acute type A aortic dissection

Variables	SCI (n=8)	No SCI (n=86)	P value	Univariate			Multivariate		
				OR	95% CI	P value	OR	95% CI	P value
Sex (male, %)	7 (88)	40 (47)	.0266	8.0	0.95-68.3	.0558			
Body mass index ≥30 (n,%)	2 (25)	0	<.0001	57395830	-	.9899			
Diabetes mellitus (n, %)	2 (25)	2 (2)	.0024	14.0	1.67-117.5	.0151	22.1	1.61-302.8	0205
Preoperative limb ischemia (n,%)	2 (25)	4 (5)	.0243	6.8	1.03-45.2	.0461			
Number of FLSAs at Th9-L2 (n,%)	6.9±4.6	2.2±3.2	.0002	1.3	1.11-1.62	.0021			
FLSAs at Th9-L2 ≥8 (n,%)	5 (45)	6 (55)	<.0001	22.2	4.25-116.2	.0002	27.6	4.30-176.5	.0005
Frozen elephant trunk (n,%)	5 (63)	5 (63)	.0213	5.2	1.14-23.4	.0336			
Location of false lumen thrombosis									
~~Middle t~~Thoracic aorta at Th5-8 level (n,%)	7 (88)	36 (42)	.0132	9.7	1.15-82.5	.0371			
~~Distal t~~Thoracic aorta at Th9-12 level (n,%)	6 (75)	33 (38)	.0443	4.8	0.92-25.3	.0631			
Abdominal aorta at L1-2~~SMA~~ level (n,%)	4 (50)	19 (22)	.0791	3.5	0.81-15.4	.0944			

SCI, spinal cord ischemia; SMA superior mesenteric artery

Figure legends

Figure 1. A typical case of aortic dissection in Group A; the number of segmental arteries originating from false lumen (FLSA) was ≥ 8. The site of entry tear is the ascending aorta (A) and the false lumen passes through the great curvature at the aortic arch and dorsal to right side at the thoracic descending aorta (B, C, D). It then passes the dorsal side around the diaphragm to the superior mesenteric artery level (E), the right side at the abdominal aorta (F), and terminates at the right external iliac artery (G). A typical case of aortic dissection without a dorsal false lumen (Figure 1B). The site of entry tear is the distal arch, the ascending aorta is partially thrombosed (H), and the false lumen passes through the great curvature at the aortic arch. It then passes the right side at the thoracic aorta (I, J), the right side at the celiac, superior mesenteric and renal artery level (L), and the ventral side of the abdominal aorta (M).

Supplement

Figure legend

Figure. The area under the receiver operating characteristic curve for FLSAs at Th9-L2. The optimal cut-off value of FLSAs at Th9-L2 was ≥ 8 (sensitivity, 62.5%; specificity, 93.0%).

gure 1.

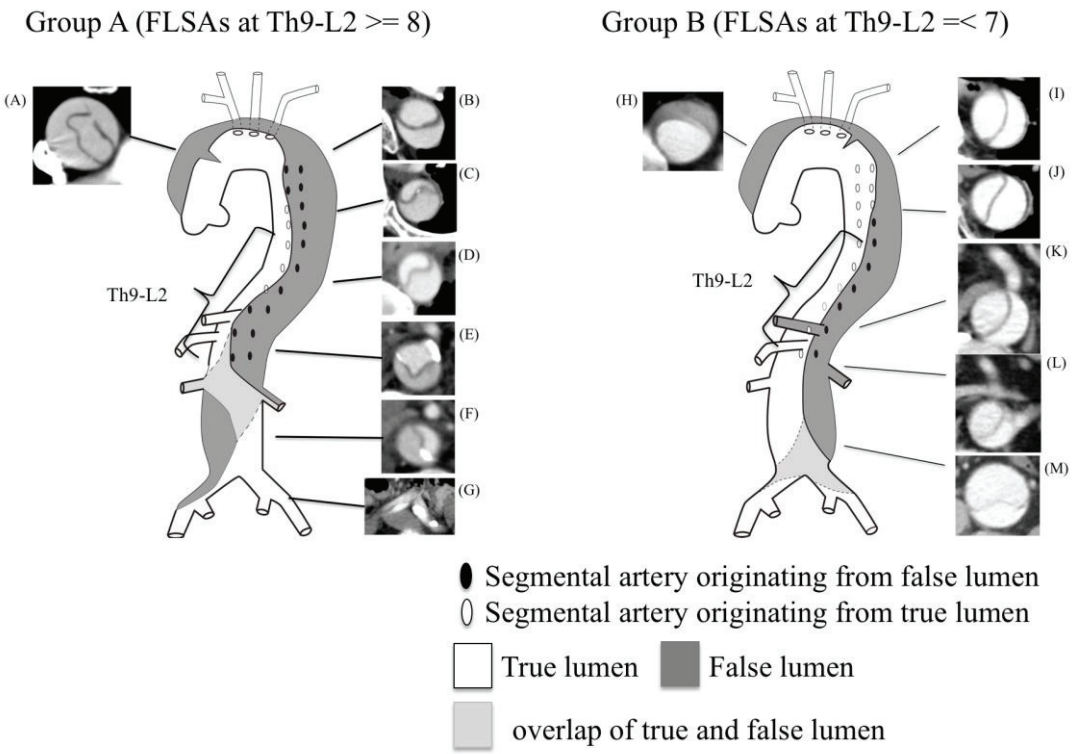
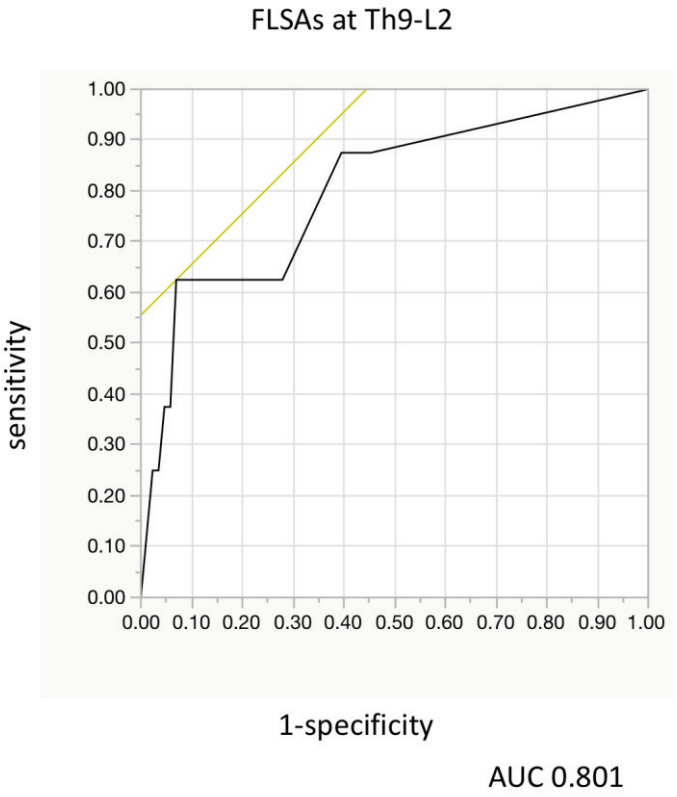


Figure .


FLSAs at Th9-L2
sensitivity
1.00
0.90
0.80
0.70
0.60
0.50
0.40
0.30
0.20
0.10
0.00
0.00 0.10 0.20 0.30 0.40 0.50 0.60 0.70 0.80 0.90 1.00
1-specificity
AUC 0.801