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Carotid artery dissection and neurological complications after surgery for type 1 aortic dissection

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ABSTRACT

Background Data on the prognostic impact of type A aortic dissection involving the common carotid arteries (CCAs) are scarce.

Methods Data on the status of the CCAs were available in 1106 patients who underwent surgery for acute DeBakey type 1 aortic dissection who were recruited in a retrospective, multicentre European registry, that is, the ERTAAD. Postoperative neurological complications were defined as ischaemic stroke, haemorrhagic stroke and/or global brain ischaemia.

Results Patients without carotid artery dissection, those with unilateral or bilateral CCA dissection had in-hospital mortality rates of 19.5%, 16.9% (OR 1.006, 95% CI 0.614 to 1.647) and 27.3% (p<0.001, OR 1.719, 95% CI 1.086 to 2.722), respectively. Bilateral, but not unilateral, dissection of the CCAs increased the risk of neurological complications (40.0% vs 18.9%, OR 2.453, 95% CI 1.683 to 3.576). The negative prognostic effect of bilateral dissection of the CCAs was increased among patients without cerebral malperfusion who underwent surgery with the use of hypothermic circulatory arrest (28.7% vs 4.3%, p=0.014).

Conclusions Bilateral, but not unilateral, dissection of the CCAs may increase the risk of neurological complications and in-hospital mortality after surgery for DeBakey type 1 aortic dissection.

Trial registration number NCT04831073.

WHAT IS ALREADY KNOWN ON THIS TOPIC

⇒ Data on the prognostic impact of acute type A aortic dissection involving the carotid arteries are scarce.

WHAT THIS STUDY ADDS

⇒ Bilateral dissection of the carotid arteries increased the risk of neurological complications.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

⇒ Hypothermic circulatory arrest may further increase the risk of neurological complications in these patients.

INTRODUCTION

Acute type A aortic dissection (TAAD) is a severe emergency condition requiring prompt surgical treatment. Patients with dissection of the aortic arch, that is, DeBakey type 1 aortic dissection, involving the common carotid arteries (CCAs) may have an increased risk of postoperative neurological complications and mortality when blood flow to the cerebral circulation is diverted or reduced.[1–4] However, controversial findings on the prognostic importance of TAAD-related CCA dissection have been reported as well.[5] Preoperative unstable haemodynamic conditions and anaemia may further reduce the oxygen delivery to the brain in patients with dissection of the carotid arteries, even when carotid artery dissection results in mild vessel stenosis. The possible negative prognostic effect of dissection of the epiaortic vessels is of relevance because surgery of TAAD is frequently complicated by severe neurological complications, and these in turn are associated with high risk of early and late mortality.[6] Noteworthy, carotid artery dissection can be amenable to concomitant surgical treatment,[7–9] and preoperative, intraoperative and postoperative endovascular treatment can be considered potentially effective treatments to prevent or relieve brain ischaemia.[10–17] The prognostic impact of TAAD-related CCA dissection on

postoperative neurological complications and mortality has been investigated in the present multicentre study.

METHODS

Study population

This is an analysis of data from the European Registry of Type A Aortic Dissection (ERTAAD),[18] which was a multicentre study of retrospective nature including 3735 consecutive TAAD patients operated at 17 centres of cardiac surgery in eight European countries (Belgium, Czech Republic, Finland, France, Germany, Italy, Spain and the UK) from January 2005 to March 2021. Data on the date of death were collected retrospectively from electronic national registries as well as by contacting regional hospitals, patients and their relatives. In this study, we followed the Strengthening the Reporting of Observational Studies in Epidemiology guidelines for cohort studies.[19]

The inclusion criteria of the ERTAAD were the following: (1) TAAD or intramural haematoma involving the ascending aorta; (2) patients aged 18 years or older; (3) symptoms related to TAAD started within 7 days from surgery; (4) primary surgical repair of acute TAAD and (5) any other major cardiac surgical procedure concomitant with surgery for TAAD. The exclusion criteria of this registry were the following: (1) patients aged <18 years; (2) onset of symptoms >7 days from surgery; (3) prior procedure for TAAD; (4) retrograde TAAD (with primary tear located in the descending aorta); (5) concomitant endocarditis and (6) TAAD secondary to blunt or penetrating chest trauma.[18] Patients with DeBakey type 1 aortic dissection with data on preoperative CT angiography (CTA) were included in this analysis.

