Original Article

Prevalence of carotid artery stenosis and intra-cranial lesions in patients with aortic arch aneurysm and its association with intraoperative regional cerebral oxygen saturation and postoperative neurological outcomes

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Background: Although aortic aneurysm is associated with vascular aging and atherosclerosis, carotid and intracranial vascular disease prevalence in patients with aortic arch aneurysm remains unclear. Similarly, the effect of carotid and intracranial lesions on postoperative outcomes is unknown. This study aimed to investigate the prevalence of carotid artery stenosis and intracranial lesions in patients with aortic arch aneurysm and its association with intraoperative regional cerebral oxygen saturation (rScO2) and postoperative neurological outcomes, including delirium and cerebral infarction.

Methods: This retrospective observational study included 133 patients with true aortic arch aneurysm who underwent preoperative magnetic resonance imaging (MRI). We evaluated the prevalence of carotid and intracranial arterial lesions. Symptomatic cerebral infarction and delirium, defined by the confusion assessment method for the intensive care unit, were evaluated for their association with preoperative cerebrovascular lesions. Additionally, changes in regional saturation of the cerebral tissue at different surgical phases were evaluated for patients with and without cerebrovascular lesions.

Results: Fifteen (11.3%) patients experienced symptomatic cerebral infarction, and 64 (48.1%) had postoperative delirium. Preoperative MRI showed old infarction, microbleeds, significant carotid artery stenosis, and intracranial lesions in 21.1%, 14.3%, 10.5%, and 7.5% of the patients, respectively. White matter hyperintensities with Fazekas scale 2 were observed in 40.6% of the patients, while Fazekas scale 3 were observed in 18.8% of the patients. Preoperative MRI findings and postoperative neurological outcomes were not significantly different. Seventy-six patients underwent rScO2 monitoring intraoperatively. Changes in rScO2 in patients with and without carotid/cerebrovascular lesions were not significantly different. However, rScO2 was significantly lower in patients who developed cerebral infarction.

Conclusions: Significant carotid artery stenosis and intracranial lesions were observed in 10.5% and 7.5% of the patients, respectively. Although preoperative MRI findings and changes in rScO2 or postoperative outcomes showed no significant association, patients with postoperative cerebral infarction showed significantly lower rScO2 intraoperatively.

Keywords: White matter hyperintensities (WMH); aortic arch aneurysm; near-infrared spectroscopy (NIRS); carotid artery; infarction

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Submitted Jan 03, 2024. Accepted for publication Mar 15, 2024. Published online May 24, 2024.
doi: 10.21037/jtd-24-78
View this article at: https://dx.doi.org/10.21037/jtd-24-78

Introduction

With the progressive increase in the aging population, more patients require treatment for vascular diseases associated with vascular aging and atherosclerosis. Vascular aging and atherosclerosis may lead to stenotic lesions such as coronary artery disease and carotid artery stenosis or aortic dilation, resulting in aortic aneurysm. Since atherosclerotic changes occur in the whole vascular system, several aortic lesions may co-exist in a patient with vasculopathy. However, the prevalence of intracranial or carotid artery stenosis in patients with thoracic aortic aneurysm is unclear.

The prevalence of carotid artery stenosis in patients undergoing coronary artery bypass grafting (CABG) has been estimated to be approximately 12–17% (1). In such patients, mechanical circulatory support or concomitant revascularization of the carotid artery has been suggested to reduce the risk of insufficient cerebral perfusion during off-pump CABG (1). Cerebral protection during aortic arch aneurysm repair is managed by hypothermia and mechanical cerebral perfusion, which are also often guided by near-infrared spectroscopy (NIRS). Despite these techniques, the incidence of postoperative cerebral vascular events for patients with aortic arch aneurysms remains high, reported to be approximately 4.7–7.3% and 10.3% for permanent and temporary neurological deficits, respectively (2).

