Spinal cord ischemia after simultaneous and sequential treatment of multilevel aortic disease

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Objectives: The aim of the present study is to report a risk analysis for spinal cord injury in a recent cohort of patients with simultaneous and sequential treatment of multilevel aortic disease.

Methods: We performed a multicenter study with a retrospective data analysis. Simultaneous treatment refers to descending thoracic and infrarenal aortic lesions treated during the same operation, and sequential treatment refers to separate operations. All descending replacements were managed with endovascular repair.

Results: Of 4320 patients, multilevel aortic disease was detected in 77 (1.8%). Simultaneous repair was performed in 32 patients (41.5%), and a sequential repair was performed in 45 patients (58.4%). Postoperative spinal cord injury developed in 6 patients (7.8%). At multivariable analysis, the distance of the distal aortic neck from the celiac trunk was the only independent predictor of postoperative spinal cord injury (odds ratio, 0.75; 95% confidence interval, 0.56-0.99; $P = .046$); open surgical repair of the abdominal aortic disease was associated with a higher risk of spinal cord injury but did not reach statistical significance (odds ratio, 0.16; 95% confidence interval, 0.02-1.06; $P = .057$). Actuarial survival estimates at 1, 2, and 5 years after the procedure were $80\% \pm 5\%$, $68\% \pm 6\%$, and $63\% \pm 7\%$, respectively. Spinal cord injury did not impair survival ($P = .885$).

Conclusions: In our experience, the risk of spinal cord injury is still substantial at 8% in patients with multilevel aortic disease. The distance of the distal landing zone from the celiac trunk is a significant predictor of spinal cord ischemia. (J Thorac Cardiovasc Surg 2014;148:1435-42)

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Multilevel aortic disease (MAD) is an uncommon finding that still poses a treatment dilemma for cardiovascular surgeons.¹⁻³ Simultaneous treatment has been associated with severe perioperative complications, whereas sequential repair requires 2 major surgical interventions, and rupture of the residual lesion can occur while waiting for the second operation.^{1,4} Regardless of the strategy

adopted, these patients require extensive aortic treatment and are at high risk for spinal cord injury (SCI).

Up to now, definitive recommendations about the appropriate treatment are lacking. The rate of SCI after thoracic endovascular repair (TEVAR) has been reported as significantly higher with a history of abdominal aortic repair, but there are few data regarding the comparison of simultaneous versus sequential treatment of MAD.⁵⁻¹⁰

The aim of the present study is to report a risk analysis for SCI in a recent cohort of patients with simultaneous or sequential MAD treatment.

MATERIALS AND METHODS

Patient Population

Between October 2001 and March 2013, all patients undergoing simultaneous or sequential repair of separate descending thoracic and infrarenal abdominal aortic lesions were identified and included in the analysis. Patients with ascending aortic disease, thoracoabdominal extent, or mega-aortic syndrome were excluded. This is a retrospective multicenter study. All clinical and procedural data were prospectively collected and recorded in a dedicated computerized database that remained consistent over the study period. The authors had full access to and take full responsibility for the integrity of the data. The study protocol was in compliance with the local institutional review board and received full approval.

Patient Management

All patients underwent preoperative total aortic computed tomography angiography. The risk of SCI was debated case by case, and the type of

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Abbreviations and Acronyms

ASIA	= American Spinal Injury Association
CI	= confidence interval
CSFD	= cerebrospinal fluid drainage
LSA	= left subclavian artery
MAD	= multilevel aortic disease
OR	= odds ratio
SCI	= spinal cord injury
TEVAR	= thoracic endovascular repair

