Early and late outcomes of type A acute aortic dissection with common carotid artery involvement

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ABSTRACT

Objective: The relationship between common carotid artery (CCA) involvement in acute type A aortic dissection (ATAAD) and postoperative outcomes remains unclear. We investigated outcomes and described our current advanced strategy.

Methods: Of 492 patients who underwent surgical repair for ATAAD between September 1999 and February 2021, CCA involvement was identified in 114. Eighty of these 114 patients (70.2%) were classified as having a thrombosed CCA and 34 (29.8%) were classified as nonthrombosed. To prevent postoperative cerebral malperfusion, we initiated a strategy of early reperfusion and direct reconstruction of dissected and thrombosed CCAs regardless of neurologic symptoms.

Results: Fifty-five patients (48.2%) showed preoperative neurologic symptoms. No significant differences between the thrombosed and nonthrombosed groups were seen in postoperative mortality (20.0% vs 11.8%; $P = .421$) or frequency of postoperative modified Rankin scale (mRS) score ≥ 5 (30.0% vs 17.6%; $P = .245$). The rate of postoperative neurologic deficit was significantly higher (48.8% vs 23.5%; $P = .013$) and long-term survival rate was significantly lower (5 years; 59.1 ± 6.3% vs 77.9 ± 7.4%; 10 years: 50.0 ± 7.9% vs 72.3 ± 8.7%; $P = .041$) in the thrombosed group. Risk factors for mRS ≥ 5 from multivariable analysis included occluded thrombosed CCA, preoperative coma, preoperative shock, and prolonged operation time. Fifteen patients were treated with the early reperfusion and direct reconstruction strategy; postoperative mortality 13.3% (2 patients). No patients showed cerebral reperfusion syndrome.

Conclusions: In patients with CCA involvement, a thrombosed false lumen, especially an occluded CCA, resulted in worse outcomes regardless of preoperative neurologic symptoms. Further study is needed to evaluate the efficacy of the current strategy. (JTCVS Open 2022;10:1-11)

A thrombosed false lumen results in worse outcomes.
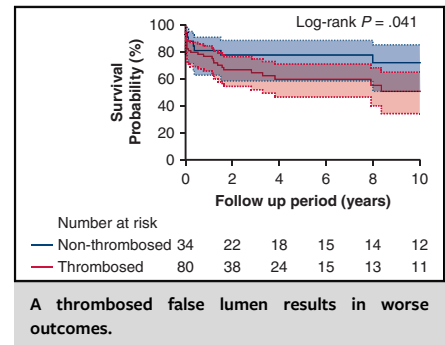
CENTRAL MESSAGE

A thrombosed false lumen of the common carotid artery (CCA), particularly an occluded CCA, results not only in worse neurologic outcomes, but also in worse long-term outcomes.

PERSPECTIVE

A thrombosed false lumen of the common carotid artery (CCA), particularly an occluded CCA, results not only in worse neurologic outcomes, but also in worse long-term outcomes. To prevent postoperative stroke with a thromboembolic etiology, a strategy of early perfusion and direct reconstruction of the CCA might provide better outcomes for patients with thrombosed CCA involvement.

▶ Video clip is available online.

Surgical outcomes for acute type A aortic dissection (ATAAD) have been improving over the last decade,[1,2] but the surgical outcomes of ATAAD complicated by cerebral malperfusion remain suboptimal. In previous studies, 8%-18% of patients with ATAAD had preoperative cerebral malperfusion, and in-hospital mortality rates after surgery were quite high, at 26%-56%.[3-8]

We have previously reported on surgical strategies. The conventional strategy reported in 2005[3] is straightforward immediate surgery to minimize the interval between onset

Abbreviations and Acronyms
ATAAD = acute type A aortic dissection
BSS = brain-saving system
CCA = common carotid artery
CT = computed tomography
ICA = internal carotid artery
IQR = interquartile range
mRS = modified Rankin scale
PND = persistent neurologic deficit
TND = transient neurologic deficit

time and a central repair. Another strategy, reported in 2009,[9,10] involved earlier brain reperfusion using a brain-saving system (BSS) comprising a bypass circuit between the common femoral artery and the occluded common carotid artery (CCA) in the emergency room before transfer to the operating room. Although we believe that these strategies focusing on limiting the cerebral ischemic insult reflect highly important concepts, we recognize that this concept alone is insufficient to decrease neurologic complications in the overall surgical management of ATAAD, regardless of preoperative neurologic symptoms. A dissected CCA potentially causes not only preoperative cerebral malperfusion, but also intraoperative new-onset and deteriorating cerebral malperfusion during cardiopulmonary bypass. The association between dissected CCA and postoperative neurologic outcomes is a crucial issue that has been explored in several recent studies,[11-13] and various forms of surgical management of the dissected CCA in addition to the tear-oriented strategy have been advocated to decrease neurologic complications.[13,14]

The main aim of the present study was to examine the outcomes of ATAAD with CCA involvement in our institution and to provide a supplementary report on our current strategy, focusing on providing further insight into dissected CCA.