This study aimed to investigate the prevalence of intracranial or carotid artery disease in patients undergoing surgery for a true aortic arch aneurysm. In addition, the association between concomitant carotid/cerebrovascular lesions and changes in intraoperative regional cerebral oxygen saturation (rScO2), as well as postoperative outcomes, were evaluated. The study's hypotheses were: (I) intracranial or carotid artery disease in patients with aortic arch aneurysm has a high prevalence, and (II) patients with cerebrovascular lesions have lower rScO2 during cardiopulmonary bypass (CPB) and are at higher risk for postoperative delirium and cerebral infarction. We present this article in accordance with the STROBE reporting checklist (available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-78/rc).

Highlight box

Key findings

- There were old infarction in 21.1%, microbleeds in 14.3%, significant carotid artery stenosis in 10.5% and intracranial lesion in 7.5% of the patients with true aortic arch aneurysm. Preoperative cerebrovascular lesion was not a risk factor for postoperative cerebral infarction or delirium. There were no significant changes in regional cerebral oxygen saturation (rScO2) in patients with and without carotid/cerebrovascular lesions. However, patients with postoperative cerebral infarction had significantly lower rScO2 during surgery.

What is known and what is new?

- rScO2 of the affected hemisphere has been reported to be lower in patients with perioperative cerebral infarction. However, prevalence of carotid/cerebrovascular lesion in patients with aortic arch aneurysm and its association with differences in rScO2 is not well known.
- The present study showed high prevalence of carotid/cerebrovascular lesions in patients with aortic arch aneurysm. There were no significant differences in rScO2 during cardiopulmonary bypass and selective cerebral perfusion in patients with and without carotid/cerebrovascular lesion. However, rSCO2 was lower in patients with postoperative cerebral infarction.

What is the implication, and what should change now?

- Although patients with aortic aneurysm have high prevalence of cerebrovascular lesions, this was not a risk factor for postoperative delirium and cerebral infarction. Special attention should be given to patients with low rScO2 during surgery, as this was observed in patients with postoperative cerebral infarction.

Methods

In our institution, 146 patients underwent aortic arch replacement for true aortic arch aneurysm between March 2017 and March 2023. This retrospective observational study included 133 of these patients who were not contraindicated for preoperative magnetic resonance imaging (MRI). Patients with acute or chronic aortic dissection were excluded (*Figure 1*). Significant carotid artery stenosis was defined as stenosis >70%, and white matter hyperintensities (WMH) were classified using the Fazekas scale (3,4). For postoperative outcomes, symptomatic postoperative cerebral infarction and delirium defined by the confusion assessment method for the intensive care unit (CAM-ICU) were evaluated.

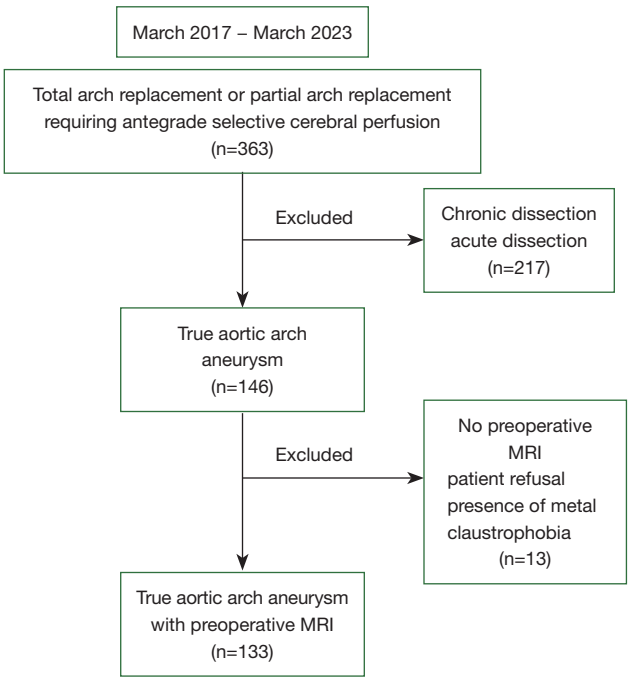

Figure 1 Flow diagram of patient selection for the study. MRI, magnetic resonance imaging.