treatment was discussed and approved collegially by the operating team. All patients gave their consent to participate. All descending thoracic treatments were TEVAR. When open abdominal aortic repair was preferred, it was performed via a midline laparotomy and transperitoneal approach. When simultaneous repair was performed with an endograft in both aortic areas, TEVAR was the first procedure of the operation. When abdominal repair was performed with an open approach (hybrid intervention) during simultaneous repair, TEVAR was performed after infrarenal graft replacement. When it was planned to cover the left subclavian artery (LSA), adjunctive prophylactic revascularization was performed in selected cases at higher risk for SCI (aortic coverage: extent type C or >20 cm plus previous abdominal aortic repair in sequential cases), in those presenting with coronary circulation supplied by the LSA through the left internal thoracic artery, when there was a necessity to cover the origin of the left LSA with hypoplastic contralateral vertebral artery, in left-handed professionals, and in those with left arm arteriovenous fistula for dialysis. Cerebrospinal fluid drainage (CSFD) (Duet; Medtronic Vascular, Santa Rosa, Calif) was not placed on a routine basis but selectively: extensive aortic coverage (entire descending aorta, thoracic coverage >20 cm, thoracic coverage <20 cm with involvement of T₈) or when the LSA was scheduled to be overstented.¹¹ Volume expansion or vasopressors were used to maintain mean arterial pressure greater than 75 mm Hg.¹¹ Perioperative homologous packed red blood cells were administered to optimize hemoglobin concentration greater than 9 g/dL. In patients with CSFD, cerebrovascular pressure was monitored continuously in the perioperative period to achieve a target level of 10 to 12 mm Hg.¹¹ The endovascular devices and conventional grafts used in this experience are reported in the [Appendix E1](#).

Definition

Simultaneous intervention defines the treatment of 2 separate aortic lesions (eg, descending thoracic and infrarenal abdominal) during the same operation. Sequential intervention refers to aortic repair of the 2 aortic lesions in 2 separate operations. All variables analyzed in the study were defined according to the Society of Thoracic Surgeons Adult Database definitions.¹² Morphologic features and outcomes were classified according to the Society for Vascular Surgery/American Association for Vascular Surgery reporting standards.¹³ The extent of the thoracic aortic disease was classified as type A, B, or C as described by Estrera and colleagues.¹⁴ The proximal landing zone of the thoracic endograft was defined following the “arch map” classification.¹⁵ Grading the aortic arch atheroma was defined accordingly to a modified classification.¹⁶ SCI was classified accordingly to the American Spinal Injury Association (ASIA) classification.¹⁷

Data Analysis

Clinical data were prospectively recorded and tabulated with Microsoft Excel (Microsoft Corp, Redmond, Wash). Continuous variables were compared between groups with the Mann–Whitney *U* test

or Kruskal–Wallis test, and Fisher exact tests were used in case of dichotomous variables. A stepwise logistic regression model was developed to identify variables associated with SCI development. The stepwise approach was confirmed by backward and forward methods. The significance within the models was evaluated with the Wald test, and the strength of the association of variables with postoperative delirium was estimated by calculating the odds ratio (OR) and 95% confidence intervals (CIs). The model was calibrated by the Hosmer–Lemeshow goodness-of-fit test and residual diagnostics (deviance and degrees of freedom of β). The discrimination of the model was obtained by calculating the area under the receiver operating characteristic curve. Survivals were estimated by means of the Kaplan–Meier method, and log-rank test was performed for the SCI mortality rate comparison. Results are expressed as mean $\pm$ standard deviation for continuous variables and absolute number with frequencies for the categorical variables. Statistical analysis was computed with SPSS, release 20.0 for Windows (IBM SPSS Inc, Chicago, Ill).

RESULTS

General Population

In the examined period, 4320 patients were treated for thoracic and abdominal aortic diseases. MAD was detected in 77 patients (1.8%), of whom 45 (58.4%) were diagnosed simultaneously ([Figure 1](#)). Thoracic aortic diseases included degenerative atherosclerotic aneurysms ($n = 53$, 68.8%), chronic dissecting aneurysm of the descending aorta ($n = 14$, 18.1%), and penetrating aortic ulcer ($n = 10$, 12.9%). Simultaneous repair was performed in 32 patients (41.5%): In this group, 15 patients (19.5%) had total endovascular treatment ([Figure 2](#)). Sequential repair was performed in 45 patients (58.4%): In this group, 9 patients (11.7%) had total endovascular repair. In the sequential group, the median interval time between the 2 operations was 182 weeks (range, 2–528 weeks). Interventions were as follows: sequential hybrid repair ($n = 34$, 44.1%), simultaneous hybrid ($n = 17$, 22.1%), simultaneous total endovascular repair ($n = 15$, 19.5%), and sequential endovascular ($n = 11$, 14.3%). CSFD was placed in 27 patients (35.0%). Endograft deployment was

FIGURE 1. Combinations of aortic pathologies that were treated for multilevel aortic repair. AAA, Abdominal aortic aneurysm; ADA, abdominal dissecting aneurysm; DTA, thoracic dissecting aneurysm; PAU, penetrating atherosclerotic ulcer; PAU-A, abdominal penetrating aortic ulcer; PAU-T, thoracic penetrating aortic ulcer; TAA, thoracic aortic aneurysm.