METHODS

Between October 1999 and February 2021, a total of 492 consecutive patients with ATAAD underwent emergent aortic repair at our institution. Of these patients, 411 (83.5%) underwent preoperative CCA evaluation, and 114 showed CCA involvement. All CCA involvements were detected by preoperative computed tomography (CT) or carotid duplex scan. We classified patients into a thrombosed false lumen group (n = 80; 70.2%) and a nonthrombosed false lumen group (n = 34 patients; 29.8%). The thrombosed group was subdivided into 3 types according to the ratio of the diameters of the false lumen and the CCA: occluded (>99%), severe stenosis (70%-99%), and mild stenosis (<70%) (Figure 1).[15] Thrombosed CCA was defined as the presence of a low-density area of the false lumen on contrast-enhanced CT in the late phase. If preoperative contrast-enhanced CT was unavailable, then the presence of a high-density area on plain CT or a lack of blood flow on carotid duplex scan was used to define thrombosed CCA. Ninety-seven patients (85.1%) were evaluated by contrast-enhanced

Occluded (>99%) n=31   Severe stenosis (70-99%) n=30   Mild stenosis (<70%) n=19   Non-thrombosed n=34
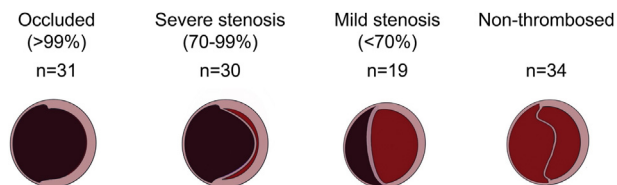
FIGURE 1. Details and numbers of cases classified by severity of common carotid artery (CCA) stenosis. Cases involving a thrombosed CCA were divided into 3 subgroups by severity.

CT, 9 patients (7.9%) by plain CT, and 8 (7.0%) by carotid duplex scan. The diameter of the CCA was measured to evaluate the severity of CCA stenosis. We measured the severity using the diameters of the CCA and the true lumen. One hundred and one patients (88.6%) were postoperatively evaluated for CCA dissection by contrast-enhanced CT. As some patients presented with bilateral CCA dissections, we examined a total of 122 preoperatively dissected CCAs (thrombosed, n = 72 [59.0%]; nonthrombosed, n = 50 [41.0%]).

Early and long-term outcomes were compared among 4 groups (3 subgroups of the thrombosed group plus the nonthrombosed group). Each neurologic outcome was evaluated using the modified Rankin scale (mRS). An mRS score ≥5 reflected a bedridden condition.

Data were extracted from the medical record of patients who were followed up in our outpatient clinic. For patients who did not undergo follow-up in the outpatient setting, data were collected via telephone follow-up.

This study was approved by the Institutional Review Board at Kobe University Hospital (approval B190201; approved on October 8, 2019), and the need to obtain individual consent was waived.

Definition of Malperfusion

Malperfusion syndrome was defined as organ ischemia secondary to arterial obstruction caused by aortic dissection.[5,16] Persistent neurologic deficit (PND) was defined as symptoms persisting before the operation (preoperative PND) or at hospital discharge (postoperative PND). Transient neurologic deficit (TND) was defined as any neurologic dysfunction that disappeared before the operation (preoperative TND) or by hospital discharge (postoperative TND). New-onset or deteriorated neurologic deficit was defined as a deterioration in the manual muscle test grading scale from 3 to 5 (normal to fair) to 0 to 2 (poor to trace) or the emergence of previously undocumented neurologic symptoms, including coma.

Surgical Strategy

In 2018, we initiated the current strategy of providing direct reperfusion and direct reconstruction before performing central repair for all ATAAD patients with CCA involvement with or without preoperative neurologic symptoms. In this strategy, we follow the method of early direct reperfusion and direct reconstruction described by Gomibuchi and colleagues.[13] The dissected CCA is exposed through an oblique neck incision anterior to the sternocleidomastoid muscle at the same time as the sternotomy for a patient with a thrombosed CCA. Cardiopulmonary bypass is established first. The arterial cannulation site was the ascending aorta or femoral artery. During systemic cooling, the dissected CCA is carefully clamped and transected, as much thrombus in the false lumen is removed as possible, and a 12 Fr SCP cannula is inserted directly into the true lumen of the dissected CCA and reconstructed CCA using an 8-mm Propaten vascular graft (W. L. Gore & Associates). Antegrade cerebral perfusion is initiated at a flow rate of 1 to 2 mL/kg/minute and gradually increased to a maintenance flow rate of 3 to 5 ml/kg/minute or 50 mm Hg of the flow pressure. If regional oxygen

saturation (rSO_2) becomes elevated due to reperfusion, the maintenance flow rate is adjusted to low as possible to prevent reperfusion syndrome. After tympanic and rectal temperatures drop to 23 °C and 30 °C, respectively, bilateral antegrade selective cerebral perfusion is applied. After central repair, the graft is anastomosed to one of the branches of the main graft (Figure 2, Video 1). The details of management for central repair have been described in a previous report.[17] As in the current strategy, early direct reperfusion and direct reconstruction a performed using an 8-mm Propaten graft before the initiation of central repair.

Statistical Analysis

Data were processed using R version 3.5.0 (R Foundation for Statistical Computing) and Prism version 9.0.1 (GraphPad Software). All continuous variables were analyzed using the *t* test or Mann–Whitney *U* test and are expressed as mean ± standard deviation or median and interquartile range (IQR). The Shapiro–Wilk test was performed for all continuous variables to check for normal distribution. Categorical variables were analyzed using the χ^2 test or Fisher exact test and expressed as number and percentage of patients. Overall survival rates were calculated using Kaplan–Meier methods and are expressed as rate and 95% confidence interval. Risk factors for mRS ≥5 were identified using logistic regression analysis. Clinically relevant variables[16,18,19] with values of $P < .05$ in univariable analyses were incorporated into the multivariable model. The subsequent multivariable analysis was performed using the forced entry method. Linear trends were assessed by the Cochran–Armitage test for outcomes classified by the severity of CCA stenosis. $P < .05$ was considered to indicate statistical significance. Multiple-comparison corrections were not applied.