Variables of interest and study outcomes

Cerebral malperfusion was defined as any clinical evidence before surgery of unconsciousness, hemiplegia/hemiparesis, dysarthria/aphasia, severe vision disturbances or vision loss and/or severe confusion. Additional, concomitant or tandem carotid revascularisations were defined as surgical and endovascular procedures for revascularisation of the epiaortic vessels other than reimplantation of the innominate artery and CCA directly to the aortic prosthesis and its branches.

Data on the extent of aortic dissection and involvement of the CCAs were gathered from radiologists' reports of CTA findings or were retrospectively reviewed by on-site experienced cardiac surgeons. The degree of stenosis or occlusion of the carotid arteries was not estimated. Hypothermic circulatory arrest referred to a period of arrest of the systemic circulation with or without the use of antegrade or retrograde cerebral perfusion. The primary outcome of this study was any postoperative neurological event which occurred during the index hospitalisation defined as ischaemic stroke, haemorrhagic stroke and/or global brain ischaemia.[18] The secondary outcomes were in-hospital and 10-year mortality.

Statistical analysis

Categorical variables were reported as counts and percentages. Continuous variables were reported as the means and SDs as well as median and IQR. Missing values were not replaced. Multilevel mixed-effect logistic regression and parametric survival regression analyses considering the potential cluster effects of the participating centres were used to identify the independent predictors of neurological complications and mortality. Regression models included covariates with $p<0.05$ in univariate analysis. Risk estimates were reported as ORs and HRs with their 95% CIs. The discrimination of the regression model was evaluated by estimating the area under the receiver operating characteristics curve of the logit. The Akaike's information criterion (AIC) was estimated to evaluate the goodness-of-fit of the regression models. A $p<0.05$ was considered statistically significant. Statistical analyses were performed with Stata statistical software (V.15.1, StataCorp).

RESULTS

The ERTAAD dataset comprised data of 3735 consecutive TAAD patients. Complete data from CTA were available in 1250 patients, and 1106 (88.6%) patients with DeBakey type 1 aortic dissection were the subjects of this analysis. Characteristics of these patients and operative data are summarised in table 1. The mean delay from the onset of symptoms to surgery was 18±27 hours (median 8.0 hours, IQR 11). Dissection of a CCA was detected at CTA in 38.9% of patients and involved both CCAs in 18.5% of patients.

Cerebral malperfusion was observed in 14.9% of patients without CCA dissection, 28.9% of those with unilateral CCA dissection and 42.9% of those with bilateral CCA dissection ($p<0.001$).

Overall, 15 patients underwent additional carotid revascularisation. Seven of these patients had preoperative signs of cerebral malperfusion. 11 patients underwent concomitant carotid artery revascularisation, of whom 3 had bilateral carotid artery revascularisation. Two patients underwent stenting of the innominate artery, one patient underwent stenting of the left carotid artery and one patient underwent carotico-subclavian bypass during the index hospitalisation.

Postoperative neurological complications occurred in 252 (22.8%) patients. Ischaemic stroke was detected in 168 (15.2%) patients, haemorrhagic stroke in 41 (3.7%) patients and global brain ischaemia was detected in 65 (5.9%) patients.

Postoperative neurological complications were associated with an increased rate of in-hospital mortality (39.7% vs 14.8%, $p<0.001$) in the overall series as well as in patients with cerebral malperfusion (42.5% vs 20.9%, $p<0.001$) and without cerebral malperfusion (37.7% vs 13.5%, $p<0.001$).

Among patients who underwent concomitant carotid revascularisation, four (26.7%) patients suffered postoperative neurological complications and one (6.7%) patient died during the index hospitalisation.

