Brachiocephalic artery dissection is a marker of stroke after acute type A aortic dissection repair

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Abstract

OBJECTIVE Postoperative stroke is a serious unsolved complication after acute type A aortic dissection repair. We investigated the incidence and risk factors of stroke and hypothesized that dissection of supra-aortic vessels is an important risk factor of this morbidity. METHODS Between 2012 and 2019, 202 (56% men, median age 68 years) patients with acute type A aortic dissection underwent surgical repair. Clinical data, image findings, methods of circulatory support, and repair technique were retrospectively investigated to explore the risk factor of postoperative stroke. RESULTS Of 202 patients, operative mortality was 6% and the incidence of postoperative stroke was 12% (n=25). Brachiocephalic artery dissection was associated with a higher risk of stroke (odds ratio, 3.89, 95%CI 1.104-13.780; P= .035) having no relation with the presence or absence of left common carotid artery dissection. Preoperative malperfusion syndrome, circulatory arrest time, isolated cerebral perfusion time, repair technique (total arch replacement), and femoral artery perfusion alone were not related to the incident rate of postoperative stroke. Stroke occurred in both hemispheres, regardless of the laterality of carotid artery dissection. CONCLUSION Brachiocephalic artery dissection was an independent risk factor of stroke after acute type A aortic dissection repair.

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Structured abstract

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METHODS

Between 2012 and 2019, 202 (56% men, median age 68 years) patients with acute type A aortic dissection underwent surgical repair. Clinical data, image findings, method of circulatory support, and repair technique were retrospectively investigated to explore the risk factor of postoperative stroke.

RESULTS

Of 202 patients, operative mortality was 6% and the incidence of postoperative stroke was 12% (n=25). Brachiocephalic artery dissection was associated with higher risk of stroke (odds ratio, 3.89, 95%CI 1.104-13.780; P= .035) having no relation with the presence or absence of left common carotid artery dissection. Preoperative malperfusion syndrome, circulatory arrest time, isolated cerebral perfusion time, repair technique (total arch replacement), and femoral artery perfusion alone were not related to the incident rate of postoperative stroke. Stroke occurred in both hemispheres, regardless of the laterality of carotid artery dissection.

CONCLUSION

Brachiocephalic artery dissection was an independent risk factor of stroke after acute type A aortic dissection repair.

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Abbreviations and acronyms

ATAAD=acute type A aortic dissection

BCA=brachiocephalic artery

CT=computed tomography

LCCA=left common carotid artery

LSCA=left subclavian artery

MRI= magnetic resonance imaging

INTRODUCTION

Stroke is a highly morbid complication of acute type A aortic dissection (ATAAD) repair with reported incident rates of 10% to 30%.[1-3] Factors related to stroke after ATAAD repair including the selection of arterial cannulation site, brain protection method, optimal temperature control, and range of replaced aorta had been reported.[2,4-14]

Generally, malperfusion is diagnosed in patients who had clinical symptoms as well as occlusion of the corresponding arteries.[15,16] In the brain, only patients with both acute neurological symptoms and significant stenosis or obstruction in the carotid arteries are defined as brain malperfusion. It is easy to understand that postoperative stroke occurs in such patients. However, there were many cases of postoperative stroke even

in patients without significant stenosis or occlusion of carotid artery and without preoperative neurological symptoms. Stroke rate of ATAAD repair is higher than that of true aortic arch aneurysm repair.[11,14,17,18] In the case of aortic dissection, embolism due to atheroma seems to be less frequent than that in true aneurysm, so dissection itself seems to be involved in the occurrence of cerebral stroke. But the details of the mechanism of stroke after ATAAD repair are still unclear. We have previously reported that a dissection of supra-aortic vessels affected long-term outcome after ATAAD repair.[19] We hypothesized that a dissection of supra-aortic vessels was also a risk factor for stroke after ATAAD repair.

METHODS

Patient Population & definitions

The ethics committees at Yokohama city University medical center approved the study (14 May 2020, B200400073). Because this was observational retrospective study, the need for informed consent was waived.