RESULTS

Preoperative Variables

Preoperative variables of each group are shown in Table 1. The mean age of the entire cohort was 66.6 ± 12.3 years. Fifteen patients (13.2%) were octogenarians. Preoperative symptomatic neurologic deficit was present in 48.2% of all patients, in 52.5% of the thrombosed group, and in 38.2% of the nonthrombosed group, with coma observed in 14.0%, 17.5% and 5.9%,
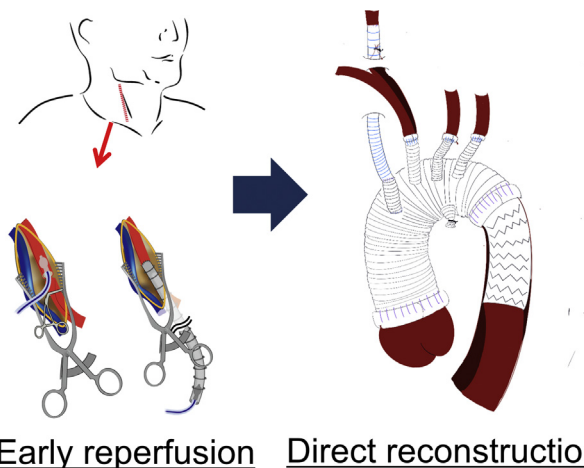
FIGURE 2. Scheme for the current strategy of early reperfusion and direct reconstruction. After the initiation of cardiopulmonary bypass, direct cannulation and direct reconstruction of the dissected common carotid artery were performed before central aortic repair.
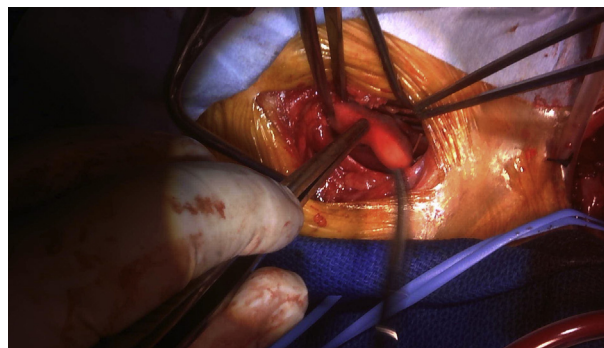
VIDEO 1. After the direct exposure of the common carotid artery (CCA), the dissected CCA is transected, a 12 Fr SCP cannula is inserted directly into the true lumen of the dissected CCA, and the CCA is reconstructed using 8-mm Propaten. Video available at: https://www.jtcvs.org/article/S2666-2736(22)00072-9/fulltext.

respectively. Malperfusion of the extremities was less frequent in the thrombosed group compared with the nonthrombosed group (10.0% vs 26.5%; $P = .041$). No other preoperative variables differed significantly between the 2 groups. Preoperative risk, as calculated by the German Registry for Acute Aortic Dissection Type A (GERAADA) score[20] and Japan score,[21] did not differ significantly between the groups.

Perioperative Variables

Table 2 presents perioperative data. The thrombosed group more frequently underwent reperfusion of the dissected CCA before central repair (26.2% vs 8.8%; $P = .045$) and showed significantly longer cerebral perfusion time (120 minutes [IQR, 64 to 156 minutes] vs 86 minutes [IQR, 33 to 105 minutes]; $P = .007$). The current strategy was used in 16.2% of patients in the thrombosed group and in 5.9% of patients of the nonthrombosed group ($P = .224$). No significant between-group differences were seen in operation time, cardiopulmonary bypass time, myocardial ischemic time, other concomitant procedures, or arterial cannulation sites. The in-hospital mortality rate of patients with CCA involvement was 17.5%. There were no significant differences in postoperative mortality rate (20.0% vs 11.8%; $P = .421$) or postoperative mRS ≥5 (30.0% vs 17.6%; $P = .245$) between the thrombosed and nonthrombosed groups (Table 2). In the thrombosed group, postoperative neurologic deficits, including TND, were observed more frequently (48.8% vs 23.5%; $P = .013$), and the frequency of an intensive care unit stay of ≥7 days was significantly higher (48.8% vs 11.8%; $P < .001$). No significant difference in new-onset or deteriorated postoperative neurologic deficits was observed between the thrombosed and nonthrombosed groups (20.0% vs 8.8%; $P = .177$). Eleven patients (13.8%) in the thrombosed group and 2 patients (5.9%) in the nonthrombosed group

