Eur J Vasc Endovasc Surg (2010) **39**, 179–186

Spinal Cord Injury is Not Negligible after TEVAR for Lower Descending Aorta☆

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Submitted 31 August 2009; accepted 15 November 2009
Available online 3 December 2009

KEYWORDS
Thoracic aortic aneurysm;
Endovascular repair;
Stent graft;
Spinal cord injury;
Paraplegia

Abstract *Objectives:* To clarify the incidence of spinal cord injury (SCI) after thoracic endovascular aneurysm repair (TEVAR), we investigate the intercostal/lumbar arteries that supply the Adamkiewicz artery (ICA-AKA).
Patients: Among 81 patients subjected to TEVAR, we retrospectively reviewed the clinical records of 50 patients (range: 57–86 (median age: 77) years, 41 males) who underwent TEVAR for part of or the whole distal descending aorta (T7 to L2) after identification of ICA-AKA by magnetic resonance angiography (MRA) or computed tomography angiography (CTA).
Results: The 50 patients were classified into group A: 17 patients whose patent ICA-AKA was not covered, group B: 24 patients whose ICA-AKA was covered and group C: nine patients in whom no patent ICA-AKA was identified. Only three patients in group B suffered paraplegia and of them two recovered full ambulation. The estimated incidence of permanent and transient paraplegia was 3.7% in all TEVAR patients, 6.0% when part of or the entire distal aorta was covered and 12.5% when the patent ICA-AKA was covered. The length of aortic coverage in patients with paraplegia was >300 mm.
Conclusions: Paraplegia after TEVAR occurred in one of eight patients in whom the stent graft covered ICA-AKA. Long coverage of the aorta including the ICA-AKA was critical. To prevent this serious complication, identification of the ICA-AKA is crucial.

☆ This paper was presented at the XXIII Annual Meeting 3–6 September, 2009, European Society for Vascular Surgery, Oslo, Norway.

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1078-5884/$36
doi:10.1016/j.ejvs.2009.11.014

The incidence of spinal cord injury (SCI) after thoracic endovascular aneurysm repair (TEVAR) has been reported to vary according to the demographics of the patients.[1–20] Whether the integrity of the Adamkiewicz artery (AKA) is essential for spinal cord function is still to be investigated.[21] However, after reattachment of the intercostal/lumbar arteries, which supply AKA (ICA-AKA), or of the adjacent intercostal/lumbar arteries during thoracoabdominal aortic replacement, motor-evoked potentials (MEPs) recover.[22] TEVAR has been reported to reduce SCI.[12] In principle, the longer the length of the aorta including both landing zones that is covered by TEVAR, the larger the number of ICAs that will be sacrificed and whose revascularisation will be impossible.[23]

To clarify the incidence and cause of SCI after TEVAR, we have investigated the patency of ICA-AKA in relation to other factors which may cause SCI.

Materials/Methods

Patient demographics

In the past 27 months, of 81 patients, we performed TEVAR with Gore TAG (W. L. Gore & Associates, Flagstaff, AZ, USA) in 47 patients, Talent thoracic stent graft (Medtronic, Inc., Santa Rosa, CA, USA) in five, both TAG and Talent in one and Matsui-Kitamura (MK) stent graft in 28 patients.[24] In this study, we included 50 patients who underwent TEVAR for part of or the whole distal descending aorta after ICA-AKA was identified by magnetic resonance angiography (MRA) or computed tomography angiography (CTA). The distal descending aorta was defined as the segment between T7 and L2.[25] Fifteen patients who underwent TEVAR above T6 and 16 patients who had not undergone MRA or CTA to identify ICA-AKA were not included in this investigation.

In general, the patients were senescent, debilitated and presented co-morbidities. (Table 1) Thirty-seven patients were ≥75 years old and the median age was 77. Thirty-seven patients were in ASA class 3 or 4, and 32 patients had a history of aortic surgery (48 surgeries in total).

Of the 18 patients who had undergone AAA repair, TEVAR had been indicated more than 1 year later in 11 patients, scheduled within 3 months in five and performed simultaneously in two. Emergency TEVAR was performed in three for haemoptysis, acute aneurysm dissection and persistent back pain. They were haemodynamically stable and could undergo CTA for ICA-AKA.

In all patients, another CTA was carried out to precisely measure the aneurysm and access. CTA also revealed the patency of the left subclavian (LSCA) and bilateral internal iliac arteries (IIA). Occlusion of left IIA (LIIA) was confirmed in three patients but LSCA and right IIA (RIIA) were patent in all the patients regardless of whether total arch replacement (TAR) or AAA repair was performed.

Identification of ICA-AKA

ICA-AKA was identified by MRA in 39 and by CTA in 11 patients.