Table 1 Baseline and operative variables predictive of postoperative neurological complications in univariable analysis

Variables	Overall series N=1106	No neurological complications N=854	Neurological complications N=252	P values
Baseline variables				
Age, years	63.1 (12.7)	62.8 (12.9)	63.9 (11.8)	0.29
Females	348 (31.5)	267 (31.3)	81 (32.1)	0.66
eGFR, mL/min 1.73 m^2	69 (23)	71 (23)	63 (22)	<0.001
Haemoglobin, mg/L	128 (20)	128 (20)	129 (21)	0.50
Bicuspid aortic valve	37 (3.3)	31 (3.6)	6 (2.4)	0.36
Genetic syndrome	21 (1.9)	19 (2.2)	2 (0.8)	0.28
Familial history of aortic dissection/aneurysm	72 (6.5)	59 (6.9)	13 (5.2)	0.33
Prior aortic procedure	12 (1.1)	11 (1.3)	1 (0.4)	0.18
Prior cardiac surgery	28 (2.5)	21 (2.5)	7 (2.8)	0.93
Iatrogenic dissection	28 (2.5)	23 (2.7)	5 (2.0)	0.41
Diabetes	62 (5.6)	49 (5.7)	13 (5.2)	0.62
Prior stroke	47 (4.2)	33 (3.9)	14 (5.6)	0.31
Pulmonary disease	91 (8.2)	72 (8.4)	19 (7.5)	0.85
Extracardiac arteriopathy	38 (3.4)	31 (3.6)	7 (2.8)	0.76
Preoperative malperfusion				
Cerebral malperfusion	254 (23.0)	148 (17.3)	106 (42.1)	<0.001
Unconsciousness	43 (3.9)	24 (2.8)	19 (7.5)	<0.001
Hemiplegia/hemiparesis	107 (9.7)	46 (5.4)	61 (24.2)	<0.001
Dysarthria/aphasia	38 (3.4)	17 (2.0)	21 (8.3)	<0.001
Visual disturbances/visual loss	11 (1.0)	4 (0.5)	7 (2.8)	0.004
Severe confusion	124 (11.2)	79 (9.3)	45 (17.9)	<0.001
Spinal malperfusion	26 (2.4)	20 (2.3)	6 (2.4)	0.51
Renal malperfusion	142 (12.8)	97 (11.4)	45 (17.9)	0.006
Mesenteric malperfusion	76 (6.9)	56 (6.6)	20 (7.9)	0.41
Peripheral malperfusion	169 (15.3)	121 (14.2)	48 (19.0)	0.06
Preoperative cardiac massage	50 (4.5)	36 (4.2)	14 (5.6)	0.52
Cardiogenic shock requiring inotropes	166 (15.0)	113 (13.2)	53 (21.0)	0.002
Invasive mechanical ventilation	154 (13.9)	104 (12.2)	50 (19.8)	<0.001
CT angiography findings				
Dissection of the innominate artery	507 (45.8)	365 (42.7)	142 (56.3)	<0.001
Dissection of the right common carotid artery	302 (27.3)	197 (23.1)	105 (41.7)	<0.001
Dissection of the left common carotid artery	333 (30.1)	226 (26.5)	107 (42.5)	<0.001
No. of dissected carotid arteries				<0.001
None	676 (61.1)	554 (64.9)	122 (48.4)	
Unilateral common carotid artery	225 (20.3)	177 (20.7)	48 (19.0)	
Bilateral common carotid arteries	205 (18.5)	123 (14.4)	82 (32.5)	
Dissection of any common carotid artery	430 (38.9)	300 (35.1)	130 (51.6)	<0.001
Tear involving the aortic arch	204 (18.4)	148 (17.3)	57 (22.2)	0.060
Operative variables				
Salvage procedure	50 (4.5)	36 (4.2)	14 (5.6)	0.57
Arterial cannulation sites				0.18
Direct aortic	159 (14.4)	126 (14.8)	33 (13.1)	
Innominate artery	221 (20.0)	176 (20.6)	45 (17.9)	