TABLE 1. Preoperative variables

Variable	CCA involvement (N = 114)	Thrombosed (N = 80)	Nonthrombosed (N = 34)	P value
Age, y, mean ± SD	66.6 ± 12.3	66.5 ± 11.4	66.9 ± 14.5	.865
Octogenarians, n (%)	15 (13.2)	10 (12.5)	5 (14.7)	.767
Male sex, n (%)	63 (55.3)	40 (50.0)	23 (67.6)	.127
Body surface area, cm^2, mean ± SD	1.68 ± 0.21	1.70 ± 0.21	1.71 ± 0.19	.321
Body mass index, kg/m^2, mean ± SD	23.0 ± 4.22	23.2 ± 4.9	22.6 ± 2.9	.550
Preoperative status, n (%)				
Shock	17 (14.9)	12 (15.0)	5 (14.7)	>.999
Cardiopulmonary resuscitation	8 (7.0)	5 (6.2)	3 (8.8)	.694
Organ malperfusion, n (%)				
Central nervous system	55 (48.2)	42 (52.5)	13 (38.2)	.234
Transient	20 (17.5)	16 (20.0)	4 (11.8)	.421
Persistent	35 (30.7)	27 (33.8)	8 (23.5)	.375
Coma (GCS ≤8), n (%)	16 (14.0)	14 (17.5)	2 (5.9)	.143
Hemiplegia, n (%)	16 (14.0)	11 (13.8)	5 (14.7)	>.999
Coronary, n (%)	19 (16.7)	13 (16.2)	6 (17.6)	>.999
Visceral, n (%)	9 (7.9)	7 (8.8)	2 (5.9)	.723
Extremities, n (%)	17 (14.9)	8 (10.0)	9 (26.5)	.041
Two territories, n (%)	21 (18.4)	15 (18.8)	6 (17.6)	>.999
Preoperative brain ischemic time, n (%)				
<4.5 h	18 (15.8)	14 (17.5)	4 (11.8)	.579
<6.0 h	44 (38.6)	33 (41.2)	11 (32.4)	.495
Aortic valve insufficiency ≥ moderate, n (%)	22 (19.3)	14 (17.5)	8 (23.5)	.449
Chronic kidney disease, n (%)				
eGFR <30 mL/min/1.73 m^2	9 (7.9)	7 (8.8)	2 (5.9)	.723
Distal extent of aortic dissection, n (%)				
Arch	14 (12.3)	11 (13.8)	3 (8.8)	.549
Descending	8 (7.0)	5 (6.2)	3 (8.8)	.694
Thoracoabdominal	7 (6.1)	5 (6.2)	2 (5.9)	>.999
Abdominal or beyond abdominal	85 (74.6)	59 (73.8)	26 (76.5)	.818
Entry site, n (%)				
Aortic root–sinotubular junction	1 (0.9)	0 (0.0)	1 (2.9)	.298
Ascending	63 (55.3)	42 (52.5)	21 (61.8)	.481
Ascending–aortic arch	23 (20.2)	17 (21.2)	6 (17.6)	.801
Distal arch	23 (20.2)	19 (24.1)	4 (11.8)	.203
Unknown	4 (3.5)	2 (2.5)	2 (5.9)	.581
CCA dissections, n (%)				
Right	102 (89.5)	71 (88.8)	31 (91.2)	>.999
Left	35 (30.7)	25 (31.2)	10 (29.4)	>.999
Bilateral	23 (20.2)	16 (20.0)	7 (20.6)	>.999
GERAADA score, median (IQR)	17.4 (12.9-23.5)	17.5 (13.0-23.0)	16.9 (12.6-25.6)	.790
Japan score, 30-d mortality, median (IQR)	8.9 (7.2-12.7)	8.9 (7.6-13.8)	8.4 (5.6-11.7)	.215
Surgical era, n (%)				
1999-2005	17 (14.9)	11 (13.8)	6 (17.6)	
2006-2021	97 (85.1)	69 (86.2)	28 (82.4)	.578

CCA, Common carotid artery; *SD*, standard deviation; *GCS*, Glasgow Coma Scale; *eGFR*, estimated glomerular filtration rate; *GERAADA*, German Registry for Acute Aortic Dissection Type A; *IQR*, interquartile range.

developed cerebral reperfusion injuries, which we defined as cerebral herniation, hemorrhage, or edema requiring decompressive craniotomy ($P = .338$). Postoperative CT revealed residual dissection in 80 of 122 patients (65.6%) with dissected CCA preoperatively (Table E1). With the current strategy, 2 patients in thrombosed group showed residual CCA dissection, but these dissections were limited to just distal to anastomosis sites.