The details of contrast MRA were previously reported by Yamada et al.[26] For the CTA, an Aquilion 16 multi-detector row CT scanner (Toshiba, Tokyo, Japan) was used. To detect AKA, the reconstruction field of view was set to the area around the aorta and spine. The images were processed in a workstation (Ziostation; Amin, Tokyo, Japan). Volume-rendered images of the entire aorta were routinely generated. Multiplanar reformation (MPR) images, including oblique coronal images with craniocaudal angulations and curved planar reformation images, were reconstructed to investigate the side and level of the origin of AKA.

Diagnostic criteria for the anterior spinal artery and AKA were as previously reported.[26] We preferred MRA as CTA is disadvantageous due to the influence of the spine and lack of accurate differentiation of the AKA from the anterior radicular vein.[22] However, the selection of MRA or CTA

Table 1 Patient demographics.

Number of Patients	50			
Age	57—86 [median 77] year-old			
Gender	41 male			
ASA class	Class 2: 13, Class 3: 19, Class 4: 18			
History of aortic surgery	Root to Ascending	3		
	Arch	21	Total arch replacement	20
			TEVAR after debranch	1
	Descending		Replacement	3
			TEVAR	1
	Thoraco-abdominal	2		
	AAA	18	Replacement	17
			EVAR	1
Aortic pathology	Degenerative aneurysm	39		
	Chronic dissection	3		
	Acute dissection on aneurysm	2		
	Penetrating atherosclerotic ulcer	3		
	Anastomotic false aneurysm	3		

Table 2 Distribution of ICA-AKA.

	Right	Left	(Occlusion at origin)
Th7	0	1	
Th8	1	6	(2)
Th9	0	18	(1)
Th10	1	10	
Th11	0	7	(1)
Th12	2	4	(2)
L1	0	1	
L2	0	0	
Total	4	47	(6)

ICA-AKA: intercostal/lumbar arteries which supplies Adamkiewicz artery.

depended on the availability of the equipment. CTA was used in all three emergency cases.

When AKA was not identified by MRA, it was diagnosed as 'absent' ($n = 3$). In 47 patients, 51 ICA-AKAs were identified (Table 2). In four patients, there were double ICA-AKAs. Occlusion of ICA-AKA at its origin was diagnosed in six patients, in all of them on the left side. When the ICA-AKA was occluded, blood supply from adjacent intercostal or lumbar arteries was suspected to be significant. However, we were unable to distinguish the critical collateral flow to AKA.

TEVAR

To create a landing zone, a carotid—subclavian bypass was performed in two and visceral vessel bypass was performed in one. In nine patients who had extensive/multiple aneurysm(s) from the aortic arch to the descending aorta, TAR was performed using elephant trunk (ET) implantation. Regarding patients who had a history of aortic surgery, an artificial graft was used to create a proximal landing zone in 19 and a distal landing zone in three.

In all patients, TEVAR was carried out under general anaesthesia. The access route for TEVAR was a native artery in 35, an iliac conduit in 13 and a graft limb or a side branch of AAA graft in two patients.

MEP monitoring and cerebrospinal fluid drainage

In all patients trans-cranial MEPs were monitored during TEVAR and a cerebrospinal fluid drainage (CSFD) tube was placed before TEVAR in 31 patients.

Immediately after the stent graft was placed, the mean blood pressure was raised above 80 mmHg and MEP was monitored every 5 min. When the amplitude of MEPs decreased under general anaesthesia, or when symptoms and signs of SCI were noted during the postoperative period, CSFD (<15 cmH_2O) was started with the infusion of methylprednisolone (30 mg kg^{-1} bolus and 5.4 mg kg^{-1} h^{-1} for 23 h followed by 2.7 mg kg^{-1} h^{-1} for 2 days) and naloxone (1200 μg day^{-1}). Intensive spinal care with CSFD, methylprednisolone and naloxone was continued for 72 h if the symptom did not resolve or was discontinued 24 h after full recovery.

CSFD was started only after paraplegia or a decrease of less than 25% of the amplitude of MEPs was noted. CSFD was not indicated as a prophylactic measure after TEVAR.

Measurement of the aortic length

The length of 'proximal uncovered aorta' (from LSCA to stent graft), 'aortic coverage' by stent graft and 'distal uncovered aorta' (from stent graft to coeliac axis (CA)) was measured on CTA using curved planar reformation images processed in a workstation (GE Advantage workstation 4.3).

After TAR with a multibranch graft, the length of aortic coverage was measured from the distal anastomosis. This site coincided with the origin of ET and was several centimetres distal to the branch graft of LSCA. When ET was installed, the proximal edge of the stent graft was positioned inside the multibranch graft and not only inside ET. After replacement of the descending or the thoracoabdominal aorta, the position of LSCA and/or CA served as the point of reference for the measurement.