Continued

Table 1 Continued

Variables	Overall series N=1106	No neurological complications N=854	Neurological complications N=252	P values
Axillary/subclavian artery	343 (1.0)	255 (29.9)	88 (34.9)	
Femoral artery	334 (30.2)	259 (30.3)	75 (29.8)	
Multiple arteries	44 (4.0)	36 (4.2)	8 (3.2)	
Carotid artery	3 (0.3)	1 (0.1)	2 (0.8)	
Left ventricular apex	1 (0.1)	1 (0.1)	0 (0)	
Unknown site	1 (0.1)	0 (0)	1 (0.4)	
Aortic root replacement	328 (29.7)	269 (31.5)	59 (23.4)	0.028
Partial/total arch replacement	285 (25.8)	212 (24.8)	73 (29.0)	0.008
Total aortic arch replacement	201 (18.2)	150 (17.6)	51 (20.2)	0.06
Coronary artery bypass grafting	83 (7.5)	60 (7.0)	23 (9.1)	0.32
Hypothermic circulatory arrest	915 (82.7)	690 (80.8)	225 (89.3)	0.007
Hypothermic circulatory arrest time, min	42 (27)	42 (27)	43 (29)	0.45
Temperature during hypothermic circulatory arrest, °C	24 (4)	23 (4)	24 (4)	0.62
Antegrade cerebral perfusion	738 (66.7)	553 (64.8)	179 (71.0)	0.06
Antegrade cerebral perfusion time, min	46 (31)	45 (30)	46 (31)	0.71
Bilateral antegrade cerebral perfusion	497 (44.9)	370 (43.3)	127 (50.4)	0.16
Retrograde cerebral perfusion	65 (5.9)	45 (5.3)	20 (7.9)	0.18
Retrograde cerebral perfusion time, min	39 (19)	40 (17)	36 (24)	0.43
Aortic cross-clamping time, min	123 (60)	123 (58)	124 (65)	0.41
Cardiopulmonary bypass time, min	218 (91)	215 (89)	230 (96)	0.032

Continuous values are reported as mean and SD (in parentheses). Categorical variables are reported as counts and percentages (in parentheses). P values refer to the results of univariate analysis. Hypothermic circulatory arrest refers to a period of arrest of systemic circulation with or without the use of antegrade or retrograde cerebral perfusion.
CT, computed tomography; eGFR, estimated glomerular filtration rate.

Patients without CCA dissection, those with unilateral dissection and bilateral dissection of the CCAs had in-hospital mortality of 19.5%, 16.9% (compared with no carotid dissection, adjusted OR 1.006, 95% CI 0.614 to 1.647) and 27.3% (compared with no carotid dissection, adjusted OR 1.719, 95% CI 1.086 to 2.722), respectively. Among hospital survivors, at 10-year, all-cause mortality of patients without CCA dissection, and those with unilateral dissection and bilateral dissection of the CCAs were 36.1%, 38.2% (adjusted HR 1.058, 95% CI, 0.712 to 1.571) and 26.2% (HR 1.027, 95% CI 0.641 to 1.648), respectively.

Patients without CCA dissection, those with unilateral dissection and bilateral dissection of the CCAs, had neurological complications in 18.0%, 21.3% and 40.0% (overall p<0.001; no CCA dissection vs unilateral CCA dissection: p=0.280) of patients, respectively. These figures were 15.1%, 19.4% and 23.9% (p<0.001), respectively, among patients without cerebral malperfusion, and 34.7%, 26.2% and 61.4% (p=0.042), respectively, among those with cerebral malperfusion.

Preoperative estimated glomerular filtration rate (eGFR), invasive mechanical ventilation, cerebral malperfusion and hypothermic circulatory arrest were independent predictors of neurological complications. Partial or total aortic arch repair tended towards a significantly increased risk of neurological complications and, based on the low AIC value of this regression model, was retained in the other regression models (table 2). Dissection of the CCA was associated with increased risk of neurological complications only when involving both CCAs (table 2). Bilateral dissection of the CCAs as a dichotomous variable had an adjusted OR of 2.453 (95% CI 1.683 to 3.576) in predicting postoperative neurological complications. The adjusted risk estimate of patients with bilateral dissection of the CCAs for ischaemic stroke was 1.996 (95% CI 1.267 to 3.147), for haemorrhagic stroke was 1.305 (95% CI 0.616 to 2.767) and for global brain ischaemia was 3.182 (95% CI 1.764 to 5.740).

The negative prognostic effect of bilateral dissection of the CCAs was most evident among patients without cerebral malperfusion who underwent surgery with the use of hypothermic circulatory arrest (28.7% vs 4.3%, p=0.014), while it was not significant among patients with cerebral malperfusion (p=0.807) (figure 1). Neurological complications were rather frequent in patients with cerebral malperfusion associated with bilateral dissection of the CCAs (figure 1). The arterial cannulation site did

Table 2 Variables predictive of postoperative neurological complications in multivariable analysis