FIGURE 2. Preliminary CT-angiography (A) showing the simultaneous extent type C thoracic aortic aneurysm and infrarenal aortic aneurysm. Follow-up CT-angiography (B) after total endovascular treatment.

technically successful in all areas. Intraoperative mortality was not observed, and conversion to open repair was never required. Forty-four patients (57.1%) were admitted to the intensive care unit. There were no complications at the site of CSFD insertion; no cerebral hemorrhagic complication was observed.

Spinal Cord Ischemia

Postoperative SCI developed in 6 patients (7.8%) (Table 1). Neurologic injury was ASIA grade C in 4 patients (5.2%) and grade A or B in 1 patient (1.3%) each. Four (5.2%) SCIs resolved (all ASIA grade C), and 2 cases

(2.6%) (ASIA grade A and B) were permanent injuries. There were no significant differences between patients with SCI and patients without SCI in terms of comorbidities and operative risk profile (Table 2). No differences were noted for the characteristics of aortic disease, for thoracic or abdominal location, although patients with SCI had a slightly higher prevalence of dissection in the abdominal aorta (33% vs 6%, $P = .051$). The distance of the distal aortic neck from the celiac trunk was the only significant variable associated with SCI development (2.5 ± 2.3 cm vs 7.4 ± 5.9 cm, $P = .038$). In regard to technical and operative data, CSFD, LSA coverage, and preventive LSA revascularization did not influence SCI occurrence ($P = .659$, $P = .999$, and $P = .494$, respectively). Likewise, operative strategies were not SCI related (OR, 1.2; 95% CI, 0.50-2.86; $P = .688$) (Table 3). On multivariate analysis, the distance from the celiac trunk was the only independent predictor of postoperative SCI (OR, 0.75; 95% CI, 0.56-0.99; $P = .046$); open surgical repair of the abdominal aortic aneurysm was associated with a higher risk of SCI but did not reach statistical significance (OR, 0.16; 95% CI, 0.02-1.06; $P = .057$) (Table 4). With all patients considered, the variables of the model correctly predicted 92% of the observed cases of postoperative SCI. The area under the receiver operating characteristic curve for this model was .856, suggesting a good ability to distinguish between individuals in whom SCI will develop and those in whom SCI will not develop. The Hosmer–Lemeshow goodness-of-fit test was not significant for lack of fit (chi-square [8 df] = 7.75, $P = .458$), indicating that there was no statistically significant departure from a perfect fit. Figure 3 displays the probability of SCI occurrence in relation to the distance from celiac trunk and type of approach to abdominal region.

Outcomes and Survival

Operative mortality was 9.1% ($n = 7$). Although patients with SCI had a 2-fold risk of operative mortality, it was not statistically significant (17% vs 9%, $P = .447$). Patients with SCI had a longer intensive care unit stay (8 ± 5 days vs 3 ± 14 days, $P = .006$) and a longer, although not significantly different, hospitalization (21 ± 14 days vs 14 ± 15

TABLE 1. Summary of procedural details and neurologic deficits of 6 patients with postoperative spinal cord injury

Patient	Intervention	Thoracic disease	Delay (wk)	LSA coverage	Aortic coverage (cm)	CT distance (mm)	AAA procedure	CSFD	Neurologic injury*	Onset	Recovery
#1	Sequential	TAA	56	No	C (35)	6.0	EVAR	No	Grade A	<6 h	No
#2	Simultaneous	Ulcer	0	No	A (10)	5.4	EVAR	No	Grade C	>6 h	Yes
#3	Sequential	DTA	6	Yes	B (15)	0	EVAR	Yes	Grade B	<6 h	No
#4	Sequential	DTA	172	No	C (30)	0	EVAR	No	Grade C	<6 h	Yes
#5	Simultaneous	TAA	0	No	C (25)	2.0	Open	Yes	Grade C	<6 h	Yes
#6	Simultaneous	TAA	0	No	B (15)	2.4	Open	Yes	Grade C	<6 h	Yes

AAA, Abdominal aortic aneurysm; CSFD, cerebrospinal fluid drainage; CT, celiac trunk; DTA, dissecting thoracic aortic aneurysm; EVAR, endovascular aortic repair; LSA, left subclavian artery; TAA, thoracic aortic aneurysm. *Severity grade according to American Spinal Injury Association.

