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ORIGINAL ARTICLE

Spinal cord ischemia after endovascular aortic repair versus open surgical repair for descending thoracic and thoracoabdominal aortic aneurism

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Abstract
Purpose Thoracic endovascular aortic repair (TEVAR) an emerging less invasive alternative to surgery, is now being increasingly employed, but spinal cord ischemia (SCI) is still a threat with this procedure. Delayed paraplegia has been frequently observed after TEVAR, suggesting there may be different courses of SCI between TEVAR and the conventional open surgical repair (OSR) of thoracic and thoracoabdominal aneurysms. Therefore, we conducted a study to investigate the risk factors for and the course of SCI after TEVAR and OSR.
Methods We studied a series of 414 OSR and 94 TEVAR patients prospectively. Postoperative motor function, sensory disturbance, and bladder disturbance were assessed daily to evaluate the course of SCI. Previously reported risk factors for SCI were investigated.
Results Spinal cord ischemia occurred in 6 patients (6.4 %) in the TEVAR group, and in 18 patients (4.3 %) in the OSR group, resulting in no significant difference ($p = 0.401$). A greater percentage of patients ($n = 4$, 66.7 %) with SCI in the TEVAR group had a delayed onset, compared with 16.7 % ($n = 3$) in the OSR group ($p = 0.038$). The rate of recovery of walking function after SCI and the incidence of sensory disturbance and bladder dysfunction was similar in the two groups. Multivariate analysis demonstrated that, in the TEVAR group, the stent length of aortic coverage was a significant risk factor for SCI.
Conclusion The incidence of SCI was similar in the OSR and TEVAR groups, but delayed SCI occurred more frequently in the TEVAR group. Except for the delayed onset of SCI, SCI showed a similar course of recovery in the two groups.

Keywords Spinal cord ischemia · Motor evoked potentials · Thoracic aneurysm surgery

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Introduction

Spinal cord ischemia (SCI) is one of the most devastating complications of thoracic aortic aneurysm (TAA) and thoracoabdominal aortic aneurysm (TAAA) repair. Patients with postoperative SCI have a mortality rate three times that of patients without SCI [1]. Thoracic endovascular aortic repair (TEVAR) is an emerging less invasive alternative to surgery, with a reduced incidence of SCI (3%) as compared with that in conventional open surgical repair (OSR) (14 %) [2].

However, little information exists concerning the severity of SCI after either TEVAR or OSR. It is known that TEVAR is associated with delayed SCI [3], suggesting that SCI may follow different courses after TEVAR and OSR. Recovery of ambulation is one of the main goals in patients with SCI. In addition, recovery of bladder function could improve quality of life. The purpose of this study was to compare the clinical presentation of SCI between patients undergoing OSR and those undergoing TEVAR. This study also attempted to identify risk factors for SCI after TEVAR and OSR.

Patients, materials, and methods

The study population consisted of a series of 414 patients who underwent open TAA and TAAA surgical repair (June 1998–June 2010) and 94 patients who underwent TEVAR for part of or the entire distal descending aorta (May 2007–June 2010). In the TEVAR group, the distal descending aorta included the segment between T7 and L2. Criteria for the TEVAR group included a high risk for open surgery or that the patient was considered a nonsurgical candidate, or a combination of these. The patients were senescent, debilitated and presented comorbidities. Our institutional ethics committee approved our study.

All patients received general anesthesia. After 3 min of pre-oxygenation, anesthesia was induced with fentanyl 4 μg/kg, midazolam 0.1 mg/kg, and propofol 1 mg/kg. Rocuronium 1 mg/kg or vecuronium 0.2 mg/kg was given for tracheal intubation. Total intravenous anesthesia (TIVA) with propofol 4–6 mg/kg/min and remifentanil 0.2–0.5 μg/kg/min was used to maintain general anesthesia.