Variables	Model A OR, 95% CI	Model B OR, 95% CI	Model C OR, 95% CI
eGFR	0.99, 0.98 to 0.99	0.96, 0.98 to 0.99	0.98, 0.98 to 0.99
Cerebral malperfusion	3.19, 2.29 to 4.45	2.66, 1.88 to 3.75	2.50, 1.76 to 3.55
Invasive mechanical ventilation	2.38, 1.55 to 3.67	2.23, 1.51 to 3.59	2.19, 1.41 to 3.41
Hypothermic circulatory arrest	1.78, 1.07 to 2.96	2.01, 1.23 to 3.29	1.82, 1.01 to 3.04
Partial or total aortic arch repair	1.46, 0.99 to 2.14	1.40, 0.95 to 2.07	1.36, 0.92, 2.01
Any common carotid artery dissection	–	1.60, 1.16 to 2.22	–
No. of dissected common carotid arteries			
None	–	–	Reference
Unilateral common carotid artery	–	–	1.06, 0.70 to 1.59
Bilateral common carotid arteries	–	–	2.57, 1.73 to 3.82
AUC	0.75, 0.71 to 0.78	0.75, 0.72 to 0.78	0.76, 0.72 to 0.79
P value compared with model A's AUC	–	0.381	0.14
AIC	1054	1049	1037

Model A=excluding CT angiographic findings; Model B=including any common carotid artery dissection; Model C=including the number of dissected common carotid arteries.
AIC, Akaike's information criterion; AUC, area under the curve; eGFR, estimated glomerular filtration rate.

not affect the risk of neurological complications in the adjusted model C (direct arterial cannulation: reference; axillary/subclavian artery cannulation p=0.732; innominate artery cannulation p=0.555; femoral artery cannulation p=0.810).

Among patients who underwent surgery with either unilateral or bilateral antegrade cerebral perfusion, unilateral carotid artery dissection was not predictive of neurological complications (adjusted OR 1.264, 95% CI 0.788 to 2.028), while the risk estimate was even higher for bilateral CCA dissection (adjusted OR 2.906, 95% CI 1.767 to 4.780). In this subset of patients, the duration of hypothermic circulatory arrest (adjusted OR 1.002, 95% CI 0.986 to 1.018), temperature of hypothermic circulatory arrest (adjusted OR 0.989, 95% CI 0.928 to 1.054) and duration of antegrade cerebral perfusion (adjusted OR 0.994, 95% CI 0.979 to 1.010) did not have any impact on neurological complications.

In the subset of patients who underwent surgery with bilateral antegrade cerebral perfusion, unilateral CCA dissection tended to be associated with increased risk of neurological complications (adjusted OR 1.744, 95% CI 0.984 to 3.093), while the risk estimate increased even more for bilateral CCA dissection (adjusted OR 3.393, 95% CI 1.820 to 6.327).

DISCUSSION

The present study showed that in patients who underwent surgery for acute DeBakey type 1 aortic dissection: (1) the prevalence of TAAD-related CCA dissection in this series was high; (2) bilateral, but not unilateral, dissection of the CCAs was associated with significantly increased rates of major neurological complications and in-hospital mortality; (3) cerebral malperfusion, invasive mechanical ventilation, decreased eGFR and hypothermic circulatory arrest were independent risk factors of postoperative neurological complications and (4) bilateral CCA dissection was associated with a very high risk of global brain ischaemia.

The present findings are of clinical relevance because they may guide in tailoring the extent of aortic repair and the perfusion strategies. It is unclear whether this subset of patients may benefit from concomitant carotid revascularisation.[3 4 9] We speculate that knowledge of the poor prognosis of patients with bilateral CCA dissection may possibly indicate surgical or endovascular carotid revascularisation as concomitant or tandem salvage procedures. Luehr *et al*[4] and Inoue *et al*[3] proposed early cannulation and perfusion of the dissected carotid arteries in TAAD patients. These authors applied their technique in a limited number of patients with encouraging low rates of severe neurological complications and mortality. Sun *et al*[9] performed extra-anatomic and selective cannulation of dissected CCA with good clinical outcomes as well. Endovascular treatment has also been performed with success to prevent or relieve brain ischaemia in a few patients with TAAD.[10–16] However, data on these treatment strategies are limited, and their potential benefits in reducing postoperative stroke and mortality after surgery for TAAD complicated by CCA dissection should be further investigated.