ACD

TABLE 2. Demographic data, risk factors, and operative outcomes

Variable*	All patient n = 77	No SCI n = 71	SCI n = 72	P value
Demographic				
Age, y	72 ± 8	72 ± 7	66 ± 14	.093
Gender (male)	70 (91)	65 (92)	5 (83)	.447
Comorbidities				
CAD	27 (35)	26 (37)	1 (17)	.417
LVEF <25%	3 (4)	3 (4)	0 (0)	.999
Arrhythmia	21 (27)	21 (30)	0 (0)	.181
Hypertension	71 (92)	65 (92)	6 (100)	.999
Diabetes	12 (16)	11 (16)	1 (17)	.999
Dyslipidemia	45 (58)	41 (58)	4 (67)	.999
COPD	38 (49)	37 (52)	1 (17)	.200
CVA	8 (10)	8 (11)	0 (0)	.999
eGFR <30 mL/min/1.73 m ²	5 (7)	5 (7)	0 (0)	.622
Thoracic aorta				
Pathology				.542
Atherosclerotic	53 (69)	50 (70)	3 (50)	
Dissection	14 (18)	12 (17)	2 (33)	
Penetrating ulcer	10 (13)	9 (13)	1 (17)	
Diameter (mm ± SD)	61 ± 18	61 ± 18	55 ± 21	.435
Thoracic disease extent				.398
Type A	17 (22)	17 (24)	0 (0)	
Type B	30 (39)	27 (38)	3 (50)	
Type C	30 (39)	27 (38)	3 (50)	
Arch atheroma				.774
Grade 0	2 (3)	2 (3)	0 (0)	
Grade 1	50 (65)	45 (63)	5 (83)	
Grade 2	21 (27)	20 (28)	1 (17)	
Grade 3	4 (5)	4 (6)	0 (0)	
Distance from CT (mm ± SD)	7 ± 6	7 ± 6	3 ± 2	.038
Abdominal aorta				
Pathology				.051
Atherosclerotic	70 (90)	66 (93)	4 (67)	
Dissection	6 (8)	4 (6)	2 (33)	
Inflammatory	1 (1)	1 (1)	0 (0)	
Diameter (mm ± SD)	60 ± 11	60 ± 11	56 ± 4	.381
Intraoperative details				
Timing				.688
Simultaneous	32 (39)	29 (41)	3 (50)	
Sequential	45 (58)	42 (59)	3 (50)	
Proximal landing zone				.579
“zone 0”	4 (6)	4 (6)	0 (0)	
“zone 1”	3 (4)	3 (4)	0 (0)	
“zone 2”	10 (13)	8 (11)	2 (33)	
“zone 3”	35 (46)	33 (47)	2 (33)	
“zone 4”	25 (33)	23 (32)	2 (33)	
CSFD	27 (35)	24 (34)	3 (50)	.659
LSA coverage	17 (22)	16 (23)	1 (17)	.999
LSA revascularization	8 (10)	7 (10)	1 (17)	.494
Aortic coverage (mm ± SD)	200 ± 85	201 ± 84	217 ± 98	.694
EG diameter (mm ± SD)	38 ± 4	38 ± 4	37 ± 4	.651
EG implanted (n ± SD)	1.5 ± 0.7	1.5 ± 0.7	1.5 ± 0.6	.897
Open abdominal repair	51 (66)	49 (69)	2 (33)	.171

(Continued)

TABLE 2. Continued

Variable*	All patient n = 77	No SCI n = 71	SCI n = 72	P value
Postoperative details				
ICU (d ± SD)	4 ± 13	6 ± 18	9 ± 4	.002
ICU admission	44 (57)	39 (55)	5 (83)	.230
LOS (d ± SD)	15 ± 15	14 ± 15	21 ± 14	.078
Hospital mortality	7 (9)	6 (9)	1 (17)	.447

CAD, Coronary artery disease; COPD, chronic obstructive pulmonary disease; CSFD, cerebrospinal fluid drain; CT, celiac trunk; CVA, cerebrovascular accident; EG, endograft; eGFR, estimated glomerular filtration rate; ICU, intensive care unit; LOS, length of stay; LSA, left subclavian artery; LVEF, left ventricular ejection fraction; SCI, spinal cord ischemia; SD, standard deviation. *Data for continuous variables are shown as mean SD, and data for categorical variables are shown as number (%).