TABLE 2. Perioperative variables

Variable	CCA involvement (N = 114)	Thrombosed (N = 80)	Nonthrombosed (N = 34)	P value
Operation time, min, median (IQR)	418 (341-496)	418 (344-504)	418 (341-478)	.599
Cardiopulmonary bypass duration, min, median (IQR)	212 (174-252)	211 (177-257)	216 (159-243)	.292
Cardiac ischemic time, min, median (IQR)	113 (88-138)	113 (89-140)	116 (88-137)	.963
Lower body circulatory arrest, min, median (IQR)	46 (38-56)	46 (40-56)	45 (34-54)	.097
Antegrade cerebral perfusion, n (%)	104 (91.2)	75 (93.8)	29 (85.3)	.161
Antegrade cerebral perfusion duration, min, median (IQR)	105 (39-151)	120 (64-156)	86 (33-105)	.007
CCA early perfusion, n (%)	24 (21.1)	21 (26.2)	3 (8.8)	.045
Current strategy, n (%)	15 (13.2)	13 (16.2)	2 (5.9)	.224
Concomitant surgery, n (%)	21 (18.4)	14 (17.5)	7 (20.6)	.793
Aortic root repair/replacement, n (%)	8 (7.0)	4 (5.0)	4 (11.8)	.236
CABG, n (%)	11 (9.6)	9 (11.2)	2 (5.9)	.501
Distal anastomosis of aorta, n (%)				
Zone 0	40 (35.1)	24 (30.0)	16 (47.1)	.126
Zone I	7 (6.1)	6 (7.5)	1 (2.9)	.672
Zone II	6 (5.3)	6 (7.5)	0 (0)	.176
Zone III	61 (53.5)	44 (55.0)	17 (50.0)	.776
Arterial cannulation site, n (%)				
Ascending aorta	25 (22.0)	20 (25.0)	5 (14.7)	.323
Femoral artery	80 (70.2)	55 (68.8)	25 (73.5)	.661
Axillary artery	10 (8.8)	5 (6.2)	5 (14.7)	.161
Additional cannulations	18 (15.8)	14 (17.5)	4 (11.8)	.579
Early outcomes, n (%)				
30-d mortality	16 (14.0)	13 (16.2)	3 (8.8)	.386
Hospital mortality	20 (17.5)	16 (20.0)	4 (11.8)	.421
Postoperative neurologic deficit	47 (41.2)	39 (48.8)	8 (23.5)	.013
Transient neurologic deficit	6 (5.3)	6 (7.5)	0 (0)	.176
Persistent neurologic deficit	41 (36.0)	33 (41.3)	8 (23.5)	.089
Coma	15 (13.2)	13 (16.3)	2 (5.9)	.224
Hemiplegia	22 (19.3)	17 (21.3)	5 (14.7)	.604
mRS score ≥5	30 (26.3)	24 (30.0)	6 (17.6)	.245
New-onset/deteriorated neurologic deficit	19 (16.7)	16 (20.0)	3 (8.8)	.177
Cerebral reperfusion syndrome*	13 (11.4)	14 (17.5)	2 (5.9)	.338
ICU stay ≥7 d	43 (37.7)	39 (48.8)	4 (11.8)	<.001
Duration of ventilation ≥48 h	53 (46.5)	41 (51.2)	12 (35.3)	.175
Hemodialysis requirement	13 (11.4)	10 (12.5)	3 (8.8)	.752
Mediastinitis	5 (4.4)	2 (2.5)	3 (8.8)	.156
Tracheostomy	15 (13.2)	11 (13.8)	4 (11.8)	>.999

CCA, Common carotid artery; *IQR*, interquartile range; *CABG*, coronary artery bypass grafting; *mRS*, modified Rankin Scale; *ICU*, intensive care unit. *Cerebral reperfusion syndrome includes cerebral hemorrhage, cerebral herniation, or cerebral edema with decompressive craniotomy postoperatively.

Outcomes by Severity of CCA Stenosis

Table 3 shows the surgical outcomes in each group categorized by the severity of CCA stenosis. Significant trends in postoperative outcomes, including in-hospital death (*P* = .014), mRS ≥5 (*P* = .022), postoperative neurologic deficit (*P* = .002), postoperative coma (*P* = .004), and 5-year mortality (*P* = .036) were observed with increasing severity of CCA stenosis.

Risk Factor Analysis of mRS ≥5

Univariable logistic regression analysis for patients with CCA involvement identified occluded CCA, octogenarian age group, preoperative coma, preoperative shock, and prolonged operation time as risk factors for mRS ≥5. Multivariable analysis identified occluded CCA, preoperative coma, preoperative shock, and prolonged operation time as risk factors for mRS ≥5 (Table 4).

Long-Term Outcome

The complete follow-up rate was 93.9%, and the median duration of follow-up was 15.6 months (range, 3 to 81 months). The 5-year and 10-year survival rates for the total cohort were 65.2 ± 4.9% and 57.8 ± 5.9%, respectively. Overall survival rates as assessed by the log-rank test were significantly lower in the thrombosed group (5 years: 59.1 ± 6.3% vs 77.9 ± 7.4%; 10 years: 50.0 ± 7.9% vs 72.3 ± 8.7%; P = .041) (Figure 3).

Outcomes of the Current Strategy

The current early reperfusion and direct reconstruction strategy was used in 15 consecutive patients with a mean age of 71.5 years (Table E2). Seven patients (46.7%) showed occlusion of the thrombosed CCA, and 6 (40.0%) had preoperative hemiplegia or coma. With this strategy, in-hospital mortality was observed in 2 patients (13.3%), and 3 patients (20%) had a postoperative mRS ≥5. No patient experienced coma, cerebral reperfusion syndrome, or 30-day mortality. The causes of death were visceral malperfusion in 1 patient and mediastinitis in 1 patient, with no instances of critical reperfusion syndrome, such as cerebral infarction, hemorrhage, or cerebral herniation, resulting in cerebral death. All patients with preoperative coma improved to the point of being able to communicate. Only 1 patient (6.7%) had new onset of neurologic symptoms postoperatively. That patient (case 12) also had coronary malperfusion and received postoperative extracorporeal membrane oxygenation support for several days but was discharged home with an mRS of 2.

DISCUSSION

In this study comparing outcomes of ATAAD in patients with thrombosed dissected CCAs and nonthrombosed dissected CCAs, the 2 patient groups had a similar preoperative status, but postoperative neurologic deficits were significantly more frequent in the thrombosed group, and these patients had significantly worse long-term outcomes. In addition, patients with an occluded thrombosed CCA showed more severe neurologic outcomes with mRS scores ≥5. To improve the neurologic outcomes, we applied the early perfusion and direct reconstruction strategy

(Figure 4). With the current strategy, no patient experienced cerebral reperfusion syndrome, coma, or 30-day mortality.

Cerebral malperfusion is a devastating complication in ATAAD. Several studies using national registry data have shown that 8% to 18% of patients experienced preoperative symptomatic cerebral malperfusion in ATAAD.[1,5,8] Postoperative cerebral malperfusion was present in 17% to 69% of patients with preoperative cerebral malperfusion, compared with 4% to 15% of those with no preoperative cerebral malperfusion.[3,5,6,8,22-24] Postoperative stroke was associated with significant morbidities[25] and also was a risk factor for long-term mortality, as reported by Chemtob and colleagues.[6] In the present study, the thrombosed group had more postoperative neurologic deficits and worse long-term mortality. Furthermore, no patients with mRS ≥5 were discharged to home. This result may suggest that severity is also a significant contributor to long-term quality of life.