Motor evoked potential (MEP) monitoring

A recording device (Neuropack; Nihon Kohden, Tokyo, Japan) was used for recording MEPs. The stimulating electrodes were positioned at C3 and C4. Trains of five stimuli. 50 μs in duration. were delivered at a voltage of 500 V. MEPs were monitored in the right thenar muscle, the bilateral flexor hallucis brevis muscles, and the anterior tibial muscle contralateral to the femoral artery cannulation site. In the TEVAR group, MEP was monitored every 5 min starting immediately after the stent graft was placed. In the OSR group, MEP was monitored in the same way as in the TEVAR group and recorded immediately after the aorta was clamped and every 5 min until the aorta was unclamped. MEP amplitude below 25 % of the baseline was considered to indicate SCI. In the OSR group, when a critical reduction in MEP amplitude was observed, reimplantation of the lumbar or intercostal arteries was performed. Patients in this group in whom deep hypothermic circulatory arrest (DHCA) was used were excluded from MEP evaluation due to the disappearance of MEP waves during DHCA.

Cerebrospinal fluid drainage

In the OSR group, a cerebrospinal fluid drainage (CSFD) tube was placed preoperatively for type 2 TAAA; for other types of aortic aneurysm, a CSFD tube was placed when SCI was found postoperatively. In the TEVAR group, a CSFD tube was placed preoperatively if a stent graft would be covering the area where the Adamkiewicz artery (AKA) was included. In other patients, a CSFD tube was placed when SCI was found postoperatively. When the MEP amplitude decreased below 25 % of the baseline intraoperatively or after paraplegia, a CSFD tube inserted was opened to maintain cerebrospinal fluid pressure more than 14 cmH_2O.

Surgical procedure

In the OSR group, the aneurysm was approached through a left thoracic or thoracoabdominal incision. In patients with true aneurysm of the descending aorta, surgeons cross-clamped the proximal portion of the descending aorta. During aortic cross-clamping, distal aortic perfusion at a pressure above 60 mmHg was maintained by partial cardiopulmonary bypass, consisting of a heparin-coated femorofemoral circuit, with mild hypothermia, at 32–34 °C. In patients with chronic type B dissection of the descending aorta or true aneurysms where there was an insufficient proximal portion to cross-clamp, systemic hypothermia was induced by cardiopulmonary bypass (CPB) and the target temperature was set at 18 °C for DHCA. When ventricular fibrillation occurred, the left ventricle was vented through the apex if necessary for decompression. Cooling was continued until the nasopharyngeal temperature reached 18 °C. After the patient was placed in the head-down position and 20–40 mEq KCl was administered into the venous reservoir, circulatory arrest in the upper body was established.

The relevant intercostal arteries (particularly around T9 or T10) connecting to the AKA, as demonstrated by magnetic resonance angiography, were preserved or reimplanted during the CPB. When the aneurysm involved a long segment (such as in TAAA), the clamps were placed sequentially, if possible ("segmental clamp technique").

In the TEVAR group, TEVAR was carried out under general anesthesia. In patients who had extensive/multiple aneurysms from the aortic arch to the descending aorta, total arch replacement was performed using elephant trunk (ET) implantation prior to TEVAR. In patients who underwent aortic surgery, an artificial graft was used to create a proximal landing zone. When there was an insufficient portion for a landing zone, a carotid-subclavian bypass was performed to create a landing zone. An endovascular graft was inserted from the femoral artery. Prior to the placement of the endovascular graft in the descending aorta, MAP was raised to more than 120 mmHg to prevent SCI. When major type 2 leakages were observed, an aortic balloon was inflated to reduce the leakage.

Postoperative management

Postoperative neurologic deficits were prospectively evaluated by certified neurologists. Neurologic deficits were

excluded if they resulted from confirmed perioperative strokes or prior surgery. Neurologic deficits at discharge were graded according to the modified Tarlov scale [4] (Table 1). Paraplegia and paraparesis observed immediately upon recovery from anesthesia were defined as immediate neurologic deficits. Deficits occurring after a period of normal neurologic function were classified as delayed motor deficits. Sensory disturbance and dysuria at discharge were categorized dichotomously (0: asymptomatic, 1: symptomatic).

