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ORIGINAL ARTICLE

Spinal cord ischemia in endovascular repair of thoracoabdominal and complex abdominal aortic aneurysm: a single-center cohort study

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ABSTRACT

INTRODUCTION: Spinal cord ischemia (SCI) is an uncommon but one of the most terrifying complications associated with the treatment of extensive aortic aneurysms. Its pathophysiology, risk factors, and event characteristics must be clarified. This study aimed to perform a descriptive analysis of the cases of SCI after complex aortic repair at our center and to understand which factors contributed to the event.

METHODS: A retrospective, single-center, cohort study of prospectively collected data was performed. From January 2013 to October 2022, we included all consecutive patients with thoraco-abdominal aneurysms (TAAA) and complex abdominal aneurysms submitted to an endovascular procedure using fenestrated or branched endografts. According to the Society for Vascular Surgery (SVS) reporting standards, SCI was defined as the development of new motor or sensitive deficits after endovascular treatment.

RESULTS: One hundred and thirty-five patients (87% male) were included, with a median age of 72. A total of 53,7% (n=72) had thoraco-abdominal aneurysms (type I-V), of which 63 were extent I-III\V and 9 were extent IV. In total, 12 (9%) SCI events were identified; 7 patients had a grade 2 SCI, 5 had a grade 3 injury, and all of them had a TAAA. Of these, 10 had pre-procedure cerebrospinal fluid (CSF) drainage. Five (42%) developed symptoms after 48 hours (delayed SCI), and seven patients developed early SCI, being identified on the first evaluation after surgery. Five cases received rescue CSF drainage (after the beginning of symptoms), with some degree of improvement observed in all cases. Among patients with grade 2 SCI (n=7), five recovered completely, one recovered partially (maintaining sensitive symptoms – grade 1), and one did not recover.

This corresponded to an overall incidence of permanent SCI of 5.2% (n=7, any grade) and 1.5% of permanent paraplegia (n=2, grade 3, 2.8% only in TAAA).

CONCLUSION: In this study, SCI only occurred in patients with TAAA, and the incidence of permanent paraplegia in this group was 2,8%. In five patients, rescue CSF drainage was applied, with some degree of clinical improvement. The small number of events precluded the investigation of predictors. Notably, 42% of SCI cases happened after 48 hours, showing a high prevalence of delayed SCI in this population. Consequently, more extended monitoring of these patients should be considered.

Keywords: Spinal cord ischemia; Complex aortic aneurysm; Complex endovascular aortic repair; Thoracoabdominal aortic aneurysm.

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INTRODUCTION

Spinal cord ischemia (SCI) remains one of the most terrifying complications associated with the treatment of extensive aortic aneurysms, having a significant impact on a patient's survival.[1] Endovascular treatment avoids aortic cross-clamping and minimizes visceral ischemia, but due to intercostal artery exclusion, hemodynamic repercussion, and impaired lower limb perfusion during the procedure, SCI remains a dreadful complication in endovascular repair of extensive aortic aneurysms.[1]

While endovascular treatment with branched or fenestrated stent grafts offers many practical advantages, it is essential to acknowledge that the occurrence of SCI after the procedure is not negligible. Previous studies show rates of permanent SCI (any symptom) of 1-10% and permanent paraplegia of 1-4%.[2,3]

Given the loss of thoracic and lumbar segmental arteries that mainly contribute to spinal cord vascularization[4], the extension of aortic disease and consequent coverage is one of the main predictors of the development of neurological deficits.[5]

Cerebrospinal fluid (CSF) drainage, staged treatment, and perioperative prevention protocols have led to a significant decrease in the development of SCI in endovascular complex aortic repair.[6,7] However, disease comprehension, risk factors, and event characteristics still need to be clarified entirely.

This study aimed to perform a descriptive analysis of the cases of SCI after complex aortic repair at our center and understand which factors contributed to the event.

METHODS

Reporting guidelines from the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) statement for cohort studies were followed.[8]

Study design and population

A retrospective, single-center, cohort study of prospectively collected data was performed. We included all consecutive patients with thoracoabdominal and complex abdominal aneurysms submitted to an endovascular procedure using fenestrated or branched endografts enrolled from January 2013 to October 2022.