A patient flow chart was shown in Figure 1. Between September 2012 and September 2019, a total of 283 patients visited our institution with a diagnosis of ATAAD. We evaluated 202 ATAAD patients (56.4% male, median age 68 years, IQR 57-76 years) who underwent open aortic repair surgery for ATAAD in this period. A comparative study was conducted in two groups based on the presence or absence of postoperative stroke. All patients who were suspected of having neurological complications underwent brain computed tomography (CT) and/or magnetic resonance imaging (MRI). We consulted with a neurologist and made a definite diagnosis of cerebral infarction based on the results of CT or MRI. Patients with both imaging and neurological findings were diagnosed stroke. We retrospectively examined the preoperative background factors, CT findings, intraoperative factors, change in dissection of supra-aortic vessels before and after ATAAD repair, and the long-term results of all patients. Supra-aortic vessels were divided into three groups: (1) no dissection, (2) dissection without severe stenosis of true lumen, (3) dissection with severe stenosis of true lumen. The stenosis rate of supra-aortic vessels was measured using the ECST method (European Carotid Surgery Trial) using preoperative CT images. A stenosis rate of 75% or higher was diagnosed as severe stenosis.

Operative techniques

Our basic surgical strategies of central repair operations for ATAAD are as follows. As arterial infusion lines for cardiopulmonary bypass, both the axillary and femoral arteries were regularly used. The absence of true-lumen collapse was always confirmed on transesophageal or epi-aortic echography at the start of extracorporeal circulation. In some patients, ascending aorta was clamped and proximal procedure was started during systemic cooling. For brain protection, antegrade selective cerebral perfusion was performed. Under hypothermic circulatory arrest with rectal temperature 25, balloon-tipped cannulas with pressure monitor lines were inserted individually to a brachiocephalic artery (BCA), left common carotid artery (LCCA), and left subclavian artery (LSCA) from the true lumen of the aortic arch. Cerebral perfusion was conducted by three separate small roller pumps. Start-up perfusion flow rate was 5ml/kg for BCA, 3.3ml/kg for LCCA, and 1.7ml/kg for LSCA, and was regulated to keep adequate perfusion pressure and regional oxygen saturation of frontal lobe. The extent of aortic replacement was determined on the basis of the entry site.

Statistical analysis

All analyses were performed with SAS jmp Statistic, version12.2.0 (SAS Institute Inc., Cary, NC). Categorical variables are presented as frequency (percentage) and continuous variables are presented as median (interquartile range [IQR]) as all were not normally distributed based on Shapiro-Wilk tests. Univariable analysis was performed between patients with and without postoperative stroke using χ^2 tests for categorical variables and Wilcoxon tests for continuous variables. All P values were 2-sided. P values of less than 0.05 were considered to indicate statistical significance. A multivariable logistic regression was performed to evaluate the independent contribution of the 3 primary variables of interest (dissection of brachiocephalic artery, cardiopulmonary arrest time, and preoperative neurological dysfunction). These variables were selected based on previous reports and their clinical significance. Results were displayed as odds ratio with 95%

confidence interval and$P$ -values. For analyzing long-term survival, the Kaplan-Meier curve was calculated, and the groups were tested by the log-rank test to describe differences in survival.

RESULTS

Preoperative characteristics of patients were shown in Table 1. past history of cerebral infarction was observed in 16 (8%). Preoperative malperfusion syndrome was observed in 54 (26%) and cardiogenic shock was also observed in 55 (27%). They had no significant difference between the two groups. Preoperative neurological symptoms were observed in 14 (7%), significantly more frequent in the stroke group. Hemiplegia was observed in 10 (5%) (including insufficiency), coma in 1 (0.5%), delirium in 1 (0.5%), and syncope in 2 (1%). Preoperative hemiplegia, coma, and delirium were significantly more frequently observed in the stroke group.

Preoperative CT findings were shown in Table 2. Aortic dissection with a patent false lumen was found in 73% (n=148), and 27% (n=54) were intramural hematomas. Dissection of any supra-aortic vessels was observed in 71% (n=142) of the patients, significantly more frequent in the stroke group ($P = .028$). 64% (n=129) of patients had dissection in the BCA, 38% (n=77) in the LCCA, and 35% (n=71) in the LSCA. Only the BCA dissection was significantly higher in the stroke group ($P = .004$). Of the 22 patients of BCA dissection with postoperative stroke, 6 patients had a patent false lumen of BCA and 16 had a thrombotic false lumen of BCA.