Statistical analysis

Values are the mean ± SD. Data were analysed using the chi-square test for categorical variables, and continuous variables were examined using analysis of variance (ANOVA). The level of statistical significance was set at $p < 0.05$.

Results

Mortality and morbidity

Initial success of TEVAR was achieved in all patients except for two patients with Type I endoleaks detected by CTA who were successfully treated by a repeat TEVAR. No operative (30 days) death was encountered. Injury and occlusion of access arteries occurred in one. Two patients were complicated with cerebral embolism due to the guidewire pull-through technique and atrial fibrillation.

The following three patients were complicated with paraplegia: Patient 1 was a 59-year-old man with a history of closure of ventricular septal defect, aortic valve replacement and repair of a Valsalva sinus aneurysm. He also suffered from liver cirrhosis. He developed aneurysmal dilatation of the whole thoracic aorta and underwent TAR with ET installation as the first-stage repair. MRA revealed the AKA arose from the left Th9-ICA. TEVAR with Gore TAG was performed 5 weeks later from ET (Z3) to T11. The iliac conduit was connected to the right common iliac artery but the haemostasis was time consuming because of obvious coagulopathy due to liver cirrhosis. Paraplegia was confirmed 24 h after TEVAR after the patient suffered much pain. Despite treatment for SCI, the patient could not ambulate. Retroperitoneal haematoma had to be removed twice. He eventually died from methicillin-resistant *Staphylococcus aureus* (MRSA) mediastinitis 4 months after TEVAR. The length of aortic coverage from the origin of ET to the distal flair was 325 mm.

Patient 2 was an 81-year-old man with ascending, arch and descending aorta aneurysms. MRA revealed the AKA branching from the left Th9-ICA. Four weeks after TAR with ET installation, TEVAR with TAG was performed from ET (Z3) to T12 (Fig. 1). The iliac conduit was required and

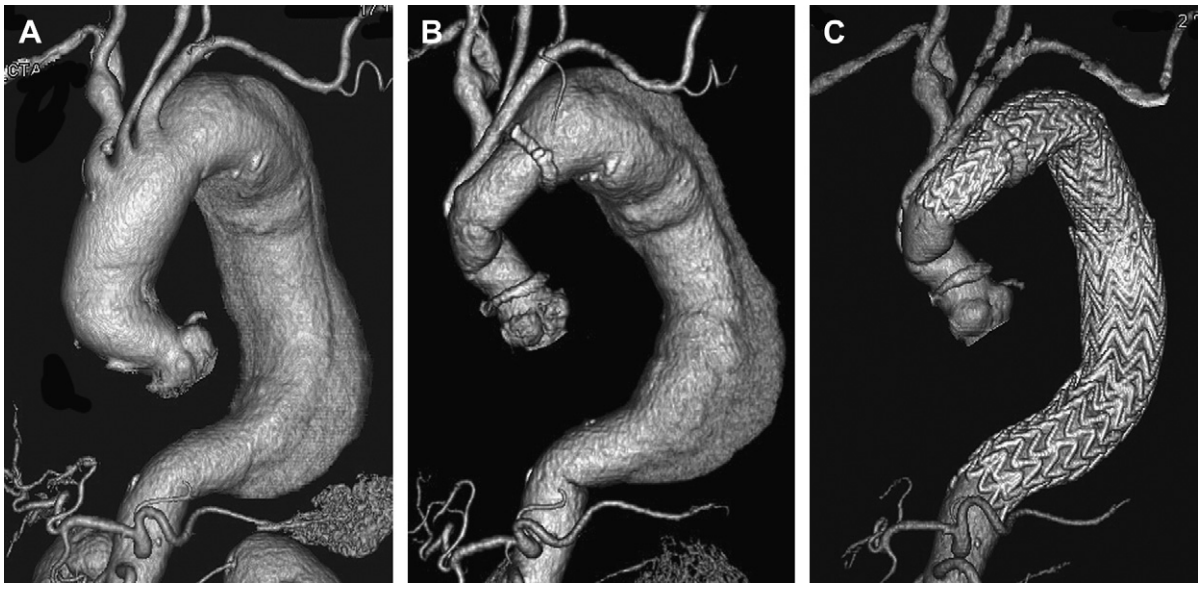

Figure 1 Sequence of CTA in Patient 2. Panel A: Preoperative, Panel B: After total arch replacement, Panel C: After TEVAR.