Patients with thoracoabdominal extent I-V, pararenal, and juxtarenal abdominal aortic aneurysms were included. Indications for repair included asymptomatic patients with a diameter above the ESVS-defined treatment threshold[9,10] or symptomatic or ruptured aneurysms. Custom-made devices using branches and/or fenestrations (COOK Medical Platform), off-the-shelf devices (T-branch, COOK Medical), or physician-modified stent grafts were used. Patients submitted to aortic arch repair or using other endovascular techniques, such as parallel grafts or endostaples, were excluded.

Procedural details

Endovascular procedures were performed using angiography equipment with fusion imaging (Phillips Azurion). A cutdown bilateral common femoral access was commonly used, and in branched cases, a left cutdown axillary access was used additionally. The procedures occurred under controlled heparinization with the aim of activated clotting time (ACT) of 250-300 seconds. A SCI prevention protocol was implemented in all cases, including selective prophylactic CSF drainage and monitoring (target cerebrospinal fluid pressure is set under ten mmHg, with a goal drainage rate of 10-15 ml/h; these targets are maintained for up to 3 days in the absence of SCI), permissive intra and postoperative hypertension (aiming for a spinal cord perfusion pressure >80mmHg), glucose control, oxygenation optimization using a 10g/dl hemoglobin threshold for transfusion and a target peripheral oxygen saturation over 95%. The staging was performed in most cases with an intended aortic coverage over the celiac trunk extending over 6cm but adapted case-to-case according to surgeon preference and estimated SCI risk.

After surgery, all patients remained at least 24 hours in an intensive care unit and were posteriorly transferred to a vascular surgery intermediate care unit for at least 48 hours. Rigorous neurological evaluation was maintained with frequent evaluation of sensitive and motor deficits of the lower limbs (every hour during the first 24h hours followed by every two h hours on the second day). In case of de novo neurologic deficits, rescue CSF drainage was implemented (after the beginning of symptoms) if not prophylactically implemented before or if the previous prophylactic drainage catheter has already been removed.

Outcomes, variables, and definitions

According to the Society for Vascular Surgery (SVS) reporting standards,[11] SCI was defined as developing new motor or sensitive deficits after endovascular treatment (Table 1). Additionally, we defined early SCI as being present in the first 48 hours after surgery or at the first clinical examination if the patient was under sedation for more than 48 hours, after which we considered it to be delayed SCI. Patient selection and data collection were based on surgical and electronic medical records. Collected data included patients' age, sex, comorbidities (hypertension, dyslipidemia, diabetes, smoking habits, chronic renal disease), type of aneurysm (thoraco-abdominal according to Crawford classification), aneurysm diameter, risk classification of American Society of anaesthesiology (ASA), previous aortic surgery, elective or urgent surgery, used technique (fenestrated or branched), extension of covered aorta (aortic zones), patency of hypogastric arteries, access used, amount of blood loss, previous staging, CSF drainage, presence of endoleak on the first angiography, time of hospital stay and 30-days mortality.

Table 1. Spinal cord ischemia classification, according to the Society for Vascular Surgery reporting standards.[11]

Grade	Description
0	No neurologic deficit
1	Minimal sensory deficit with no motor deficit and ability to walk independently
2	Paraparesis: minor motor deficit with the ability to walk with assistance or independently. This definition implies that the patient can move the extremity against gravity
3	Paraplegia, severe motor deficit causing inability to walk
3A	Nonambulatory with the ability to move extremities against gravity
3B	Nonambulatory with the ability to move extremities laterally but not against gravity
3C	Nonambulatory with minimal or no movement

A descriptive analysis of all SCI cases was performed to understand event characteristics and their incidence. A comparative analysis was also performed comparing patients who developed or did not have SCI to understand possible event predictors. Outcomes analyzed included differences regarding CSF drainage and monitoring protocol, extent of aneurysm repair, patency of hypogastric arteries, blood loss during surgery, staging, duration of hospital stay, and 30-day mortality.

Statistical analysis

Statistical analysis was carried out using STATA version 16.1 (Statistics/Data analysis, StataCorp LLC, Texas, USA). Continuous variables are presented as mean ± standard deviation when normally distributed and median (interquartile range-IQR) when not. Categorical variables are expressed in numbers (percentage). Student's t-test was used when comparing continuous variables and the $\chi 2$ test to compare categorical variables; however, when appropriate, the Mann-Whitney or Fisher Exact test was used instead, respectively.

Survival and patency estimates were obtained using Kaplan-Meier graphs and the Log Rank test for group comparison. A logistic regression was performed for the primary outcomes to adjust for confounders when comparing groups. Baseline demographics that were statistically different in univariate analysis were included in the regression model. Results were presented as odds ratios (OR) with 95% confidence intervals (CI).