days, $P = .078$). Finally, the median follow-up was 15 months (range, 1-108 months). During the follow-up, type 1 endoleak was detected in 8 patients (11.4%). Five (7.1%) thoracic distal type 1 endoleaks were successfully treated with an extender. Two (2.8%) abdominal type 1 endoleaks (proximal $n = 1$, distal $n = 1$) resolved with an extender. Two (2.8%) abdominal type 2 endoleaks resolved after coil embolization ($n = 1$) or spontaneously thrombosed ($n = 1$). Actuarial survival estimates at 1, 2, and 5 years after the procedure were $80\% \pm 5\%$, $68\% \pm 6\%$, and $63\% \pm 7\%$, respectively (Figure 4); survival was not negatively impaired by SCI occurrence ($P = .885$).

DISCUSSION

No comprehensive data are available on the real incidence of MAD, and guidelines for the appropriate treatment strategy are missing. The first potential

TABLE 3. Operative strategy analysis

Variable*	Simultaneous (n = 32)	Sequential (n = 44)	P value
Demographic (%)			
Age (± SD)	69.9 ± 8.3	72.9 ± 6.8	.121
Gender (male)	28 (87.5)	42 (93.3)	.441
Thoracic disease extent (%)			.026
Type A	4 (12.5)	13 (28.9)	
Type B	18 (56.2)	12 (26.7)	
Type C	10 (31.2)	20 (44.4)	
Distance from CT (mm ± SD)	7.5 ± 6.7	6.3 ± 4.4	.820
Operative details (%)			
Covered aorta (mm ± SD)	170.3 ± 70.5	224.6 ± 87.5	.004
CSFD	16 (50.0)	11 (24.4)	.021
LSA coverage	2 (6.2)	15 (33.3)	.005
LSA revascularization	0 (0)	8 (17.8)	.018
Outcomes (%)			
In-hospital mortality	3 (9.4)	4 (8.9)	.942
SCI	3 (9.4)	3 (6.7%)	.688

CSFD, Cerebrospinal fluid drain; CT, celiac trunk; LSA, left subclavian artery; SCI, spinal cord injury; SD, standard deviation. *Data for continuous variables are shown as mean SD, and data for categorical variables are shown as number (%).

TABLE 4. Predictors of spinal cord injury: Univariate and multivariate analyses

Variables	Univariate OR (95% CI)	P value	Multivariate OR (95% CI)	P value
Age (y)	0.92 (0.84-1.01)	.093		
Arrhythmia	0.89 (0.82-0.98)	.181		
COPD	0.18 (0.20-1.65)	.200		
Distance from CT (mm)	0.78 (0.59-1.01)	.038	0.75 (0.56-0.99)	.046
Abdominal pathology*	3.39 (0.69-16.61)	.051		
Open abdominal repair	0.22 (0.04-1.32)	.171	0.78 (0.02-1.06)	.057

CI, Confidence interval; COPD, chronic obstructive pulmonary disease; CT, celiac trunk; OR, odds ratio. *Atherosclerotic abdominal disease versus dissection and inflammatory.

explanation may lie in the fact that MAD is an uncommon finding.¹⁻⁴ We did not perform a specific analysis on the incidence of MAD, but our results are in accordance with the literature. The 1.8% rate of multilevel aortic repair in our study is similar to the 3.4% reported by Głowiczki and colleagues,² confirming that treatment for MAD accounts for a small part of all aortic aneurysm interventions. Another similarity with the literature is the incidence ratio of simultaneous and sequential aortic lesions. In our experience, 58.4% of the cases were recognized before the first operation, data that are close to the 46% to 54% reported in previous published series.^{2,3}

Uncertainty around MAD treatment strategy has been generated by the conflicting data published so far (Table 5).^{5-9,18-22} Two recent articles showed that TEVAR could increase the occurrence of SCI in patients with a history of abdominal aortic aneurysm repair by approximately 7- to 14-fold.^{5,6} On the contrary, Martin and colleagues⁷ reported that a prior abdominal aortic aneurysm repair, per se, was not significantly associated with SCI. An interim analysis of the European Registry

of Endovascular Aortic Repair Complications added uncertainty.²³ In that study, SCI was associated with the simultaneous closure of at least 2 independent vascular territories supplying the spinal cord, but previous closure of a single vascular territory was not associated with an increased risk of symptomatic SCI. An interesting point of our study is the comparison of simultaneous and sequential treatment of MAD. Although we used adjuncts, the 7.8% overall rate of SCI is still disappointingly high but not outside the reported range for an isolated TEVAR.²⁴