Reduced blood flow owing to a compressed true lumen and thromboembolism from the false lumen are potential etiologies for cerebral malperfusion in ATAAD.[26,27] The impact of blood flow reduction, along with the need to initiate early reperfusion by central repair or early direct cannulation, are topics of extensive debate. Both Estrera and colleagues[28] and Tsukube and colleagues[29] identified prolonged preoperative cerebral ischemic time as a major contributor to worse outcomes. Although a central repair-first strategy was previously selected for patients with cerebral malperfusion, accumulation of suboptimal outcomes propelled us to develop the BSS specifically for symptomatic patients with CCA involvement to reduce the cerebral ischemic injury induced by low perfusion.[9,10]

Urbanski and colleagues[30] and Uchida and colleagues[31] reported the efficacy of direct early reperfusion of the carotid artery in symptomatic patients with cerebral malperfusion. Earlier reperfusion by BSS has reduced the incidence of in-hospital mortality from 56% to 33%.[3,10] On the other hand, Chiu and colleagues[19] reported that time to operation was not predictive of outcomes. That study suggested that early perfusion to relieve the reduced blood flow alone did not improve outcomes. The present study also revealed that a preoperative cerebral ischemic time <4.5 hours was not predictive of mortality or neurologic outcomes,

TABLE 3. Outcomes classified by severity of CCA stenosis

Outcomes	Occluded (N = 31)	Severe stenosis (N = 30)	Mild stenosis (N = 19)	Nonthrombosed (N = 34)	P value
In-hospital death	11 (35.4)	4 (13.3)	1 (5.3)	4 (11.8)	.014
mRS ≥5	14 (45.2)	6 (20.0)	4 (21.1)	6 (17.6)	.022
Postoperative neurologic deficit	20 (64.5)	11 (36.7)	8 (42.1)	8 (23.5)	.002
Postoperative coma	10 (32.3)	2 (6.7)	1 (5.3)	2 (5.9)	.004
Deteriorated neurologic deficit	6 (19.4)	6 (20.0)	4 (21.1)	3 (8.8)	.256
5-y death	15 (48.4)	7 (23.3)	6 (31.6)	7 (20.6)	.036

CCA, Common carotid artery; *mRS*, modified Rankin Scale.

TABLE 4. Risk factors for postoperative mRS ≥5

Risk factors	Univariable analysis			Multivariable analysis		
	OR	95% CI	*P* value	OR	95% CI	*P* value
CCA occlusion	3.450	1.41-8.42	.007	3.100	1.04-9.22	.042
Thrombosed CCA	2.000	0.73-5.45	.176			
Current strategy	0.667	0.18-2.55	.553			
Octogenarian	4.000	1.31-12.30	.015	3.280	0.88-12.30	.078
eGFR <30 mL/min/1.73 m^2	0.786	0.15-4.01	.772			
Brain ischemic time <4.5 h	0.769	0.23-2.55	.668			
Bilateral CCA involvement	0.526	0.16-1.70	.282			
Preoperative coma	6.500	2.11-20.0	.001	3.810	1.00-14.50	.050
Preoperative hemiplegia	1.090	0.35-3.37	.878			
Coronary malperfusion	1.830	0.64-5.19	.258			
Preoperative shock	5.500	1.86-16.3	.002	4.180	1.16-15.00	.028
Visceral malperfusion	2.430	0.61-9.73	.210			
Concomitant operation	1.520	0.55-4.23	.421			
Central cannulation	0.462	0.14-1.48	.192			
Operation time, min	1.000	1.00-1.01	.002	1.000	1.00-1.01	.013

OR, Odds ratio; *CI*, confidence interval; *CCA*, common carotid artery; *eGFR*, estimated glomerular filtration rate.

suggesting that other causes of postoperative stroke should be taken into consideration.

We sometimes encountered new-onset stroke postoperatively. We detected obviously communicating reentry distal to the thrombosed false lumen in some symptomatic patients (Figure E1). Therefore, we hypothesized that the mechanism in these cases was thromboembolism from the false lumen, as discussed by Norton and colleagues.[26] In fact, in our study, patients with involvement of a thrombosed CCA had a significantly higher rate of long-term mortality, and more severe stenosis of the CCA was associated with significantly worse neurologic outcomes than nonthrombosed CCA involvement. Interestingly, the

FIGURE 3. Kaplan-Meier curves comparing survival in the thrombosed common carotid artery (CCA) and nonthrombosed CCA groups. The 95% confidence intervals are shown in parentheses. Long-term mortality was significantly higher in the thrombosed CCA group compared with the nonthrombosed group.