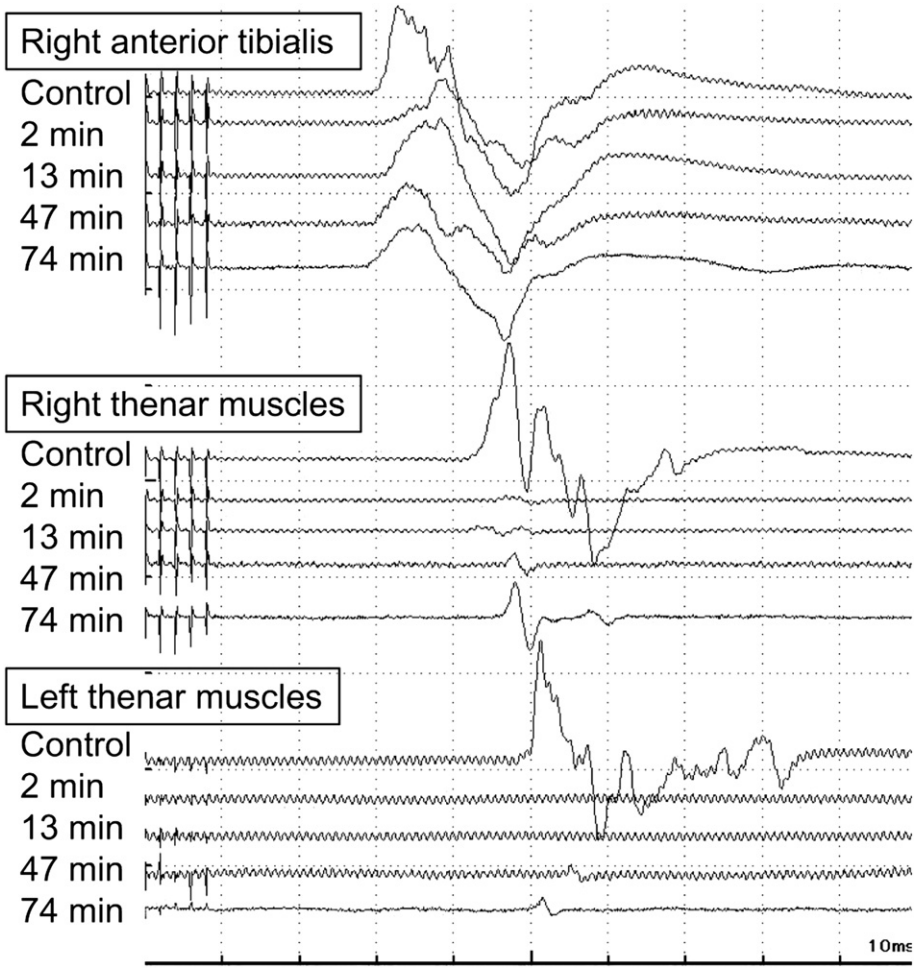

Figure 2 Sequence of MEPs in Patient 3. MEPs of the right anterior tibialis and both thenar muscles before TEVAR over Th9-ICA (control) and 2, 13, 47, and 74 minutes after TEVAR.

haemostasis took a long time due to co-existing consumption coagulopathy caused by aortic lesions. Six hours after TEVAR, the patient suddenly complained of back pain and paraplegia was confirmed. One hour after intensive spinal treatment, he could move his legs and on the next morning he could walk. The retroperitoneal, femoral and brachial haematomas were removed twice. The length of aortic coverage was 302 mm.

Patient 3 was a 78-year-old woman who had undergone TEVAR (Z3 to T7) for a proximal descending aortic aneurysm 6 months earlier. MRA revealed the ICA-AKA branching from the left Th9-ICA. Due to the rapid growth of the distal descending aortic aneurysm, TEVAR was performed again from the previous stent graft to L1. The CA was closed to create a distal landing zone. Immediately after the deployment over the Th9-ICA, the MEPs of both thenar muscles diminished and the amplitude of the MEPs of the right anterior tibialis decreased about 50% from the control amplitude (Fig. 2). Despite treatment for SCI, ankle dorsiflexion was slight when she awoke from anaesthesia. Intensive spinal care was continued and she gradually gained muscle strength within 3 h after TEVAR. On the following morning she could ambulate. The length of aortic coverage after the first TEVAR was 157 mm and was extended to 308 mm by the second TEVAR.

Incidence of paraplegia

The 50 patients were classified into group A: 17 patients whose patent ICA-AKA was not covered by TEVAR, group B: 24 patients whose ICA-AKA was covered by TEVAR and group C: nine patients in whom no patent ICA-AKA was identified. Group C included six patients whose ICA-AKA occluded at its origin and three patients whose ICA-AKA was absent.