Prolonged mechanical ventilation was defined as mechanical ventilation exceeding 48 h. Seventy-six patients had intraoperative monitoring of rScO2 with NIRS, performed at the discretion of the anesthesiologist. This study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). It was approved by the institutional ethics board of Saitama Medical Center, Jichi Medical University (No. S23-040), and individual consent for this retrospective analysis was waived.

Surgical procedure

Contrast-enhanced computed tomography and intraoperative epi-aortic echography were performed to confirm the clamping and cannulation sites. When the aorta was not suitable for cannulation, the axillary artery was used for aortic cannulation. The protocol for heparinization was consistent throughout the cohort. In brief, 300 U/kg of heparin was administered to the patient before initiation of CPB. Activated clotting time was kept above 500 s by the addition of 2–10 mL of heparin via the CPB. Protamine was as never used during CPB.

Bicaval cannulation was performed, and CPB was initiated. Patients were cooled to a rectal temperature of 25 ℃. For patients without aortic insufficiency, cardioplegia was administered every 30 min via the aortic root after clamping the ascending aorta. In contrast, for patients with aortic insufficiency, retrograde cardioplegia from coronary sinus was performed. After systemic circulatory arrest, selective antegrade cerebral perfusion was started from three aortic arch branches at a flow pressure of 100–150 mmHg, controlled at 700–800 mL/min using a flowmeter. All patients, including those requiring total and partial arch replacement, underwent selective antegrade cerebral perfusion from all three aortic arch branches. rScO2 and bilateral radial artery pressure monitoring were used to confirm sufficient perfusion of the aortic arch branches.

The distal anastomosis was performed by intermittent mattress sutures, and systemic perfusion was restarted from the side branch of the prosthetic graft. The patients were gradually re-warmed to 32 ℃ while proximal anastomosis was performed. An aortic clamp was released after the proximal anastomosis, and the patients were re-warmed further under beating heart during the reconstruction of the aortic arch branches.

The prosthetic grafts used in this study were J-Graft (Japan Lifeline Co. Ltd., Tokyo, Japan), Triplex (Terumo, Tokyo, Japan), Hemashield (Getinge, Gothenburg, Sweden), and Lupiae (Terumo, Tokyo, Japan), selected at the discretion of the surgeon.

NIRS

Two sensor pads from NIRS monitor (Foresight, CASMED, Connecticut, USA) were placed on both sides of the patient's forehead before commencing the surgery. Continuous rScO2 measurement from NIRS monitor and arterial pressure were integrated digitally into the hemodynamic monitor for recording (Intellivue, Phillips, Amsterdam, Netherlands).

Statistical analysis

Normal distribution of data was assessed using the Kolmogorov-Smirnov test. For comparing two groups, continuous data that were normally distributed were analyzed using the Student's *t*-test [mean ± standard deviation (SD)], and continuous data that were not normally distributed were analyzed using the Mann-Whitney test (median, q25–q75). Fisher's exact test was used to compare categorical variables (n, %). Statistical analysis was performed for patients with and without postoperative

delirium and cerebral infarction. Intraoperative changes in rScO2 were evaluated for each hemisphere. For patients with unilateral cerebrovascular lesions, the diseased hemisphere was grouped into cerebrovascular lesions, while the undiseased hemisphere was grouped into no-cerebrovascular lesions. Differences in rScO2 were evaluated for diseased and non-diseased hemispheres at each surgical phase (baseline, before CPB, after initiation of CPB, before circulatory arrest, circulatory arrest, and after selective cerebral perfusion). The level of statistical significance was set at $P<0.05$, and all analyses were performed using Stata (version 13.1, Stata Corp, College Station, TX, USA) and Prism 5 (GraphPad Software Inc., La Jolla, CA, USA).

Sample size calculation

Based on the previous report from Holmgaard *et al.*, the difference in mean rScO2 was 6.03%, with a standard deviation of <9.46% in patients with and without cerebral infarction after cardiac surgery (5). In addition, the prevalence of carotid artery stenosis in patients undergoing CABG was 12–17% (1). With an expected 15% prevalence of carotid artery stenosis in patients with aortic arch aneurysm, a sample size of 121 provides a statistical power of 0.80 with an alpha error of 0.05.