We also investigated the influence of the following factors as possible predictors of postoperative SCI: age, sex, diabetes mellitus, history of hypertension, hyperlipidemia, smoking, chronic obstructive pulmonary disease, renal dysfunction (serum creatinine >1.2 mg/dl), coronary artery disease, cerebrovascular disease, maximum diameter of aneurysm, acute or chronic dissection (with acute dissection defined as surgery performed within 14 days of the onset of pain and chronic dissection defined as surgery performed after 14 days), rupture, emergency operation, re-do of aortic surgery, length of endovascular stent coverage (in TEVAR), and left subclavian artery patency (in TEVAR).

The length of endovascular stent coverage was measured on computed tomography angiography using curved planar reformation images processed in a workstation (GE Advantage workstation 4.3; GE Healthcare, Fairfield, CT, USA) [5]. After total arch replacement with a multi-branch graft, the length of aortic coverage was measured from the distal anastomosis.

Statistical analysis

Statistical analysis was performed using SPSS version 16 for Windows (SPSS, Chicago, IL, USA). Data for the two groups were summarized as means ± SD or as percentages. Risk factors were evaluated for association with SCI using univariate analysis; categorical variables were analyzed using the χ^2 test or Fisher's exact test and continuous variables were analyzed using Student's *t*-test. Risk factors that emerged with *p* levels of <0.2 were included in multiple logistic regression models with stepwise model selection; *p* values of <0.05 were considered to indicate statistical significance.

Table 1 Modified Tarlov scale [4]

Score	Motor function	Deficits
0	No lower extremity movement	Paraplegia
1	Lower extremity motion without gravity	Paraplegia
2	Lower extremity motion against gravity	Paraplegia
3	Able to stand without assistance	Paraparesis
4	Able to walk with assistance	Paraparesis
5	Normal	Normal

Results

The study cohort consisted of 94 patients after TEVAR and 414 patients after OSR. Table 2 summarizes the clinical and demographic characteristics of all patients. Patients who underwent TEVAR were significantly older than those who underwent OSR. A history of renal dysfunction or coronary artery disease was significantly more prevalent in the TEVAR group. Patients with descending TAAs were observed more frequently in the TEVAR group owing to the more complex procedure of stent grafting for TAAAs than for descending TAAs. SCI occurred in 6 patients (6.4 %) in the TEVAR group, and in 18 patients (4.3 %) in the OSR group ($p = 0.401$). The AKA was covered by the stent graft in 3 of the 6 patients with SCI in the TEVAR group, but in the other 3 patients the AKA was not covered.

Analysis of risk factors for postoperative spinal cord ischemia

Tables 3 and 4 show the results of analysis of risk factors for postoperative SCI. The univariate analysis revealed that sex and the stent length of aortic coverage were significant variables in the TEVAR group, and coronary artery disease and cerebrovascular disease were significant variables in the OSR group. The multivariate analysis demonstrated that the stent length of aortic coverage was a significant preoperative risk factor for SCI.

The course of spinal cord ischemia

To clarify the course of SCI, we compared patients with SCI in the TEVAR versus OSR groups (Table 5). Deep hypothermic circulatory arrest was used in three patients with SCI in the OSR group. In these patients, the MEP amplitude became undetectable as the bladder temperature decreased. We excluded these three cases when reviewing MEP changes. MEP amplitude decreased to <25 % of the baseline in two patients with SCI (33.3 %) in the TEVAR group and in 12 of 15 patients (80 %) in the OSR group, but this difference was not significant ($p = 0.064$). Table 5 shows that 4 of the 6 patients (66.7 %) with SCI in the TEVAR group had a delayed onset (from 3 h to 3 days after the end of surgery), as opposed to 3 of the 18 patients (16.7 %) in the OSR group, resulting in a significant difference ($p = 0.038$). The length of hospital stay was not significantly different in the two groups (77.1 ± 56.6 days