All analyses were considered statistically significant if a two-tailed p-value < 0.05 was observed.

RESULTS

Demographics and characteristics

The study included 135 cases; 72 (53.7%) had thoracoabdominal aneurysms (type I-V), and 63 had complex abdominal aneurysms (suprarenal, pararenal, and juxtarenal).

The median age was 72 years (±8 years), and 87% of the patients were male. Table 2 details the patients' demographics and comorbidities.

Table 2. Baseline characteristics of patients with thoracoabdominal or complex abdominal aortic aneurysms treated with endovascular repair

Characteristics	n=135
Male – n (%)	118 (87,4)
Age, years – mean ± SD	71,6 ± 8
ASA ≥3 – n (%)	123 (91)
Arterial hypertension – n (%)	123 (91)
Diabetes mellitus – n (%)	24 (18)
Hyperlipidemia – n (%)	98 (73)
Tobacco use – n (%)	86 (64)
CKD – n (%)	46 (34)
Period of hospital stay (days) – mean ± SD	10 ± 9

ASA - American Society of Anesthesiology; **CKD** - chronic kidney disease; **SD** – standard deviation

Diagnosis and procedure details

The mean aneurysm size was 65.4mm (± 14mm). According to the Crawford classification, among the patients with thoraco-abdominal aneurysms, 63 were extent I-III\V, and 9 were extent IV (Table 3). Two complex aortic aneurysms were suprarenal, 17 were pararenal, and 44 were juxtarenal. Post-dissection aneurysm was found in 13 patients. Four of the nine acute cases corresponded to rupture (contained), and 5 cases were symptomatic (Table 3).

Table 3. Anatomical and procedural characteristics of thoracoabdominal or complex abdominal aortic aneurysms treated with endovascular repair

Characteristics	n=135
TAAA (Crawford classification) – n (%)	72 (53)
Extent I – n (%)	8/72 (11)
Extent II – n (%)	16/72 (22)
Extent III – n (%)	28/72 (40)
Extent IV – n (%)	9/72 (13)
Extent V – n (%)	11/72 (15)
Complex abdominal aortic aneurysm – n (%)	63 (47)
Supra-renal – n (%)	2/47 (3)
Para-renal – n (%)	17/47 (27)
Juxta-renal – n (%)	44/47 (65)
Post-dissection aneurysm – n (%)	12 (9)
Acute repair – n (%)	9 (7)
symptomatic unruptured – n (%)	5/9 (56)
Ruptured – n (%)	4/9 (44)
Maximum aortic aneurysm diameter, mm – mean ± SD	66 ± 14
Previous aortic surgery – n (%)	33 (24)
Aortic Mural Thrombus (Moderate-Severe) – n (%)	27 (20)

TAAA - thoracoabdominal aortic aneurysm

Outcomes - spinal cord ischemia descriptive analysis

Spinal cord ischemia (any deficit) was found in 12 (9%);7 patients had a grade 2 spinal cord injury, and 5 had a grade 3 injury. All the cases occurred in patients with a TAAA (p=0.001).

Five patients (42%) developed symptoms after 48 hours (delayed SCI), and seven patients developed early SCI, being identified on the first evaluation after surgery.

Table 4 details every case, describing the type of aneurysm according to Crawford's classification, the time of presentation and evolution of symptoms, the presence of prophylactic CSF drainage, and the associated mortality.

Table 4. Individual analysis of patients with thoracoabdominal or complex abdominal aortic aneurysms treated with endovascular repair, complicated with spinal cord ischemia

					Spinal Cord Injury (Grade)	
No.	**Crawford classification**	**Prophylactic CSF drainage**	**Symptom onset**	**Rescue CSF drainage**	**At onset**	**At discharge**
1	III	Yes	Early	No	2	0
2	III	Yes	Early	No	2	0
3	III	Yes	Delayed	No	3c	3c
4	III	Yes	Early	No	2	0
5	V	Yes	Early	No	2	2
6	III	Yes	Delayed	Yes	3c	2
7	II	Yes	Delayed	No	2	0
8	I	No	Delayed	Yes	3c	2
9	IV	Yes	Early	Yes	2	1
10	I	No	Early	Yes	3c	2
11	III	Yes	Delayed	Yes	2	0
12	II	Yes	Early	No	3c	3c

CSF - cerebrospinal fluid

Among patients with third-degree ischemia (n=5), two did not recover, and three recovered partially. Among patients with grade 2 SCI (n=7), 5 recovered completely, 1 recovered partially (maintaining sensitive symptoms—grade 1), and one patient did not recover.