Definition and outcomes

As a primary outcome, this study aimed to evaluate the prevalence of carotid artery stenosis and intracranial lesions in patients with true aortic arch aneurysms. The secondary outcome was to evaluate the effect of preoperative carotid/cerebrovascular lesions on intraoperative rScO2 measurement and the incidence of postoperative neurological outcomes, including delirium and cerebral infarction. Postoperative delirium was defined by the CAM-ICU performed by a certified nurse, and cerebral infarction was defined as symptomatic cerebral infarction, including paralysis, paresis, and disturbance of consciousness, confirmed by the existence of an ischemic lesion on MRI by a radiologist.

Results

Table 1 shows the patient demographics. Sixty-four (48.1%) patients had postoperative delirium, and 15 (11.3%) experienced postoperative symptomatic cerebral infarction. Age and comorbidities were not significantly different in patients with and without delirium and cerebral infarction. However, male patients were more likely to experience cerebral infarction postoperatively (no cerebral infarction: 72.9% *vs.* cerebral infarction: 100.0%, P=0.02).

A total arch replacement was performed in 125 patients, whereas eight patients underwent partial arch replacement with reconstruction of two aortic arch branches. Although the surgery performed was not significantly different, patients with delirium tended to have longer aortic clamp (no delirium: 106±31.1 min *vs.* delirium: 121±54.8 min, P=0.06) and CPB time (no delirium: 179±39.0 min *vs.* delirium: 195±56.7 min, P=0.05) compared to patients without delirium. There were no significant differences in the lowest hemoglobin (no delirium: 7.9±1.23 g/dL *vs.* delirium: 7.6±1.10 g/dL, P=0.25; no cerebral infarction: 7.8±1.20 g/dL *vs.* cerebral infarction: 7.9±1.03 g/dL, P=0.75) and mean partial pressure of carbon dioxide (PaCO2) levels (no delirium: 43±5.3 mmHg *vs.* delirium: 43±4.2 mmHg, P=0.91; no cerebral infarction: 43±4.8 mmHg *vs.* cerebral infarction: 44±4.6 mmHg, P=0.44) intraoperatively in patients with and without delirium or cerebral infarction. Patients with delirium (no delirium: 11.6% *vs.* delirium: 34.4%, P=0.003) and cerebral infarction (no cerebral infarction: 17.8% *vs.* cerebral infarction: 60.0%, P=0.001) had a higher incidence of prolonged ventilation. Although not significantly different, patients with cerebral infarction were at higher risk of early mortality (no cerebral infarction: 1.7% *vs.* cerebral infarction: 13.3%, P=0.06).

Preoperative MRI showed old infarction, microbleeds, unilateral hypoplastic vertebral artery, significant carotid artery stenosis, and intracranial lesions in 28 (21.1%), 19 (14.3%), 34 (25.6%), 14 (10.5%) and 10 (7.5%) patients with aortic arch aneurysm, respectively. WMH with a Fazekas scale of 0–1, 2, and 3 was observed in 54 (40.6%), 54 (40.6%), and 25 (18.8%) patients, respectively. The preoperative cerebrovascular lesions in patients with and without delirium and cerebral infarction were not significantly different (*Table 2*).

Figure 2 and *Table 3* show the perioperative changes in rScO2 measured by NIRS. The rScO2 showed a slight decrease from baseline after anesthesia, which increased with CPB initiation. There was a drop in rScO2 at circulatory arrest, which recovered after initiation of selective antegrade cerebral perfusion. rScO2 showed a slight decline in its measurement with the continuation of the surgery. rScO2 were not significantly different at each surgical phase in patients with and without significant