Operative characteristics were shown in Table 3. Cardiopulmonary bypass time in the stroke group was significantly longer than in no stroke group. Lower body circulatory arrest time and lowest temperature did not show a significant difference in two groups. Cerebral perfusion was antegrade in 99%, and there was no significant difference in cerebral perfusion time. Aortic range of replacement was also not different significantly. Coronary artery bypass grafting was administered in 6% (n=12), and aortic valve surgery or valve sparing was observed in 5% (n=11), and there was no significant difference between the two groups.

Postoperative stroke was observed in 12% (n=25), operative mortality was 6% (n=13) and the hospital mortality was 8% (n=16). In the 25 patients who developed stroke, brain CT and/or MRI showed that 12 patients had unilateral cerebral infarction (7 patients on the right and 5 patients on the left) and 13 patients had bilateral cerebral infarction. In 7 patients in whom dissection did not reach LCCA, cerebral infarction of left hemisphere was observed. In addition, Right hemisphere cerebral infarction was observed in 3 patients without BCA dissection. Of these 13 patients with bilateral cerebral infarction, preoperative cardiac shock was observed in 8 patients, and preoperative cardiac tamponade was observed in 8 patients. The mechanism of cerebral infarction seemed to be an embolic infarction in 19 patients and a watershed damage in 6 patients, judging by the distribution of infarction. Other early results showed in Table 4.

In this study, we evaluated the changes of false lumen of the BCA in 21 of the 25 patients who developed postoperative stroke after ATAAD repair. We could not perform postoperative CT in four patients. Of the 13 patients who had preoperatively thrombosed false lumen of the BCA, false lumen remained thrombosed in 7 patients, healed in 2, and became patent in 4 postoperatively. Other changes were described in Figure 2.

Combination of BCA and LCCA dissection was analyzed in Table 5. The incidence of stroke in patients without BCA dissection was 4%(n=3), while the incidence of stroke in patients with BCA dissection accompanied with severe stenosis of true lumen was as high as 24%(n=5). However, even in cases of BCA dissection without severe stenosis of true lumen, the incidence of stroke was as high as 16%(n=17). On the other hand, in cases where there was a dissection in LCCA but not in BCA, there was no occurrence of stroke after ATAAD repair. Stroke occurred in 3 of 11 LCCA dissection cases with severe stenosis of true lumen, and two of the three patients also had BCA dissection with severe stenosis of true lumen.

On univariable analysis, BCA dissection, preoperative neurological dysfunction, and longer CPB time were significant risk factors of stroke after ATAAD repair. Only BCA dissection was an important risk factor for stroke after ATAAD repair on multivariable analysis (Odds ratio 3.89, 95% CI: 1.104-13.780, $P = .035$).

(Table 6)

Mid-term results of patients with or without stroke after ATAAD repair were shown in Figure 3. The median follow-up period was 35 months (IQR, 9-53 months), and follow-up was completed in 95.5% of the patients. Overall mortality was 11.9% (n=24). Overall survival was compared with or without stroke after ATAAD repair. Overall survival was 80.0 ± 8.0% vs 91.8 ± 2.1%, 72.7 ± 10.1% vs 91.8 ± 2.1%, 72.7 ± 10.1% vs 89.1 ± 2.6% after 1, 2, 3 years, respectively. Survival rate was significantly lower in postoperative stroke group in mid-term survival (Hazard Ratio 2.95, 95% CI: 1.159-7.509, log-rank, $P = .015$).