Overall, this corresponded to an incidence of permanent SCI of 5.2% (n=7, any grade) and 1.5% of permanent paraplegia (n=2, grade 3). Considering only TAAAs, this corresponded to a rate of 9.7% and 2.8%, respectively.

Spinal cord ischemia-related factors and impact on postoperative outcomes

Prophylactic CSF drainage was performed in 61 cases. Of the 12 cases with SCI, 10 had pre-procedure CSF drainage. Specifically, among patients who developed early SCI (n=7), 6 had prophylactic CSF drainage, while in those who developed late SCI, only 1 had prophylactic CSF drainage at the onset of symptoms. SA statistically significant difference was not observed in both groups (with or without SCI). Among the cases of SCI, 5 received rescue CSF drainage (after the beginning of symptoms), with some recovery observed in all patients. Also, in association with CSF drainage, there was only one major complication with the technique (intra-dural hematoma).

The planned staging was used in 24 patients, 22 of whom were TAAAs (30.5% of TAAAs), with a time between stages 93,1 ± 107 days. Additionally, 17 patients had historical staging (meaning previous open or endovascular aortic repair, initially not intended as a staging procedure), and all of these were TAAAs (23.6% of all TAAAs). This corresponded to 41 patients with any staging, 39 of which TAAAs (54.2% of all TAAAs).

In cases of SCI (n=12), only three patients were submitted to planned staging, and an additional two patients had historical staging. There was no statistical difference regarding any sort of staging and the development of SCI (p=0.49 for planned staging, p=0.72 for historical staging, and p=0.41 for any sort of staging).

A univariable analysis was performed in patients with TAAAs, comparing patients with or without postoperative SCI (Table 5). Patients who developed SCI had lower median baseline GFR of 51 ml/min/1.73m2 (36.3-60.3) compared to 72 ml/min/1.73m2 (62.4-87.5), p=0.014 and higher rates of moderate-to-severe mural thrombus (p=0.044). Hyperlipidemia was less common in patients who developed SCI, although it did not reach a statistically significant difference (p=0.08). Surprisingly, intraoperative blood loss >1000ml was inversely related to the occurrence of SCI (p=0.010). Patients who developed postoperative SCI had a more extended length of hospital stay (median of 16 days (10-30) versus a median of 9.5 days (6-13), p=0.007).

Table 5. Univariable analysis of risk factors for spinal cord ischemia after endovascular repair of thoracoabdominal aortic aneurysm

Characteristics	**No SCI (n=60)**	**SCI (n=12)**	**P value**
Male gender – n (%)	48 (80)	10 (83.3)	0.79
Age, years – median (IQR)	71 (66.5-75.5)	71 (69-75)	0.78
Arterial hypertension – n (%)	55 (91.7)	11 (91.7)	>0.99
Diabetes mellitus – n (%)	11 (18.3)	0	0.11
Hyperlipidemia – n (%)	45 (75.0)	6 (50.0)	**0.08**
Tobacco use – n (%)	44 (73.3)	8 (66.7)	0.63
CKD – n (%)	21 (35.0)	7 (58.3)	**0.07**
GFR (ml/min/1.73m2) – median (IQR)	72 (62.4-87.5)	51 (36.3-60.3)	**0.014**
ASA ≥ III – n (%)	55 (91.7)	10 (83.3)	0.37
COPD – n (%)	20 (33.3)	4 (33.3)	>0.99
Crawford Extent I – n (%)	6 (10)	2 (16.7)	0.70
Crawford Extent II – n (%)	14 (23.3)	2 (16.7)	
Crawford Extent III – n (%)	22 (36.7)	6 (50)	
Crawford Extent IV – n (%)	8 (13.3)	1 (8.3)	
Crawford Extent V – n (%)	10 (16.7)	1 (8.3)	
Maximum aneurysm diameter, mm – median (IQR)	62 (57.5-74.5)	71 (60-80.5)	0.28
Occlusion ≥ 1 hypogastric artery – n (%)	8 (13,3)	1 (8,3)	>0.99
Prophylactic CSF drainage – n (%)	50 (83.3)	9 (75.0)	0.49
Staging – n (%)	19 (31.7)	3 (25.0)	0.65
Historical staging – n (%)	15 (25.0)	2 (16.7)	0.53
Any staging – n (%)	34 (56.7)	5 (41.7)	0.34
Blood loss ≥1000 – n (%)	29 (48.3)	1 (8.3)	**0.010**
Mural thrombus (moderate-severe) – n (%)	17 (28.3)	7 (58.3)	**0.044**
Endoleak at final angiography – n (%)	19 (31.7)	3 (25.0)	0.65
Period of hospital stay (days) – n (%)	9.5 (6-13)	16 (10-30)	**0.007**
30-day mortality – n (%)	7 (11.7)	1 (8.3)	0.74