Table 2 Clinical and demographic characteristics of the study patients

	TEVAR ($n = 94$)	OSR ($n = 414$)	p value
Age (years)	74.6 ± 9.4	62.9 ± 14.0	<0.001
Sex (male)	75 (79.8 %)	296 (71.5 %)	0.102
DM	17 (18.1 %)	59 (14.3 %)	0.347
HT	84 (89.4 %)	343 (82.9 %)	0.347
HL	31 (33.0 %)	135 (32.6 %)	0.945
Smoker	60 (63.8 %)	247 (60.0 %)	0.456
COPD	6 (6.4 %)	26 (6.3 %)	0.97
Cr > 1.2 mg/dl	39 (41.5 %)	85 (20.5 %)	<0.011
CAD	42 (44.7 %)	128 (30.9 %)	0.011
CVD	13 (13.8 %)	63 (15.2 %)	0.733
Aneurysm diameter (mm)	57.6 ± 10.8	59.0 ± 8.8	0.23
Aortic dissection			
Acute	4 (4.3 %)	7 (1.7 %)	0.11
Chronic	4 (4.3 %)	171 (41.3 %)	<0.001
Rupture	7 (7.4 %)	23 (5.6 %)	0.435
Location of aneurysm			
TAA	87 (92.6 %)	222 (53.6 %)	<0.001
Crawford 1	4 (4.3 %)	25 (6.0 %)	0.501
2	0	37 (8.9 %)	0.003
3	2 (2.1 %)	93 (22.5 %)	<0.001
4	0	29 (7.0 %)	0.008
5	1 (1.1 %)	8 (1.9 %)	0.564

TEVAR thoracic endovascular aortic repair, *OSR* open surgical repair, *TAA* thoracic aortic aneurysm, *DM* diabetes mellitus, *HT* hypertension, *HL* hyperlipidemia, *COPD* chronic obstructive pulmonary disease, *Cr* creatinine, *CAD* coronary artery disease, *CVD* cerebrovascular disease

in the TEVAR group, 100.5 ± 112.3 days in the OSR group, $p = 0.634$). At discharge, 4 of the 6 (66.7 %) patients in the TEVAR group and 12 of the 18 patients in the OSR group scored either a 4 or 5 on the Tarlov scale ($p = 0.698$). Similarly, there were no statistically significant differences between the groups in sensory deficits ($p = 0.238$) or bladder dysfunction ($p = 0.314$) at discharge.

Discussion

In this study, the incidence of SCI in patients undergoing OSR and TEVAR was similar (4.3 and 6.4 %, respectively). The incidence of delayed paraplegia was significantly higher in the TEVAR group than in the OSR group. In regard to risk factors for SCI, the length of the stent graft was identified as a significant risk factor in the TEVAR group. There were no significant differences in neurologic outcomes of SCI at discharge, such as the Tarlov scale, sensory disturbances, and bladder dysfunction between patients undergoing TEVAR versus OSR.

The reported incidence of SCI or infarction as a consequence of OSR of TAAs and TAAAs remains in the range of 8–28 % [6, 7]. The incidence of SCI or infarction is lower after TEVAR, but SCI still occurs with an incidence of approximately 4–7 % [6, 8, 9]. The present study found no significant difference in the incidence of SCI between the two groups, but a previous meta-analysis and accumulated data indicate a statistically significant lower incidence of SCI in patients receiving TEVAR [2, 8, 9]. The incidence of SCI appears to be lower in the present study (4.3 %) as compared with that in previous studies (8.2 % in Cheng et al. [8] and 6.1 % in Glade et al. [9]). The reason for the lower incidence of SCI in the OSR group in our study is not clear, but the lower incidence in the OSR group may have contributed to the similar incidence of SCI found in the two groups in our study.