incidence of postoperative neurologic deteriorations showed little correlation with the severity of CCA stenosis (Table 3). This result also may indicate that not only reduced blood flow, but also thromboembolism is the cause of postoperative neurologic complications. The present study failed to show any significant association of thrombosed CCA with neurologic deterioration (thrombosed type vs nonthrombosed type: 20.0% vs 8.8%; *P* = .177), postoperative mortality (20.0% vs 11.8%; *P* = .421) and mRS ≥5 (30.0% vs 17.6%; *P* = .245). However, we believe that the incidence rates of these outcomes in the thrombosed group were double those in the nonthrombosed group, and that there were clinically significant differences between the 2 groups. This result may be due to the fact that the severity of CCA lesions and internal carotid artery (ICA) lesions were not fully reflected in the comparisons between the thrombosed and nonthrombosed groups. In fact, we showed relationships between severity of thrombosed CCA and outcomes, including hospital mortality rate, postoperative neurologic symptoms, and postoperative mRS ≥5 (Table 3). On the other hand, only 19 patients (23.8%) in the thrombosed group and 4 patients (11.8%) in the nonthrombosed group were evaluated for preoperative ICA, whereas 9 patients (47.4%) in the thrombosed group and 1 patient (25.0%) in the nonthrombosed group had ICA dissection (*P* = .604). Further studies are warranted to confirm and explore the associations between a thrombosed false lumen and these outcomes.

Gomibuchi and Okada[13] also reported that occlusion or severe stenosis of supra-aortic branch vessels was associated with postoperative PND regardless of preoperative
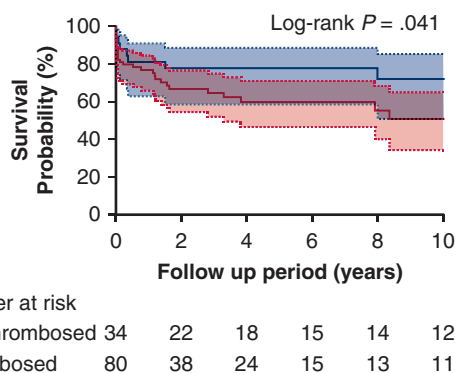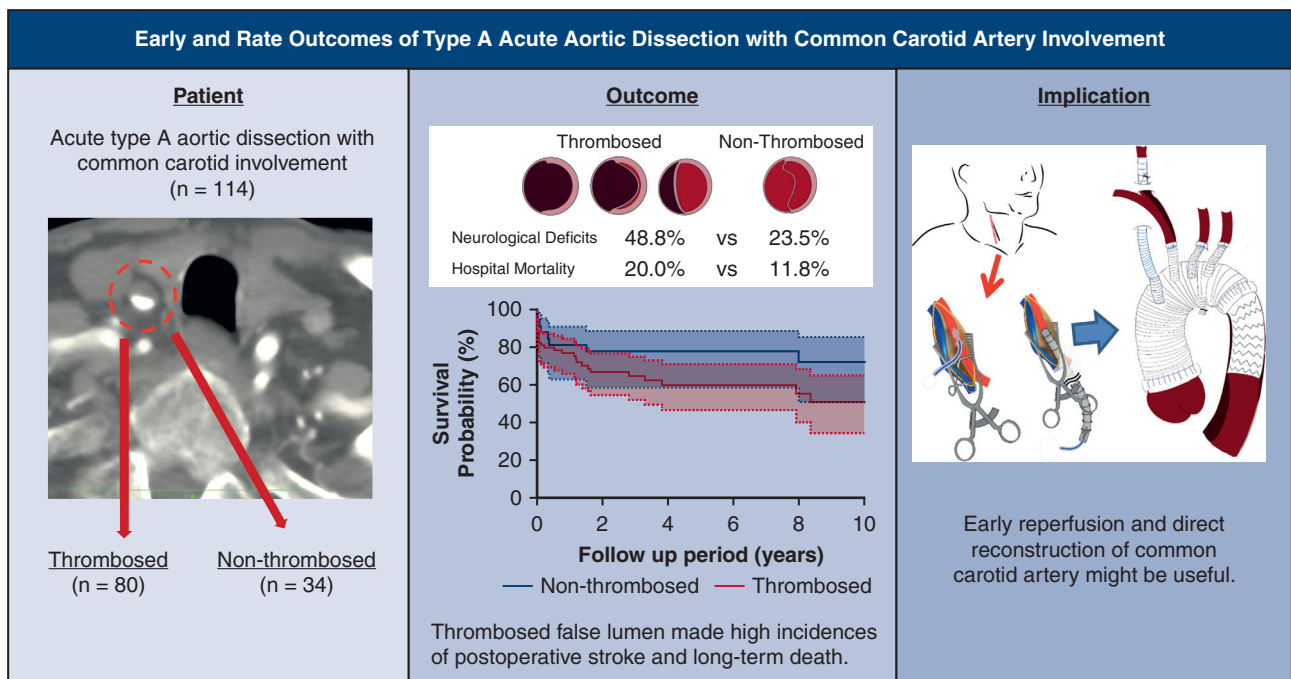
FIGURE 4. Outcomes for acute type A aortic dissection with common carotid artery (CCA) involvement. A thrombosed false lumen was associated high incidences of postoperative stroke and long-term death. Early reperfusion and direct reconstruction of the CCA might be useful. The 95% confidence intervals are also shown in the survival curve.

clinical neurologic symptoms. They advocated early reperfusion and extra-anatomic revascularization for dissected CCA to reduce the severity of postoperative neurologic complications. They defined occlusion or severe stenosis of a supra-aortic branch as imaging cerebral malperfusion.[13] When we classified CCA involvement by the severity of CCA stenosis, an occluded CCA was associated with a greater risk of in-hospital death (35.4%) and worse neurologic outcome (Table 4). Furukawa and colleagues[27] reported a thrombosed CCA as a greater risk factor for perioperative cerebral malperfusion than preoperative neurologic deficits. Collectively, these studies suggest that severely thrombosed CCA stenoses can play a synergistic role in reduced blood flow and postoperative thromboembolism, contributing to unfavorable clinical outcomes.