Table 1 Patient demographics, surgical details and outcomes

Variables	Delirium			Cerebral infarction		
	No delirium, n=69	Delirium, n=64	P	No cerebral infarction, n=118	Cerebral infarction, n=15	P
Demographics						
Age (years)	73±8.4	74±9.3	0.75	73±9.0	78±6.2	0.05
Male	51 (73.9)	50 (78.1)	0.69	86 (72.9)	15 (100.0)	0.02
Hypertension	60 (87.0)	59 (92.2)	0.40	105 (89.0)	14 (93.3)	>0.99
Dyslipidemia	29 (42.0)	37 (57.8)	0.08	60 (50.8)	6 (40.0)	0.59
Diabetes	11 (15.9)	11 (17.2)	>0.99	19 (16.1)	3 (20.0)	0.71
Ischemic heart disease	10 (14.5)	6 (9.4)	0.43	15 (12.7)	1 (6.7)	0.69
Cerebral vascular disease	5 (7.2)	10 (15.6)	0.17	14 (11.9)	1 (6.7)	>0.99
Chronic obstructive pulmonary disease	14 (20.3)	16 (25.0)	0.54	26 (22.0)	4 (26.7)	0.74
ARB/ACE-I	19 (27.5)	18 (28.1)	>0.99	34 (28.8)	3 (20.0)	0.56
Calcium channel blocker	48 (69.6)	46 (71.9)	0.85	85 (72.0)	9 (60.0)	0.37
Beta blocker	21 (30.4)	22 (34.4)	0.71	36 (30.5)	7 (46.7)	0.25
Anti-coagulation therapy	6 (8.7)	10 (15.6)	0.29	14 (11.9)	2 (13.3)	>0.99
Anti-platelet therapy	12 (17.4)	20 (31.3)	0.07	26 (22.0)	6 (40.0)	0.20
Statin	28 (40.6)	34 (53.1)	0.17	55 (46.6)	7 (46.7)	>0.99
Surgery						
Concomitant coronary artery bypass grafting	9 (13.0)	6 (9.4)	0.58	14 (11.9)	1 (6.7)	>0.99
Concomitant valve surgery	13 (18.8)	11 (17.2)	0.82	23 (19.5)	1 (6.7)	0.46
Aortic clamp time (min)	106±31.1	121±54.8	0.06	112±40.3	122±71.0	0.41
Pump run (min)	179±39.0	195±56.7	0.05	184±44.9	206±72.1	0.10
Lowest hemoglobin (g/dL)	7.9±1.23	7.6±1.10	0.25	7.8±1.20	7.9±1.03	0.75
Mean PaO2 (mmHg)	429±50.1	432±50.0	0.75	432±50.4	423±46.7	0.48
Mean PaCO2 (mmHg)	43±5.3	43±4.2	0.91	43±4.8	44±4.6	0.44
Peak PaCO2 (mmHg)	53±8.8	54±8.2	0.69	53±8.6	54±8.3	0.68
Total arch replacement	64 (92.8)	61 (95.3)	0.72	110 (93.2)	15 (100.0)	0.60
Prosthetic graft						
J-graft	59 (85.5)	53 (82.8)	0.30	101 (85.6)	11 (73.3)	0.06
Triplex	9 (13.0)	8 (12.5)		15 (12.7)	2 (13.3)	
Others	1 (1.5)	3 (4.7)		2 (1.7)	2 (13.3)	
Staff surgeon	40 (58.0)	36 (56.3)	0.86	66 (55.9)	10 (66.7)	0.58
Post-operative outcome						
Prolonged ventilation	8 (11.6)	22 (34.4)	0.003	21 (17.8)	9 (60.0)	0.001
Delirium				53 (44.9)	11 (73.3)	0.05
30-day mortality	2 (2.9)	2 (3.1)	>0.99	2 (1.7)	2 (13.3)	0.06

Data are presented as mean ± standard deviation or n (%). ACE-I, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; PaCO2, partial pressure of carbon dioxide; PaO2, partial pressure of oxygen.