DISCUSSION

There are many reports on the occurrence of stroke after ATAAD repair. Conzelmann and colleagues examined 2137 cases in the German Registry of Acute Aortic Dissection type A (GERAADA) and reported a mortality rate of 16.9% and a stroke rate of 12.9%. In addition, they reported that the progression of dissection to supra-aortic vessels and the preoperative malperfusion syndrome were risk factors for stroke after ATAAD repair, and that the choice of arterial cannulation site did not contribute to the occurrence of stroke.[18] Ghoreishia and colleagues examined the Society of Thoracic Surgeons Adult Cardiac Surgery Database (STS ACSD), and found 17% mortality rate and 13% stroke rate in 7353 patients. They reported that type A repair using axillary arterial cannulation significantly reduced postoperative stroke compared to that using femoral cannulation, and retrograde cerebral perfusion had a lower incidence of stroke than that in deep hypothermic circulatory arrest or selective antegrade cerebral perfusion.[14] However, this report did not consider dissection of supra-aortic vessels. Zhao and colleagues examined preoperative CT angiography and preoperative MRI in 281 patients at a single center, and reported that 103 patients (36.7%) had cerebral infarction before ATAAD repair. They concluded that moderate or higher aortic insufficiency, narrowing of the true lumen of the ascending aorta, and dissection of the common carotid artery were risk factors for preoperative stroke.[20] However, they did not mention surgical results or postoperative cerebral infarction. In our study of 202 patients, the incidence of stroke after ATAAD repair was almost the same (12%) with the report from GERAADA and STS ACSD, although the mortality rate was better than them. Furthermore, these stroke rate of ATAAD repair was higher than that of total arch replacement for atherosclerotic arch aneurysms in Japanese registry.[11,21] In recent years, detailed multi-detector row CT images have been obtained before ATAAD repair in many patients, and a detailed study of dissection into supra-aortic vessels has become possible. Our study is the first report to investigate the dissection progress to supra-aortic vessels in detail and to examine the relationship with postoperative stroke after ATAAD repair. The results of our study showed that only the progression of dissection to BCA was a significant risk factor for postoperative stroke.

Many factors are thought to be associated with the occurrence of stroke in perioperative period of ATAAD repair. One is the hypoperfusion of the area due to severe stenosis or occlusion of the carotid artery. If the thrombosed dissection progresses to BCA and the true lumen is occluded without forming a re-entry, cerebral infarction is predictable. Next, in cases where a re-entry is formed in the periphery of the supra-aortic vessels and supra-aortic vessels have patent false lumen, the thrombus formed in the false lumen can flows through the re-entry into the true lumen. Eight of the 21 patients had a patent false lumen of BCA after ATAAD repair (Figure 2). However, four patients did not have dissection, and nine had thrombosed false lumen without severe stenosis of true lumen. The mechanism of cerebral infarction in these patients cannot be explained by above two mechanisms. And cerebral infarction occurred in both left and right hemispheres, even in the side without dissection of BCA and LCCA.

Although the cerebral artery is the terminal artery, there is a Willis' circle as left and right communicating arteries. The formation of a Willis' circle varies greatly between individuals. In patients with a well developed Willis' circle, even complete occlusion of the carotid artery may not exhibit neurological symptoms. A hypothesis came to our mind. In patients whose perfusion pressure of the unilateral carotid artery rapidly decrease due to acute aortic dissection, even if blood flow to the brain tissue is maintained, a stagnation of the blood flow occurs at the peripheral part of the occluded carotid artery (proximal to Willis' circle) and a thrombus may be formed. This thrombus may cause bilateral cerebral embolism when blood flow in the

carotid artery was restored by central repair for ATAAD. In patients with dissection of BCA, even if true lumen of BCA is not significantly stenotic at the time of CT imaging, it is possible that significant stenosis of true lumen temporarily occurs in the early stage of onset. Currently, we have no data to elucidate this mechanism. Preoperative CT angiography of intracranial arteries should be investigated on ward.

There were some limitations to this study that should be addressed. We didn't perform central repair in the patients with coma, and they were excluded from this study. This is a single-center study with a small number of cases. ATAAD repair was performed promptly after admitting our hospital, but it was transferred to our hospital after being diagnosed at another hospital in many cases, and the time from onset to ATAAD repair varied. Diagnostic imaging around a Willis' circle has not yet been performed. The mechanism of cerebral infarction discussed in this study is speculative. In addition, CT and MRI were not performed in all postoperative cases, small cerebral infarction without neurological symptoms may be overlooked.

CONCLUSION

In this study, we identified independent preoperative risk factors for stroke after ATAAD repair. On univariable analysis, BCA dissection, preoperative neurological dysfunction, and longer cardiopulmonary bypass time were significant risk factors. Only BCA dissection was an important risk factor for stroke after ATAAD repair on multivariable analysis.