Because of the heterogeneity of MAD presentation and the multiple aspects involved in every type of treatment, our work does not have the presumption to indicate definitively the superiority of one treatment over the other. The reason why simultaneous and sequential MAD treatment had no significantly different SCI rate might be explained in 2 different ways: on the one hand, the hypothetical increased risk of the simultaneous treatment of the 2 districts can benefit from the rapidity and minimally invasiveness of TEVAR, especially for less-extensive aortic coverage. On the other hand, although sequential repair required a more extensive aortic coverage, spinal cord blood flow might have benefited from the recruitment of collaterals over time.²⁵

This hypothesis deserves further consideration. Etz and colleagues²⁶ showed that SCI may occur less frequently when the repair is performed in a 2-stage approach rather than a single procedure despite a higher number of intercostal arteries sacrificed. This experience included open repair only, but Johnston and colleagues²⁷ obtained the same results using a hybrid operation, taking advantage of a TEVAR-first approach that was subsequently completed with an abdominal open graft repair. On the basis of these observations, if a sequential repair is advocated as

FIGURE 3. Probability of SCI occurrence in relation to the distance of the distal neck from celiac trunk and type of abdominal aortic repair. EVAR, Endovascular repair; SCI, spinal cord injury.

FIGURE 4. Kaplan-Meier estimates of survival. SCI, Spinal cord injury.

the best strategy for the treatment of MAD, it should be equally noted that an increased interval time among 2 aortic interventions has been found to be predictive of SCI.¹⁰ It is disturbing that in our sequential interventions, 3 cases of SCI developed in sporadic time intervals. Although MAD is a noncontinuous disease, it could be at increased risk for SCI similar to the thoracoabdominal extent; therefore, it would be more useful to identify the ideal time interval between the 2 operations when MAD is planned to be repaired sequentially.

The length of aortic coverage has been reported as a predictive factor of SCI; however, definitive consensus on that has not been reached so far.^{7,28,29} We expected the length of aortic coverage would play a role in SCI development, but in our experience the only determinant of SCI was the distance of the landing zone from the celiac trunk. The “collateral network” concept showed intercostal artery occlusion may not be sufficient per se to induce SCI, but those intercostals between T₈ and L₂ play a critical role in spinal perfusion.^{8,25,28} Although Amabile and colleagues³⁰ found aortic coverage to be predictive of SCI, they were able to hypothesize only a greater risk of SCI with the coverage of the distal third of the thoracic aorta.

Our analysis confirms the importance of this anatomic aspect. By analyzing our SCI cases, we observe this event occurred only after an extent type B or C coverage. Even in the TEVAR era, the involvement of the most distal segment of the descending thoracic aorta in MAD is at a higher risk of SCI development. Another interesting observation comes from the experience of Takagi and colleagues.³¹ They did not report SCI in patients with proximal thoracic aortic repair, whereas their only case of paraplegia occurred in the descending group despite the reattachment of the seventh and eighth intercostal arteries. With all these aspects considered, we speculate that simultaneous repair of MAD might be more suitable in case of extent type A aortic lesion, whereas distal descending replacements should be approached with caution.³⁰

To date, only 1 article has reported a comparison of the type of MAD treatment in which an endograft is used in 1 or both aortic segments. Aguiar Lucas and colleagues⁸ published the results of 38 MAD cases and did not observe SCI with hybrid or totally endovascular repair, with a simultaneous or sequential strategy. The SCI rate of hybrid and total endovascular strategy also did not differ significantly in our experience, but we did observe some cases of SCI. In view of the predictor of SCI identified in our analysis, these data may be justified by the fact that in our series the most distal segment of the descending aorta was involved 4 times more frequently than in their experience.