In regard to delayed paraplegia, it is unclear why the incidence of delayed paraplegia was higher in the TEVAR group in our study. Reduced flow to the collateral network perfusing the spinal cord [10] may gradually lead to delayed paraplegia. Carroccio et al. [11] have suggested that important intercostal arteries maintain cross-collateral flow and endoleak would be evident, but subsequent delayed intercostal artery thrombosis with endoleak resolution presents with spinal cord compromise. Delayed intercostal artery thrombosis may contribute to delayed paraplegia.

Table 3 Univariate and multivariate analyses of risk factors for spinal cord ischemia (SCI) in the TEVAR group

	SCI ($n = 6$)	No SCI ($n = 88$)	Univariate	Multivariate
Age (years)	76.7 ± 9.48	74.5 ± 9.38	0.294	
Sex (male)	3 (50 %)	72 (81.8 %)	0.095	0.102
DM	2(33.3 %)	15 (17.0 %)	0.296	
HT	5(83.3 %)	79 (89.8 %)	0.501	
HL	1 (16.7 %)	30 (34.1 %)	0.66	
Smoker	5 (83.3 %)	55 (62.5 %)	0.413	
COPD	0 (0 %)	6 (6.8 %)	0.509	
Cr > 1.2 mg/dl	4 (66.7 %)	35 (39.8 %)	0.229	
CAD	1 (16.7 %)	41 (46.6 %)	0.22	
CVD	1 (16.7 %)	12 (13.6 %)	0.835	
LSCA closure	0 (0 %)	7 (8.0 %)	0.473	
Length of aortic	296.6 ± 28.0	184.0 ± 64.2	<0.001	0.026
Coverage (mm)				
Aneurysm diameter (mm)	58.3 ± 5.12	57.3 ± 11.3	0.869	
Acute dissection	0 (0 %)	4 (4.5 %)	0.594	
Chronic dissection				
Rupture	1 (16.7 %)	6 (6.8 %)	0.39	
Emergency surgery	1 (16.7 %)	11 (12.5 %)	0.57	
History of aortic surgery				
Root to ascending	1 (16.7 %)	7 (8.0 %)	0.423	
Arch	5 (83.3 %)	33 (37.5 %)	0.038	0.213
Descending	1 (16.7 %)	8 (9.1 %)	0.463	
Thoracoabdominal	0 (0 %)	6 (6.8 %)	0.509	
AAA	1 (16.7 %)	36 (40.9 %)	0.398	

TEVAR thoracic endovascular aortic repair, *DM* diabetes mellitus, *HT* hypertension, *HL* hyperlipidemia, *COPD* chronic obstructive pulmonary disease, *Cr* creatinine, *CAD* coronary artery disease, *CVD* cerebrovascular disease, *LSCA* left subclavian artery, *AAA* abdominal aortic aneurysm

Multivariate logistic regression analysis in our study revealed that the length of aortic coverage had a significant association with SCI after TEVAR. Our result was compatible with previous studies [5, 12]. With longer stent graft coverage, more intercostal arteries are sacrificed, resulting in damage to the collateral network and the facilitation of SCI. Spinal cord vascularization depends on many interchangeable collateral arteries that supply the anterior spinal cord artery, rather than a single dominant Adamkiewicz artery (AKA) [10].

In the present study, 66.7 % of the patients with SCI from both groups recovered to a point where they could walk with support (Tarlov scale 4). There were no significant differences in recovery from SCI between the OSR and TEVAR groups. The rate of recovery from SCI in previous studies varied from 25 to 42.9 %, probably due to differences in the rehabilitative environment [13, 14]. Scivoletto and Di Donna [15] suggested that patients with both motor and sensory deficits after SCI showed poor prognosis in regard to walking. We found no significant differences in the incidence of sensory deficits or bladder dysfunction between our OSR and TEVAR groups. Taken together, there were no differences in recovery from SCI between the OSR and TEVAR groups.