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Tables Table 1. Preoperative characteristics of patients

	Overall cohort(N=202)	Stroke (N=25)	No stroke (N=177)	*P* value
Age(y)	68 [57-76]	69 [59-73]	67 [56-77]	.904
Male	114 (57)	14 (56)	101 (57)	.963
Diabetes mellitus	9 (4)	0 (0)	9 (5)	.119
Hypertension	122 (60)	12 (48)	111 (63)	.180
Dialysis	5 (2)	0 (0)	5 (3)	.510
Chronic lung disease	3 (1)	0 (0)	3 (2)	.371
Cerebrovascular disease	16 (8)	1 (4)	15 (8)	.611
Preoperative malperfusion syndrome	54 (26)	9 (36)	45 (25)	.276
Cerebral	14 (7)	5 (20)	9 (5)	.018
Coronary	11 (5)	3 (12)	8 (4)	.169
Spinal	4 (2)	0 (0)	4 (2)	.030
Abdominal	8 (4)	1 (4)	7 (4)	.991
Renal	5 (2)	0 (0)	5 (3)	.247
Leg	25 (12)	4 (16)	21 (12)	.569

	Overall cohort(N=202)	Stroke (N=25)	No stroke (N=177)	*P* value
Preoperative neurological dysfunction	14 (7)	5 (20)	9 (5)	.018
Hemiplegia	10 (5)	5 (20)	9 (5)	.003
Coma	1 (0.5)	1 (4)	0 (0)	.040
Delirium	1 (0.5)	1 (4)	0 (0)	.040
Syncope	2 (1)	0 (0)	2 (1)	.466
Reoperation	3 (1)	0 (0)	3 (2)	.371
Cardiogenic shock	55 (27)	11 (44)	44 (25)	.053
Cardiac tamponade	60 (30)	9 (36)	51 (29)	.469
Aortic insufficiency				.223
None	50 (25)	7 (29)	43 (24)	
Trivial	38 (19)	2 (8)	36 (20)	
Mild	45 (22)	3 (13)	42 (24)	
Moderate	42 (21)	7 (29)	35 (20)	
Severe	25 (12)	5 (21)	20 (11)	

Table 2. Preoperative CT findings

	Overall cohort (N=202)	Stroke (N=25)	No stroke (N=177)	*P* value
False lumen of aorta				.739
Patent	148 (73)	19 (76)	129 (73)	
Thrombosed	54 (27)	6 (24)	48 (27)	
Supra-aortic dissection	142 (71)	22 (88)	120 (68)	.028
one	55 (27)	8 (32)	47 (27)	
two	42 (21)	5 (20)	37 (21)	
three	46 (23)	9 (36)	37 (21)	
Brachiocephalic artery				.004
Dissection (+)	129 (64)	22 (88)	107 (61)	
Dissection (-)	72 (36)	3 (12)	69 (39)	
Left common carotid artery				.282
Dissection (+)	77 (38)	12 (48)	65 (37)	
Dissection (-)	125 (62)	13 (52)	112 (63)	
Left subclavian artery				.329
Dissection (+)	71 (35)	11 (44)	60 (35)	
Dissection (-)	131 (65)	14 (56)	117 (66)	

Table 3. Surgical procedures

	Overall cohort (N=202)	Stroke (N=25)	No stroke (N=177)	*P* value
CPB time (min)	220 [179-263]	235 [200-301]	219 [175-260]	.023
Crossclamp of ascending aorta	118 (58)	15 (60)	103 (58)	.664
Cannulation strategy				.448
Axillary + Femoral	189 (94)	22 (88)	167 (94)	

	Overall cohort (N=202)	Stroke (N=25)	No stroke (N=177)	*P* value
Femoral	10 (5)	2 (8)	8 (5)	
Direct aorta	2 (1)	1 (4)	1 (0.5)	
Inominate	1 (0.5)	0 (0)	1 (0.5)	
Lowest temperature()	23.4 [22.6-24.3]	23.3 [22.7-23.8]	23.4 [22.6-24.3]	.112
Circulatory arrest time (min)	56 [46-70]	62 [48-75]	56 [45-70]	.287
Cerebral perfusion strategy				.850
Antegrade	200 (99)	25 (100)	175 (99)	
Retrograde	2 (1)	0 (0)	2 (1)	
Cerebral perfusion time (min)	61 [40-152]	67 [39-175]	60 [40-150]	.882
Aortic Procedure				
Root	25 (13)	5 (20)	20 (11)	.243
Ascending	98 (48)	11 (44)	87 (49)	.819
Total arch	88 (44)	10 (40)	78 (44)	.521
Aortic valve	11 (5)	2 (8)	9 (5)	.158
CABG	12 (6)	3 (12)	9 (5)	.798
Postoperative VA-ECMO	6 (3)	2 (8)	4 (2)	.082

CPB , cardiopulmonary bypass; *CABG* , coronary artery bypass grafting; *VA-ECMO* , venoarterial extracorporeal membrane oxygenation.