Perioperative adjuncts have been associated with improved neurologic outcomes after open conventional thoracic aortic repair.³² Although CSFD and LSA bypass have been recognized as important adjuncts to limit the risk of SCI, there are no definitive statements regarding their mandatory use during TEVARs.³² Hnath and colleagues³³ prospectively evaluated their standardized protocol of CSFD during TEVAR and showed that selective use may offer the same benefit as mandatory drainage. Similarly for LSA management, in the experience of Maldonado and colleagues,³⁴ prior aortic repair did not seem to be a predictor of SCI when a strategy of selective revascularization was adopted. Our SCI rate was not irrelevant, even in this endovascular era. However, we should remember that in contrast to that study, our cohort inherently was at a higher risk of SCI. Admittedly, our

TABLE 5. Multilevel aortic disease treatment: Literature summary

Authors	Year	Treatment (Simultaneous/sequential)	SCI (%)	Permanent (%)	Predictors
Baril and colleagues ⁵	2006	1/27	14.3	10.7	NSR
Martin and colleagues ⁷	2009	0/51	26.0	NSR	History of AAA repair
Schlösser and colleagues ⁶	2009	0/72	12.5	6.9	LSA coverage
Aguiar Lucas and colleagues ⁸	2009	3/36	0	0	
Scali and colleagues ¹⁰	2012	22/0	13.6	4.5	Renal insufficiency

AAA, Abdominal aortic aneurysm; LSA, left subclavian artery; NSR, not specifically reported; SCI, spinal cord injury.

selective approach of LSA bypass and CSFD can mask their real clinical impact. It is also true that using a selected approach has allowed us to have similar risks in conditions at greater risk for SCI.

Long-term results have not been widely reported in MAD series. In their remarkable experience, Crawford and Cohen¹ reported that long-term survival was better in patients with complete aortic treatment because most of the early deaths in the sequential intervention were caused by rupture of the second aneurysm.^{2,4} On the other hand, Gloviczki and colleagues² showed improved survival at 3 and 5 years after sequential repair, although it was not statistically significant. Few current series have reported long-term follow-up for MAD: Moon and colleagues⁴ published actuarial survival estimates of 87% at 1 year and 81% at 3 years, but the mean follow-up was only 13.2 months. Scali and colleagues¹⁰ demonstrated an overall survival of 81% at 1 year for simultaneous totally endovascular repair. Our data show results comparable to those of older reports on MAD; our overall 63% survival at 5 years reflects the significant impact of such an extensive disease on survival is similar to the reported long-term survival of thoracoabdominal aortic diseases.³⁵

Study Limitations

The multicenter design of this study has several limitations. The sample size of the 2 groups remains small for adequate analyses. The rarity of this condition makes a large and homogeneous study difficult to be realized. Another important limitation was the retrospective design of the study. The recruiting centers had significant expertise and experience in open and endovascular treatment of aortic pathologies, but selection bias may have occurred. Third, our data were prospectively collected and analyses finally identified a significant predictor, but potential confounding variables may have affected the results. Furthermore, the absence of guidelines for the management of MAD makes the study heterogeneous in treatment strategy. Despite these limitations, a similar design is present in other studies and our cohort is currently one of the largest available series, offering new sources of debate.^{5,7-9}

CONCLUSIONS

Our study supports previous findings that MAD is an uncommon clinical entity, either in terms of the incidence rate or the impact on overall aortic interventions. Despite the best efforts to refine treatment strategy and the use of technical adjuncts, the risk of SCI is still substantial at 8% in patients with MAD, even in this endovascular era. In our experience, the distance of the distal landing zone from the celiac trunk was the only predictive factor of SCI. This means the coverage of the distal descending thoracic aorta in MAD is at a higher risk of SCI.

Long-term survival results are satisfactory and seem to be comparable to those of thoracoabdominal aortic diseases.

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APPENDIX E1

Thoracic endograft:

- Excluder/TAG/C-TAG (WL Gore & Associates;
Flagstaff, Ariz)
- Talent/Valiant/Captivia (Medtronic Vascular, Santa
Rosa, Calif)
- Relay (Bolton Medical, Sunrise, Fla)
- TX-1/TX-2 (Cook, Bloomington, Ind)

Abdominal endograft:

- Excluder/C3 (WL Gore & Associates)
- Talent/Endurant (Medtronic Vascular)
- Zenith (Cook)

Conventional abdominal graft:

- Uni-graft (B. Braun Medical, Melsungen, Germany)
- Intergard (Maquet GmbH, Rastatt, Germany)
- Vascular graft (WL Gore & Associates)