GFR - glomerular filtration rate; **ASA** - American Society of Anesthesiology; **CKD** - chronic kidney disease; **CSF** - cerebrospinal fluid; **COPD** - chronic obstructive pulmonary disease

In the multivariable analysis (Table 6), we also considered only TAAAs. We did not identify any factor with a statistically significant odds ratio associated with the occurrence of SCI. All previous significant factors lost significance after adjusting for CSF drainage, staging, blood loss >1000ml, hyperlipidemia, mural thrombus, and chronic kidney disease.

Table 6. Logistic regression analysis of risk factors for spinal cord ischemia after endovascular repair of thoracoabdominal aortic aneurysms

Variables	OR	95% CI	P value
Prophylactic CSF drainage	0.56	0.08- 4.00	0.56
Staging (any sort)	0.68	0.14-3.28	0.63
Blood loss >1000cc	0.12	0.01-1.13	0.06
Hyperlipidemia	0.35	0.08-1.52	0.16
Mural thrombus (moderate to severe)	2.43	0.51-11.70	0.27
CKD	2.25	0.50-10.19	0.29

CKD - chronic kidney disease; **CSF** - cerebrospinal fluid; **OR** – odds ratio; **CI** - confidence interval

Overall, 30-day mortality for the whole cohort was 7.4% (n=10), 5.6% (n=7) for elective cases, and 33.3% (n=3) for urgent cases. In TAAAs, 30-day mortality was 11.1% (n=8), 7.7% for elective cases (n=5), and 42.9% for urgent cases (n=3).

30-mortality in patients in patients with or without postoperative SCI was 8.3% (n=1) for patients with SCI compared to 7.3% (n=9) for patients without (p=0.90). This difference was also non-significant for TAAAs (p=0.74).

Overall survival was 88% at 12 months (95%CI: 0.79-0.93) and 83% at three years (95%CI: 0.73-0.89). There was a significant difference in overall survival in patients who developed SCI versus those who did not (OR:2.96; 95%CI: 1.11-7.89; p=0.03, total events: SCI-5 patients; non-SCI - 24 patients). The overall survival in patients with SCI was 80% at 12 months (95%CI: 0.41-0.95) and 53% at three years (95%CI: 0.08-0.85), and for patients without SCI was 88.5% at 12 months (95%CI: 0.80-0.94) and 84% at three years (95%CI: 0.73-0.90).

DISCUSSION

In this cohort study, the overall incidence of permanent SCI was 5.2% (n=7, any grade) and 1.5% of permanent paraplegia (n=2, grade 3). Considering only TAAAs, this corresponded to a rate of 9.7% and 2.8%, respectively. The occurrence of SCI was low in our study and within the range of reported incidence in recent studies.[3,12,13] We did not observe any case of SCI in the subgroup of patients with complex abdominal aneurysms, confirming that SCI is a rare event in this subgroup of patients.[1]

Additionally, patients who developed postoperative SCI had a higher length of hospital stay and a higher rate of overall mortality during follow-up, which had also been reported previously.[14]

In our cohort, 42% of the patients with SCI had delayed the onset of symptoms after the first 48 hours of the postoperative period. Of the five patients that developed delayed SCI, only one did not have preoperative CSF drainage, and 3 received rescue CSF drainage after the onset of symptoms with some degree of improvement of the neurological deficits in all cases. The delayed SCI suggests different possibilities for the underlying pathological mechanisms. Late (>48h) occlusion of endoleaks commonly noted in the control angiography (such as type 3 endoleaks due to high intra-operative ACT values) at the end of the procedure may lead to delayed aneurysm thrombosis and occlusion of intercostal arteries and lead to impairment of spinal cord perfusion at a later stage. Additionally, reperfusion lesions and other systemic factors like postoperative hypotension may also likely play a role in late-occurring SCI.