Table 4. Early results of ATAAD repair

	Overall cohort (N=202)	Stroke (N=25)	No stroke (N=177)	*P* value
Newly Dialysis	14 (7)	4 (16)	10 (6)	.091
Transitory	10 (5)	3 (12)	7 (4)	
Permanent	4 (2)	1(4)	3 (2)	
Paraplegia	2 (1)	0 (0)	2 (1)	.466
Paraparesis	1 (0.5)	0 (0)	1 (0.6)	.607
Redo for bleeding	5 (3)	2 (9)	3 (2)	.104
SSI	3 (1)	1 (4)	2 (1)	.341
30 day mortality	13 (6)	4 (16)	9 (5)	.068
Hospital mortality	16 (8)	4 (16)	12 (7)	.147

SSI , surgical site infection

Table 5. Dissection rate of supra-aortic vessels and stroke rate respectively.

		LCCA	LCCA	LCCA
		No Dissection	Dissection without severe stenosis	Dissection with severe ste
BCA	No Dissection	67 (3): 4%	6 (0): 0%	0 (0): 0%
	Dissection without severe stenosis	47 (7): 15%	55 (9): 16%	6 (1): 17%
	Dissection with severe stenosis	11 (3): 27%	5 (0): 0%	5 (2): 40%
	Total	125 (13): 10%	66 (9): 14%	11 (3): 27%

Total number (Stroke number): Stroke rate (%). *BCA* , brachiocephalic artery; *LCCA* , left common carotid artery

Table 6. Multi-variable analysis of risk factors of postoperative stroke after ATAAD repair

Factor	OR (95% CI)	*P* value
CPB time >180min	2.341 (0.652-8.397)	.192
Dissection of Brachiocephalic artery	3.899 (1.104-13.780)	.035
Preoperative neurological dysfunction	3.322 (0.984-11.217)	.053