Using early reperfusion and a direct reconstruction strategy, the intraoperative cerebral malperfusion induced by both crucial factors potentially could be solved. With the current strategy, no patients have needed additional arterial cannulation due to intraoperative cerebral malperfusion except for direct CCA cannulation, whereas the femoral artery was the most frequent cannulation site in our hospital. This result suggests that the current strategy could prevent cerebral malperfusion after establishment of cardiopulmonary bypass. The key determinant for malperfusion induced by thromboembolism is the presence of reentry. If reentry is absent, the false lumen expands. As a result, the true lumen is obstructed by pressure from the false lumen, resulting in cerebral malperfusion. When this occurs in the CCA, we believe that restoring blood circulation by central repair alone is quite difficult. Re-entry in the CCA might represent an origin of thromboembolism, as in one of our cases (Figure E1). Therefore, we believe that both early reperfusion and direct reconstruction of the CCA can preserve true lumen flow and minimize thromboembolism, reducing the risk of devastating neurologic complications.

Study Limitations

Limitation of this study include its single-center, retrospective design. In addition, the small sample size was limited, given the relative rarity of type A acute aortic dissection with CCA involvement. Moreover, the surgical strategy changed slightly over the long study period; a small number of patients underwent central repair under deep hypothermia (20 °C at tympanic temperature) with retrograde cerebral perfusion in the early stage. Among 492 patients with ATAAD, 81 patients (16.5%) without preoperative CCA evaluation were excluded, which inadvertently could have led to a selection bias resulting in a type I error. Although 17 of the 81 excluded patients had preoperative neurologic symptoms, the CCA was not evaluated in these patients because of their preoperative status. A final, important limitation is the difference in modalities—contrast-enhanced CT, plain CT, and carotid duplex scan—adopted for defining CCA thrombosis and severity.

CONCLUSIONS

Outcomes of ATAAD with CCA involvement remain suboptimal. CCA involvement with a thrombosed false lumen, especially an occluded CCA, was associated with comparatively unfavorable clinical outcomes. Further investigation is needed to evaluate the efficacy of the early reperfusion and direct reconstruction strategy.

Conflict of Interest Statement

The authors reported no conflicts of interest.

The *Journal* policy requires editors and reviewers to disclose conflicts of interest and to decline handling or reviewing manuscripts for which they may have a conflict of interest. The editors and reviewers of this article have no conflicts of interest.

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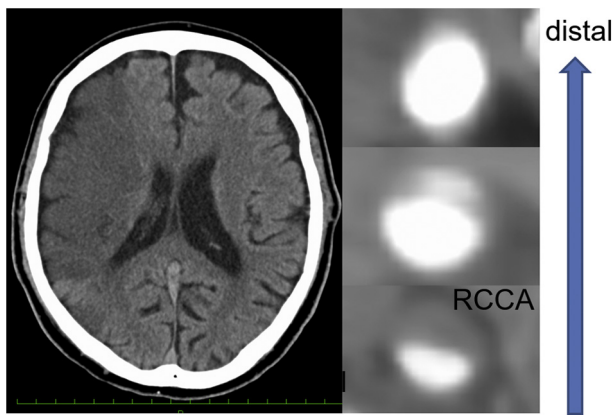
FIGURE E1. Preoperative head computed tomography (CT) and contrast-enhanced CT of the common carotid artery in patients with preoperative coma. Reentry was detected distal to the thrombosed false lumen. *RCCA,* Right common carotid artery.

TABLE E1. Postoperative CT evaluation of dissected CCAs

Approach	Dissected CCA (N = 122)	Thrombosed (N = 72)	Nonthrombosed (N = 50)	*P* value
Postoperative residual CCA dissection, n (%)	80 (65.6)	42 (58.3)	38 (76.0)	.068
Current strategy (N = 15)				
Postoperative residual CCA dissection, n/N (%)	2/15 (13.3)	2/13 (15.4)	0/2 (0.0)	>.999
Thrombosed false lumen, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	>.999
Nonthrombosed false lumen, n (%)	2 (13.3)	2 (15.4)	0 (0.0)	>.999

CT, Computed tomography *CCA,* common carotid artery.

TABLE E2. Outcomes of consecutive patients with the early reperfusion and direct reconstruction strategy

Case	Age, y/sex	CCA involvement	Preoperative neurologic symptom	Postoperative neurologic symptom	mRS ≥5	In-hospital death	30-d death
1	75 M	Occluded	Hemiplegia	None	No	No	No
2	60 F	Nonthrombosed	None	None	No	No	No
3	66 M	Severe	Coma	Hemiplegia	No	No	No
4	70 M	Mild	Coma	Hemiplegia	Yes	Yes	No
5	76 F	Severe	None	None	No	No	No
6	76 F	Occluded	None	None	No	No	No
7	62 M	Mild	Hemiplegia	Hemiplegia	No	No	No
8	70 F	Occluded	None	None	No	No	No
9	58 M	Severe	None	None	No	No	No
10	88 F	Occluded	Hemiplegia	None	Yes	No	No
11	69 M	Severe	None	None	No	No	No
12	64 F	Nonthrombosed	None	Hemiparesis	No	No	No
13	84 F	Occluded	None	None	Yes	Yes	No
14	72 F	Occluded	None	None	No	No	No
15	83 F	Occluded	Hemiplegia	Hemiplegia	No	No	No

CCA, Common carotid artery; *mRS*, modified Rankin scale.