Here, we observed that bilateral, but not unilateral, CCA dissection had a negative prognostic impact on these TAAD patients in terms of neurological complications and in-hospital mortality. Instead, Kreibich *et al*[2] did not observe an association between TAAD-related

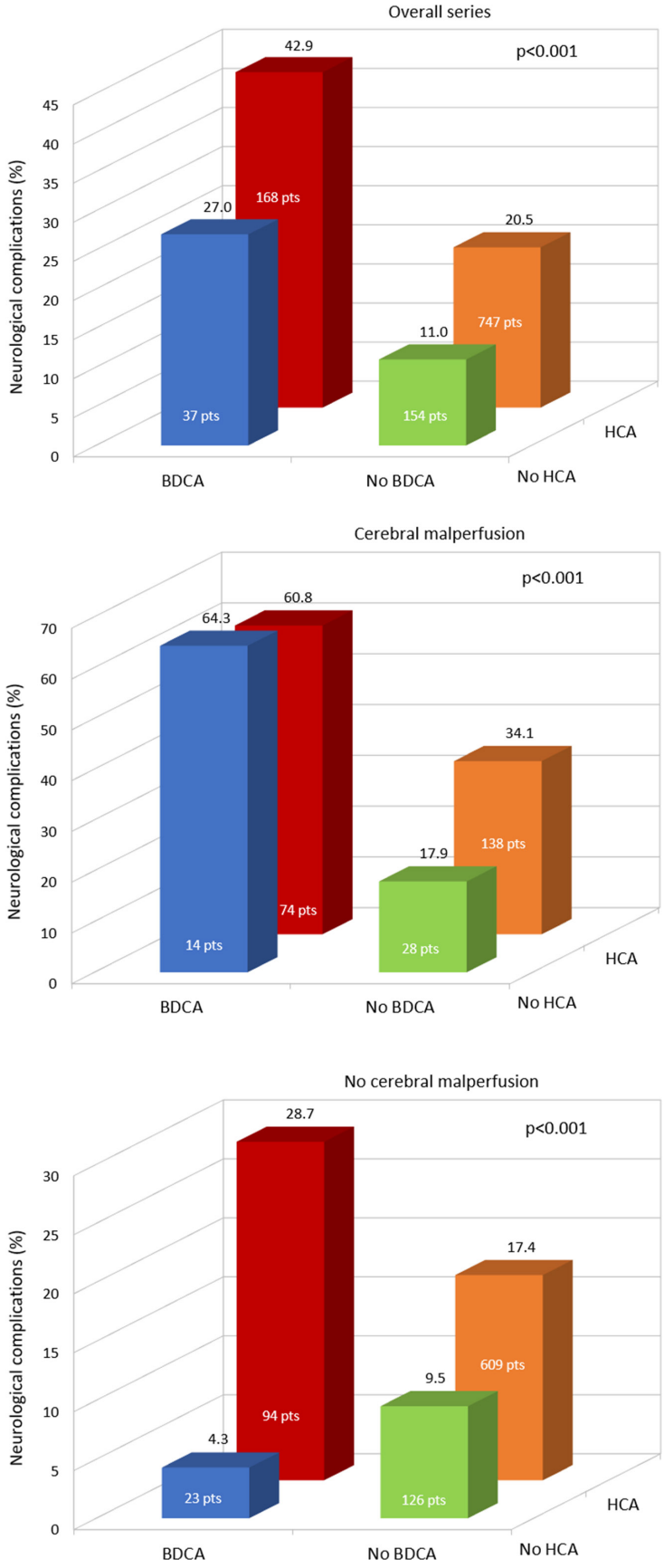

Figure 1 Crude rates of neurological complications in patients with and without bilateral dissection of the common carotid artery (BDCA) and those who underwent surgery with and without the use of hypothermic circulatory arrest (HCA). Analyses were performed in the entire series (p<0.001) and in patients with (p<0.001) and without preoperative cerebral malperfusion (p<0.001).