CI , confidence interval; *OR* , odds ratio

Figure 1

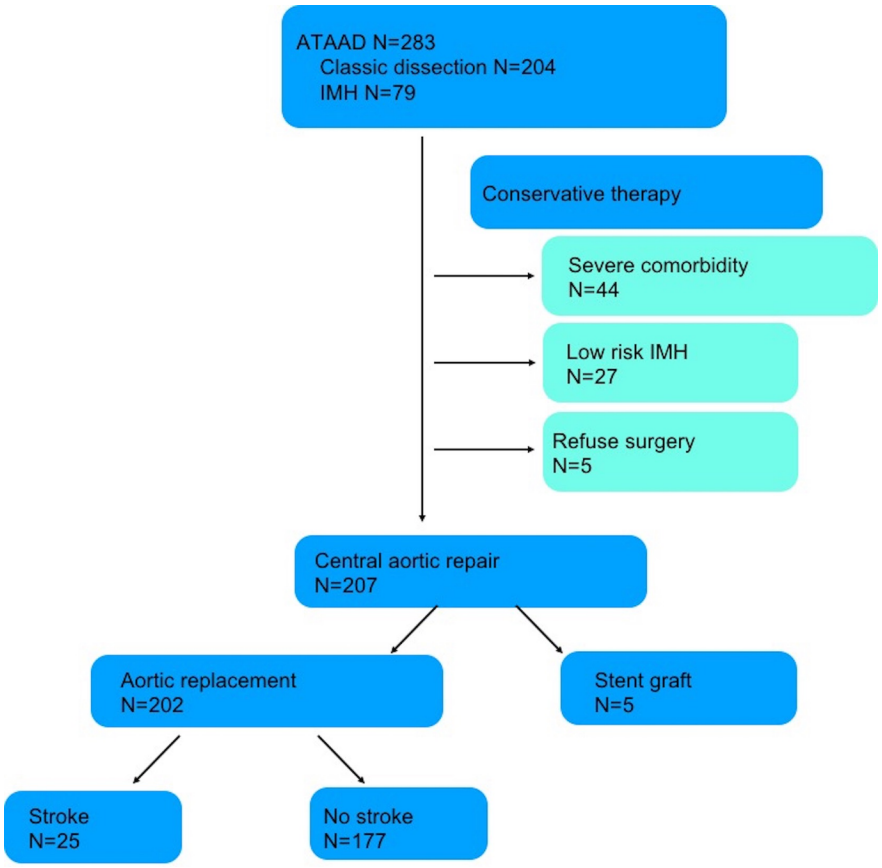

Figure 2

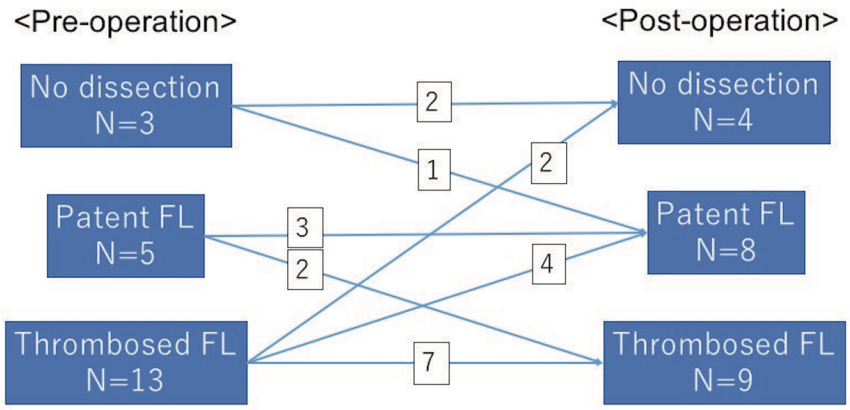

Figure 3

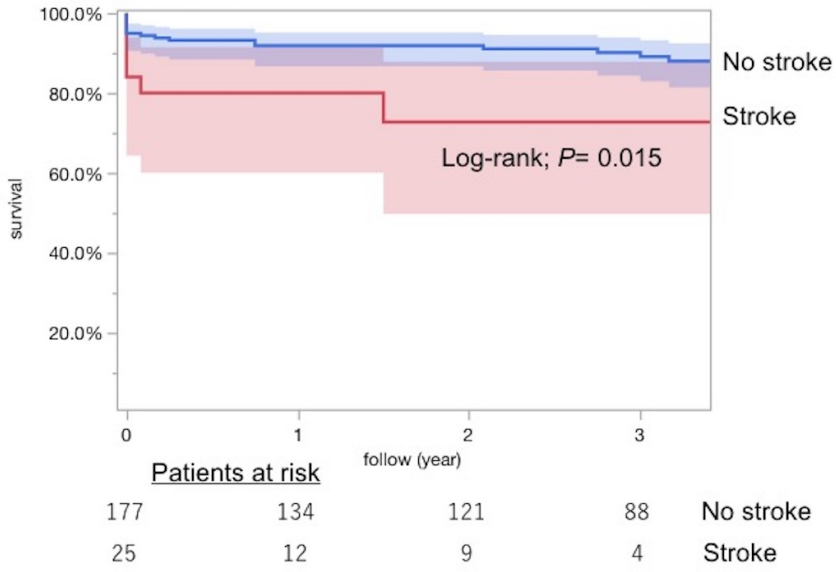

Figure legends

Figure 1

Patient flow chart demonstrating the selection of patients undergoing acute type A aortic dissection repair

Figure 2

Pre and postoperative CT findings of brachiocephalic artery of the patients who developed stroke. Of the 13 patients who had preoperatively thrombosed false lumen of the BCA, false lumen remained thrombosed in 7 patients, healed in 2, and became patent in 4 postoperatively.*FL* , false lumen

Figure 3

Mid-term survival of patients with and without postoperative stroke after ATAAD repair. Survival rate was significantly lower in postoperative stroke group in mid-term survival (Hazard Ratio 1.63, 95% CI: 1.004-2.636, log-rank, $P = .015$). *ATAAD* , acute type A aortic dissection