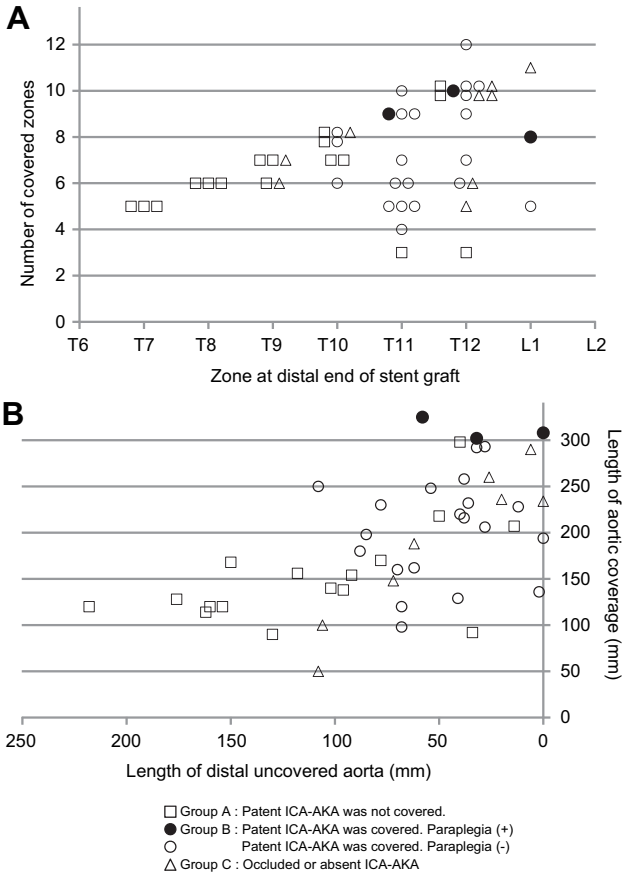

Figure 3 Distribution of patients with paraplegia in accordance with covered aorta and distal uncovered aorta expressed as number of aortic zones (Panel A) and measured length (Panel B).

Table 3 Comparison of patients with and without paraplegia.

	Paraplegia $N = 3$	No paraplegia $N = 47$	p
Age (year-old)	72.7 ± 11.9	76.1 ± 6.1	.3736
Male gender	2 (67%)	39 (83%)	.5094
ASA classification	3.7 ± 0.6	3.1 ± 0.8	.2026
Renal dysfunction	1 (33%)	18 (38%)	.3362
History of aortic repair (descending, thoracoabdominal, abdominal)	1 (33%)	19 (40%)	.8060
LSCA patency	3 (100%)	47 (100%)	—
RIIA patency	3 (100%)	47 (100%)	—
LIIA patency	3	44	.5359
Op time (minutes)	252 ± 117	141 ± 76	.0200
Blood loss (ml)	557 ± 274	363 ± 423	.4482
Use of an iliac conduit	2 (67%)	15 (32%)	.2335
Zones of aortic coverage	9 ± 1	7.2 ± 2.2	.1691
Proximal uncovered aorta (mm)	0	36 ± 49	.2191
Aortic coverage (mm)	312 ± 12	179 ± 64	.0009
Distal uncovered aorta (mm)	30 ± 29	72 ± 52	.1839
Hypotension	2 (67%)	3 (6%)	.0116

LSCA: Left subclavian artery, R(L)IIA: Right internal iliac artery.

Table 4 Reported incidence of spinal cord injury (SCI).

	Patients	Location	N	SCI	
Criado, 2002	TEVAR	Arch-descending	47	0	(0.0%)
Bergeron, 2003	TEVAR	Descending	38	0	(0.0%)
Czerny, 2004	TEVAR	Descending	54	0	(0.0%)
Orend, 2003	TEVAR	Various	74	2	(2.7%)
Mitchell, 1999	TEVAR	n/d	103	3	(2.9%)
Makaroun, 2005	TAG phase II	Various	142	4	(3.0%)
Ellozy, 2003	TEVAR	Descending	84	3	(3.6%)
Morales, 2007	TEVAR	n/d	186	7	(3.8%)
Bell, 2003	TEVAR	Various	67	3	(4.0%)
Greenberg, 2008	TEVAR	TAAA	352	15	(4.3%)
Gravereaux, 2001	TEVAR	Descending	53	3	(5.7%)
Greenberg, 2005	TXI & TXII	Various	100	6	(6.0%)
Sandroussi, 2007	TEVAR	n/d	65	4	(6.2%)
Cheung, 2005	TEVAR	Various	75	5	(6.5%)
Amabile, 2008	TEVAR	Descending	67	5	(7.5%)
Feezor, 2008	TEVAR	n/d	326	33	(10.0%)

The three patients who developed paraplegia were in group B, that is, ICA-AKA was covered by TEVAR. The estimated incidence of permanent and transient paraplegia was 3.7% in all patients subjected to TEVAR (81 patients), 4.5% in patients in whom part or the entire distal aorta was covered, regardless of ICA-AKA identification by MRA (66 patients) and 6.0% in those whose ICA-AKA was identified (50 patients, groups A, B and C). The incidence increased to 12.5% only when the patent ICA-AKA was covered by TEVAR (24 patients, group B).