The present study has several limitations. First, this study was not a randomized control study. As a result, there were some demographic differences between the OSR and TEVAR groups. The greater number of patients in the OSR group than in the TEVAR group may have led to statistical bias. In Japan, TEVAR is usually performed in patients with any of the following: comorbidities, advanced age, renal dysfunction, or low cardiac function. This selection bias may have affected recovery from paraplegia. Also, there were fewer patients with SCI in the TEVAR group than in the OSR group; thus, it was not possible to detect a difference between the two groups.

In conclusion, there were no significant differences in the incidence of SCI between the OSR and TEVAR groups, but delayed SCI occurred more frequently in the TEVAR group. The length of aorta covered by the stent graft was a significant factor associated with SCI in the TEVAR group. The rate of recovery from SCI to the point of assisted ambulation was similar in the OSR and TEVAR groups, and the incidence of sensory disturbances and bladder dysfunction were also similar in both groups.

Table 4 Univariate and multivariate analyses of risk factors for spinal cord ischemia in the OSR group

	SCI ($n = 18$)	No SCI ($n = 395$)	p value	
			Univariate	Multivariate
Age (years)	69.5 ± 8.2	62.5 ± 14.1	0.983	
Sex (male)	16 (89.5 %)	280 (70 %)	0.113	0.274
DM	4 (22.2 %)	55 (13 %)	0.288	
HT	15 (83.3 %)	328 (82.8 %)	0.95	
HL	6 (33.3 %)	129 (32.6 %)	0.976	
Smoker	13 (72.2 %)	234 (59.1 %)	0.335	
COPD	3 (16.7 %)	23 (5.8 %)	0.099	0.308
Cr > 1.2 mg/dl	6 (33.3 %)	79 (19.9 %)	0.260	
CAD	10 (55.6 %)	118 (29.8 %)	0.035	0.369
CVD	7 (38.9 %)	56 (14.1 %)	0.012	0.146
Aneurysm diameter	63.2 ± 9.4	59.2 ± 8.5	0.061	0.18
Acute dissection	1 (5.6 %)	6 (1.5 %)	0.716	
Chronic dissection	9 (50.0 %)	162 (40.9 %)	0.871	
Rupture	1 (5.6 %)	22 (5.6 %)	0.601	
TAA	10	212	0.866	
Crawford 1	0	25	0.271	
2	2	35	0.741	
3	4	89	0.98	
4	2	27	0.478	
5	0	8	0.523	
Emergency surgery	2 (11.5 %)	25 (6.3 %)	0.331	
History of aortic surgery				
Root to ascending	2 (11.1 %)	48 (12.1 %)	0.898	
Arch	2 (11.1 %)	72 (18.2 %)	0.752	
Descending	1 (5.6 %)	40 (10.1 %)	0.528	
Thoracoabdominal	0 (0 %)	12 (3 %)	0.454	
AAA	4 (22.2 %)	62 (15.7 %)	0.506	

DM diabetes mellitus, *HT* hypertension, *HL* hyperlipidemia, *COPD* chronic obstructive pulmonary disease, *Cr* creatinine, *CAD* coronary artery disease, *CVD* cerebrovascular disease, *AAA* abdominal aortic aneurysm

Table 5 Comparison of details in patients with spinal cord ischemia between the two groups

	TEVAR ($n = 6$)	OSR ($n = 18$)	p value
MEP change	2 (33.3 %)	12 out of 15 (80 %)	0.064
Delayed paraplegia	4 (66.7 %)	3 (16.7 %)	0.038
Tarlov score ≥4	4 (66.7 %)	12 (66.7 %)	0.698
Sensory deficit	1 (16.7 %)	8 (44.4 %)	0.238
Bladder dysfunction	1 (16.7 %)	7 (38.9 %)	0.314

MEP motor evoked potential, *OSR* open surgery repair, *TEVAR* thoracic endovascular aortic repair

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