CCA dissection and operative mortality. Furthermore, they reported that unilateral, but not bilateral, CCA dissection increased the risk of postoperative stroke.[2] We believe that the results of the present study are clinically sound because, once proven that CCA dissection increases the risk of postoperative neurological complications, these complications are known to significantly increase the risk of postoperative mortality after surgery for TAAD patients[6] as well as after other cardiac surgical procedures.[20] Furthermore, the magnitude of the risk of neurological complications is expected to be larger with bilateral than unilateral CCA dissection, considering that the circle of Willis, particularly through its anterior vessels, may suffice to preserve cerebral circulation in case of unilateral carotid artery occlusion.[21]

Preoperative decreased eGFR, cerebral malperfusion, invasive mechanical ventilation and hypothermic circulatory arrest increased the risk of neurological complications. Noteworthy, other cerebral protection strategies were not associated with improved neurological outcome. Patients with chronic kidney disease have generally an increased risk of stroke.[22] In patients with unstable haemodynamic conditions, serum creatinine level may increase within a few hours from the ischaemic insult.[23] Therefore, acutely increased eGFR may be a marker of the severity and extent of aortic dissection. Cerebral malperfusion and the need for invasive mechanical ventilation are risk factors consonant with the increased risk of cerebral ischaemic injury. Patients requiring invasive mechanical ventilation might have had preoperative cardiopulmonary conditions resulting in reduced oxygen delivery and brain ischaemic injury. Such a risk tended to be increased by partial or total repair of the aortic arch (table 2). However, hypothermic circulatory arrest was the only procedural variable which increased the risk of postoperative neurological complications (table 2). The use of hypothermic circulatory arrest was deleterious on neurological outcome in all patients, but in those with bilateral CCA dissection in the presence of cerebral malperfusion (figure 1). This finding may be explained by the severity of cerebral ischaemic injury occurring before surgery associated with bilateral CCA dissection. The negative prognostic effect of the use of hypothermic circulatory arrest was most evident among patients with bilateral CCA dissection without cerebral malperfusion. This subset of patients might have had decreased cerebral blood flow without clinically evident signs of brain ischaemia, which worsened with circulatory arrest. It is worth noting that a relatively large number of patients underwent surgery without the use of hypothermic circulatory arrest (17.3%). This means that surgeons are aware that adding a period of cerebral brain ischaemia may result in or aggravate any preoperative subclinical or clinically evident brain ischaemic injury. Therefore, several surgeons might have chosen to avoid hypothermic circulatory arrest in these patients.

The retrospective nature of the ERTAAD is the main limitation of this analysis. Second, CTA findings were not evaluated by an imaging core lab and therefore the status of the CCAs could be biased by interobserver variability. However, a retrospective review of the CTA scans would have been expensive and might not have been possible for those exams performed during the first years of the study period. Third, the severity and extent of carotid artery dissection, as well as the status of the subclavian arteries and vertebral arteries were not evaluated in this study. Therefore, we do not have complete information on the possible dissection of all cervical arteries. Fourth, only 13 (1.2%) patients underwent any concomitant or tandem treatment of the CCA dissection. Therefore, the possible therapeutic effect of carotid revascularisation in this setting cannot be addressed in this series. Fifth, we do not have data on the severity of impairment related to postoperative neurological complications. Sixth, we do not have information on the methods of follow-up employed at each centre. Indeed, the method of follow-up may markedly vary according to different countries' regulation and the availability of data from national registries collecting data on mortality and causes of hospital readmissions. Finally, the mean delay from the onset of symptoms to surgery was 18 hours (median 8 hours). This means that CTA was performed shortly before surgery. However, we cannot rule out that in a few patients, epiaortic vessels might have been involved by aortic dissection shortly after CTA was performed.

This study has a few strengths which deserve to be acknowledged. Although data on preoperative CTA were available in 35% of patients with DeBakey type 1 aortic dissection, the sample size of this study is rather large. The multicentre nature of this study with interinstitutional variability of referral pathways, surgical and perfusion strategies as well as perioperative care makes the research results generalisable to most contexts.

CONCLUSIONS

In the present study, the involvement of the carotid arteries by DeBakey type 1 aortic dissection increased the risks of major neurological complications and in-hospital mortality. However, such risks were limited to patients with bilateral dissection of the CCAs. The use of hypothermic circulatory arrest in these patients without cerebral malperfusion might have contributed to an increase in the rate of neurological complications. These findings suggest that surgical and perfusion strategies for aortic repair could be possibly tailored based on the status of the carotid arteries to reduce the very high rate of postoperative neurological complications in patients with DeBakey type 1 aortic dissection. The present findings should be considered hypothesis-generating and should be confirmed by further studies also evaluating the preoperative status of the subclavian arteries and vertebral arteries. Considering the relatively high prevalence and negative prognostic impact of CCA dissection in TAAD patients, the possible benefits and harms of concomitant

surgical or endovascular treatment of carotid artery dissection should be investigated.

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