Comparison of patients with and without paraplegia after TEVAR showed that the operation time and the length of aortic coverage were significantly longer in those with paraplegia (Table 3). Episodes of hypotension below 80 mmHg for more than 10 min during and after surgery were more frequent in patients with paraplegia. No difference was found in the patency rate of LSCA[27] and IIA. Other risk factors previously reported[28] such as the abdominal aortic surgery and the renal dysfunction showed no difference.

Fig. 3 shows the occurrence of paraplegia in relation to aortic coverage and distal aortic uncoverage length. When these were divided into zones, the stent grafts in the three patients with paraplegia were placed at T11 or distal to it and covered more than eight zones. Fourteen patients without paraplegia had the same range of intervened zones (Fig. 3A). In the three patients with paraplegia, the length of aortic coverage was more than 300 mm. The length of distal uncovered aorta was within 60 mm. Four other patients whose length of aortic coverage was between 270 mm and 300 mm and the length of distal uncovered aorta was less than 60 mm did not experience paraplegia (Fig. 3B).

Discussion

We have reported the low risk of paraplegia for patients subjected to descending and thoraco-abdominal aorta open repair by combined use of AKA identification by MRA and MEP measurement.[22] The risk of paraplegia has been considered to be lower after TEVAR than after open repair, but the incidence of SCI in the previous reports varied (Table 4). This variation might be due to differences in case mix as the area subjected to TEVAR was not the same and was not specified in some of the reports.[23]

The theoretical advantages of TEVAR concerning protection of the spinal cord are the maintenance of distal perfusion, stable haemodynamics and no reperfusion of the spinal cord.[12] However, additional ICAs are sacrificed for the landing zones and revascularisation of the ICAs is impossible. Paraplegia occurs after TEVAR when the arteries that supply the spinal cord are sacrificed, as well as after a period of hypotension or as a result of emboli from aortic atheromatous lesions.[29]

We encountered paraplegia in three patients whose patent ICA-AKA was covered and the length of aortic coverage was more than 300 mm. The rate of permanent and transient paraplegia was 12.5% (1/8), when the patent ICA-AKA was covered by TEVAR. This result was in agreement with that of a previous study showing that SCI occurred in 9.1% of the patients with occlusion of the ICA-AKA.[13] The authors did not encounter paraplegia in patients whose ICA-AKA was patent. This fact is also relevant to our result as none of our patients, whose ICA-AKA was already occluded at its origin before TEVAR or was absent, experienced paraplegia.

The spinal cord blood supply depends on many interchangeable collateral arteries that supply the anterior spinal cord artery, rather than a single dominant AKA.[21] However, the importance of the patency of the ICA-AKA during TEVAR[13] and the restoration of blood flow in the spinal cord after revascularisation of ICA-AKA at the aortic replacement have been reported.[22] Patency of ICA-AKA is sufficient to prevent paraplegia and occlusion of the patent ICA-AKA is critical. To preserve the patency of ICA-AKA, this should be identified preoperatively to allow the creation of an adequate landing zone.[12]

The rate of paraplegia, 12.5%, means that 87.5% (7/8) patients did not develop paraplegia after the coverage of patent ICA-AKA. Between three patients who suffered paraplegia and 21 patients who did not in group B, the threshold of the length of aortic coverage was 300 mm. The length of aortic coverage has been described as a risk for SCI in previous reports.[13,30] Amabile et al. reported that

205 mm was the critical length of aortic coverage for SCI.[18] Feezor et al. described that both the extent (>200 mm) and distal location of aortic coverage (20 mm from CA) were associated with an increased risk for SCI.[16]

We tried to locate the critical segment for paraplegia by dividing the aorta into zones but the length measured by CTA demonstrated the critical length of aortic coverage more clearly. This particular threshold, 300 mm, might vary in the future as experience accumulates and the index is modified, for instance, according to height. Nevertheless, it can be emphasised that long aortic coverage is another important risk factor for paraplegia. Long coverage of the aorta including the patent ICA-AKA is critical.

We found intra-operative coagulopathy related to prolonged operation time and postoperative retroperitoneal bleeding in patient 1 and patient 2. Hypotension associated with retroperitoneal bleeding contributes to SCI.[6] Consumption coagulopathy is another risk which is heightened by long coverage of the aorta.

Paraplegia occurred when the stent graft covered the zones at T11 or was placed less than 60 mm from CA. It can be concluded that the zones above T10 or a distance of more than 60 mm from CA are safe. However, the length of distal uncovered aorta would only express the probability of the occlusion of the ICA-AKA according to its distribution. The length of distal uncovered aorta might be less significant than the closure of the ICA-AKA or the length of aortic coverage.