Due to the time span of our cohort, the protocoled strategies to prevent SCI evolved over time, namely a trend to more selective use of LCR drainage and to increasing use of staging over time. However, none of these factors reached significance in our multivariate analysis, probably due to the low number of patients and events.

Although we did not find statistical differences among the different types of TAAAs, especially between type IV TAAAs and type I-III TAAAs, we observed that SCI did not occur in para-renal or juxta-renal AAAs. However, a significant proportion of these were treated with four fenestrated stent grafts and, in urgent cases, a T-branch. Therefore, the extension of the aneurismatic disease may impact the development of this complication. Aortic coverage, especially in the thoracic aorta, is a major predictor of SCI. This risk factor has been identified in many other studies as one of the main contributors, even when considering the aneurysm extension or the evaluation of the rate of aortic coverage.[3,5,12,13]

In our center, prophylactic CSF drainage is part of the preventive protocol for SCI, which is why almost all patients with TAAA (61 in 72 patients) presented with CSF drainage. As a result, no conclusions could be reached regarding its role in the prevention of SCI. However, prophylactic CSF drainage is described as a preventive factor capable of improving SCI.

However, CSF drainage remains controversial because the risks and complications associated with the technique persist, although the rates of SCI have diminished over time with increased experience.[6,15] Reasons why it is probably necessary to promote specific indications for its use.[6,15]

We had only one case of major complication associated with CSF drainage (intra-dural hematoma). Nevertheless, rescue CSF drainage showed clinical improvement in our patients. Our 5 cases of rescue CSF drainage presented with some degree of improvement after the procedure. Although statistical significance was not reached, previous studies reported advantages with this procedure.[6]

Staged treatment is another option for preventing SCI, supported by vascular remodeling, minimizing the loss of segmented spinal cord vascularization.[4,7,12] This technique was performed in 22 cases, and in patients that developed SCI, just 2 of them were submitted to a staged treatment. Although not statistically significant, it is possibly a protective factor and should be considered.[12]

When analyzing characteristics between groups to determine perioperative perioperative factors associated with SCI, we only identified an association between the GFR, moderate-to-severe mural thrombus, blood loss, and a trend in hyperlipidemia. However, after logistic regression, no predictive factor was identified. This can be explained by the small number of patients included in this study and the small number of events. Comorbidities such as hypertension, arterial periphery disease, chronic renal disease, COPD, and age were already identified as risk factors for the development of SCI.[3,5] This can be explained by the relationship between these diseases and atherosclerosis involving spinal cord vascularization.

Also associated with SCI, other authors report other risk factors such as blood transfusion, the use of aminergic support, and intraoperative low mean arterial pressure.[3,5,6] These results underline the importance of optimizing the hemodynamic instability during the procedure. In our center, the post-operatory protocol includes mean arterial pressure above 80 mmHg (with vasopressor if needed), hemoglobin≥ 10 mg/dL, and regular evaluation of lower limb neurological deficits.

In prophylactic CSF drainage use, it remains in place for at least 48 hours, clamped after 24 hours for symptom evaluation. Nevertheless, as mentioned above, 42% (n=5) of patients developed late SCI, requiring CSF rescue drainage in 3 cases. Contrary to the experience with open surgery, in which most patients who develop SCI present with an early onset event16, endovascular treatment appears to exhibit a higher incidence of late neurological deficits. As a result, more prolonged clinical surveillance and close monitoring of these patients might be necessary.

Our study has several limitations. As mentioned, a small sample with few events limits the ability to obtain statistically significant values and identify predictors of SCI. However, it represents the experience in our center, with descriptive results resembling those of other studies. Additionally, the retrospective nature increases the risk of reporting bias.

SCI still needs to be better understood in endovascular complex aortic repair. Additional studies looking at the characteristics of postoperative late-occurring SCI events are needed to understand what drives these events and what possible strategies might be developed to address them. Additionally, concepts such as prophylactic CSF drainage, the correct way to stage procedures, and perioperative risk factors for SCI are poorly understood and need further investigation.

CONCLUSION

In our study, SCI only occurred in patients treated with a TAAA. The permanent paraplegia rate was 2.8% in this group. Despite the absence of statistical significance, rescue CSF drainage seemed effective in reducing clinical severity. Given the higher prevalence of delayed SCI after endovascular repair, longer postoperative monitoring of these patients should be considered.

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