Similarly, high percentages of paraplegia, 12.5% and 14.3%, after TEVAR were reported in patients with prior AAA repair.[12,20] In our series, a history of abdominal, thoraco-abdominal, or descending aneurysm repair was not a significant risk for paraplegia. However, AAA repair sacrifices several pairs of lumbar arteries that significantly contribute to spinal cord perfusion and/or IIA, which are the possible sources of direct or collateral blood flow to spinal arteries. Indeed, previous AAA repair was described as a risk factor in various other reports.[2,6,12,27,31]

Limitations of this study include the retrospective review of prospectively collected data, the retrospective measurement of aortic length and the small number of patients. Further accumulation of patients treated by TEVAR after identification of ICA-AKA is crucial for more precise diagnosis of the risk for paraplegia after TEVAR.

Conclusions

Paraplegia after TEVAR occurred in 1 of 8 (12.5%) patients in whom the stent graft covered the distal descending aorta below Th7. Long (>300 mm) coverage of the aorta including the ICA-AKA is a critical risk factor for SCI and paraplegia. To prevent this serious complication, it is imperative to identify the ICA-AKA before performing TEVAR.

Conflict of Interest/Funding

None.

Acknowledgement

We express our sincere appreciation to Dr. Naoaki Yamada and Dr. Masahiro Higashi (from the Department of Radiology, National Cardiovascular Center), who contributed to the clear visualisation of the Adamkiewicz artery by MRA and CTA.

References

1 Mitchell RS, Miller DC, Dake MD, Semba CP, Moore KA, Sakai T. Thoracic aortic aneurysm repair with an endovascular stent graft: the ''first generation'' *Ann Thorac Surg* 1999;**67**:1971–4.
2 Gravereaux EC, Faries PL, Burks JA, Latessa V, Spielvogel D, Hollier LH, et al. Risk of spinal cord ischemia after endograft repair of thoracic aortic aneurysms. *J Vasc Surg* 2001;**34**:997–1003.
3 Criado FJ, Clark NS, Barnatan MF. Stent graft repair in the aortic arch and descending thoracic aorta: a 4-year experience. *J Vasc Surg* 2002;**36**:1121–8.
4 Ellozy SH, Carroccio A, Minor M, Jacobs T, Chae K, Cha A, et al. Challenges of endovascular tube graft repair of thoracic aortic aneurysm: midterm follow-up and lessons learned. *J Vasc Surg* 2003;**38**:676–83.
5 Orend KH, Scharrer-Pamler R, Kapfer X, Kotsis T, Görich J, Sunder-Plassmann L. Endovascular treatment in diseases of the descending thoracic aorta: 6-year results of a single center. *J Vasc Surg* 2003;**37**:91–9.
6 Cheung AT, Pochettino A, McGarvey ML, Appoo JJ, Fairman RM, Carpenter JP, et al. Strategies to manage paraplegia risk after endovascular stent repair of descending thoracic aortic aneurysms. *Ann Thorac Surg* 2005;**80**:1280–8.
7 Bell RE, Taylor PR, Aukett M, Sabharwal T, Reidy JF. Mid-term results for second-generation thoracic stent grafts. *Br J Surg* 2003;**90**:811–7.
8 Bergeron P, De Chaumaray T, Gay J, Douillez V. Endovascular treatment of thoracic aortic aneurysms. *J Cardiovasc Surg (Torino)* 2003;**44**:349–61.
9 Czerny M, Cejna M, Hutschala D, Fleck T, Holzenbein T, Schoder M, et al. Stent-graft placement in atherosclerotic descending thoracic aortic aneurysms: midterm results. *J Endovasc Ther* 2004;**11**:26–32.
10 Makaroun MS, Dillavou ED, Kee ST, Sicard G, Chaikof E, Bavaria J, et al. Endovascular treatment of thoracic aortic aneurysms: results of the phase II multicenter trial of the GORE TAG thoracic endoprosthesis. *J Vasc Surg* 2005;**41**:1–9.
11 Greenberg RK, O'Neill S, Walker E, Haddad F, Lyden SP, Svensson LG, et al. Endovascular repair of thoracic aortic lesions with the Zenith TX1 and TX2 thoracic grafts: intermediate-term results. *J Vasc Surg* 2005;**41**:589–96.
12 Baril DT, Carroccio A, Ellozy SH, Palchik E, Addis MD, Jacobs TS, et al. Endovascular thoracic aortic repair and previous or concomitant abdominal aortic repair: is the increased risk of spinal cord ischemia real? *Ann Vasc Surg* 2006;**20**:188–94.
13 Kawaharada N, Morishita K, Kurimoto Y, Hyodoh H, Ito T, Harada R, et al. Spinal cord ischemia after elective endovascular stent-graft repair of the thoracic aorta. *Eur J Cardiothorac Surg* 2007;**31**:998–1003.
14 Morales JP, Taylor PR, Bell RE, Chan YC, Sabharwal T, Carrell TW, et al. Neurological complications following endoluminal repair of thoracic aortic disease. *Cardiovasc Intervent Radiol* 2007;**30**:833–9.
15 Sandroussi C, Waltham M, Hughes CF, May J, Harris JP, Stephen MS, et al. Endovascular grafting of the thoracic aorta, an evolving therapy: ten-year experience in a single centre. *ANZ J Surg* 2007;**77**:974–80.
16 Feezor RJ, Martin TD, Hess Jr PJ, Daniels MJ, Beaver TM, Klodell CT, et al. Extent of aortic coverage and incidence of spinal cord ischemia after thoracic endovascular aneurysm repair. *Ann Thorac Surg* 2008;**86**:1809–14.
17 Greenberg RK, Lu Q, Roselli EE, Svensson LG, Moon MC, Hernandez AV, et al. Contemporary analysis of descending

thoracic and thoracoabdominal aneurysm repair: a comparison of endovascular and open techniques. *Circulation* 2008;**118**: 808—17.
18 Amabile P, Grisoli D, Giorgi R, Bartoli JM, Piquet P. Incidence and determinants of spinal cord ischaemia in stent-graft repair of the thoracic aorta. *Eur J Vasc Endovasc Surg* 2008;**35**:455—61.
19 Hnath JC, Mehta M, Taggert JB, Sternbach Y, Roddy SP, Kreienberg PB, et al. Strategies to improve spinal cord ischemia in endovascular thoracic aortic repair: outcomes of a prospective cerebrospinal fluid drainage protocol. *J Vasc Surg* 2008;**48**:836—40.
20 Schlösser FJ, Verhagen HJ, Lin PH, Verhoeven EL, van Herwaarden JA, Moll FL, et al. TEVAR following prior abdominal aortic aneurysm surgery: increased risk of neurological deficit. *J Vasc Surg* 2009;**49**:308—14.
21 Griepp RB, Ergin MA, Galla JD, Lansman S, Khan N, Quintana C, et al. Looking for the artery of Adamkiewicz: a quest to minimize paraplegia after operations for aneurysms of the descending thoracic and thoracoabdominal aorta. *J Thorac Cardiovasc Surg* 1996;**112**:1202—13.
22 Ogino H, Sasaki H, Minatoya K, Matsuda H, Yamada N, Kitamura S. Combined use of adamkiewicz artery demonstration and motor-evoked potentials in descending and thoracoabdominal repair. *Ann Thorac Surg* 2006;**82**:592—6.
23 Bicknell CD, Riga CV, Wolfe JH. Prevention of paraplegia during thoracoabdominal aortic aneurysm repair. *Eur J Vasc Endovasc Surg* 2009;**37**:654—60.
24 Sanada J, Matsui O, Terayama N, Kobayashi S, Minami T, Kurozumi M, et al. Clinical application of a curved nitinol stent-graft for thoracic aortic aneurysms. *J Endovasc Ther* 2003;**10**: 20—8.
25 Ishimaru S. Endografting of the aortic arch. *J Endovasc Ther* 2004;**11**(Suppl 2):II62—II71.
26 Yamada N, Takamiya M, Kuribayashi S, Okita Y, Minatoya K, Tanaka R. MRA of the Adamkiewicz artery: a preoperative study for thoracic aortic aneurysm. *J Comput Assist Tomogr* 2000;**24**: 362—8.
27 Cooper DG, Walsh SR, Sadat U, Noorani A, Hayes PD, Boyle JR. Neurological complications after left subclavian artery coverage during thoracic endovascular aorticrepair: a systematic review and meta-analysis. *J Vasc Surg* 2009;**49**: 1594—601.
28 Buth J, Harris PL, Hobo R, van Eps R, Cuypers P, Duijm L, et al. Neurologic complications associated with endovascular repair of thoracic aortic pathology: incidence and risk factors. a study from the European Collaborators on Stent/Graft Techniques for Aortic Aneurysm Repair (EUROSTAR) registry. *J Vasc Surg* 2007; **46**:1103—10.
29 Carroccio A, Marin ML, Ellozy S, Hollier LH. Pathophysiology of paraplegia following endovascular thoracic aortic aneurysm repair. *J Card Surg* 2001;**11**:359—66.
30 Greenberg R, Resch T, Nyman U, Lindh M, Brunkwall J, Brunkwall P, et al. Endovascular repair of descending thoracic aortic aneurysms: an early experience with intermediate-term follow-up. *J Vasc Surg* 2000;**31**:147—56.
31 Mitchell RS, Miller DC, Dake MD. Stent-graft repair of thoracic aortic aneurysms. *Semin Vasc Surg* 1997;**10**:257—71.