Table 2 Preoperative magnetic resonance imaging findings

MRI findings	Total, n=133	Delirium			Cerebral infarction		
		No delirium, n=69	Delirium, n=64	P	No cerebral infarction, n=118	Cerebral infarction, n=15	P
Old infraction	28 (21.1)	14 (20.3)	14 (21.9)	0.84	24 (20.3)	4 (26.7)	0.52
Microbleeds	19 (14.3)	5 (7.2)	14 (21.9)	0.02	18 (15.3)	1 (6.7)	0.70
Asymmetric vertebral artery	34 (25.6)	14 (20.3)	20 (31.3)	0.17	31 (26.3)	3 (20.0)	0.76
Significant stenosis	23 (17.3)	10 (14.5)	13 (20.3)	0.49	21 (17.8)	2 (13.3)	>0.99
Carotid artery stenosis	14 (10.5)	7 (10.1)	7 (10.9)	>0.99	13 (11.0)	1 (6.7)	>0.99
Intracranial lesion	10 (7.5)	3 (4.3)	7 (10.9)	0.20	8 (6.8)	2 (13.3)	0.31
Cerebral perfusion scintigraphy	9 (6.8)	5 (7.2)	4 (6.3)	>0.99	8 (6.8)	1 (6.7)	>0.99
White matter hyperintensities				0.25			0.46
Fazekas 0–1	54 (40.6)	33 (47.8)	21 (32.8)		47 (39.8)	7 (46.7)	
Fazekas 2	54 (40.6)	25 (36.2)	29 (45.3)		49 (41.5)	5 (33.3)	
Fazekas 3	25 (18.8)	11 (16.0)	14 (21.9)		22 (18.7)	3 (20.0)	

Data are presented as n (%). MRI, magnetic resonance imaging.

carotid/cerebrovascular lesions and those with different severity levels of WMH (*Figure 1* and *Table 3*). However, rScO2 was lower in patients who experienced postoperative cerebral infarction than in patients without cerebral infarction: baseline (no cerebral infarction: 71%±4.8% *vs.* cerebral infarction: 69%±3.4%, P=0.04); before CPB (no cerebral infarction: 67%±5.5% *vs.* cerebral infarction: 65%±5.4%, P=0.04); after initiating CPB (no cerebral infarction: 81%±5.4% *vs.* cerebral infarction: 78%±5.9%, P=0.02); before circulatory arrest (no cerebral infarction: 80%±6.2%, *vs.* cerebral infarction: 76%±6.8%, P=0.02); after initiating selective cerebral perfusion (no cerebral infarction: 81%±5.6% *vs.* cerebral infarction: 78%±7.1%, P=0.02); and lowest value during selective cerebral perfusion (no cerebral infarction 66%±6.3% *vs.* cerebral infarction: 60%±4.6%, P<0.001). In contrast, patients with delirium tended to have higher rScO2 after initiating CPB (no delirium: 80%±6.0% *vs.* delirium: 82%±4.9%, P=0.09), before circulatory arrest (no delirium: 78%±6.9% *vs.* delirium: 81%±5.4%, P=0.01), and after initiating selective antegrade cerebral perfusion (no delirium: 80%±6.4% *vs.* delirium: 82%±5.2%, P=0.08).

Discussion

The incidence of cerebral infarction and delirium in patients undergoing aortic arch replacement was 11.3% and 48.1%, respectively. Preoperative MRI showed that 21.1% of the patients with aortic arch aneurysm had old infarction. The prevalence of carotid artery stenosis, hypoplastic vertebral artery, and WMH with Fazekas scale 3 were 10.5%, 25.6%, and 18.8%, respectively. Preoperative MRI findings were not associated with changes in intraoperative rScO2 or postoperative neurological outcomes. However, rScO2 was significantly lower in patients with cerebral infarction and slightly higher in patients with delirium after CPB initiation.

Previous reports have shown that patients undergoing cardiac surgery without significant carotid artery stenosis have a perioperative stroke incidence of <2%, whereas the risk increases up to 3% in patients with unilateral arterial stenosis of >50% and 5% in patients with bilateral arterial stenosis of >50% (6). Furthermore, a history of transient ischemic attack or cerebral infarction is associated with an 8.5% frequency of perioperative stroke compared with 2.2% in neurologically asymptomatic patients (7). However, the present study did not show a higher incidence of cerebral infarction in patients with carotid artery or intracranial artery stenosis. The major difference between this study and the previous study was that this study focused on patients undergoing aortic arch replacement, characterized by the use of systemic hypothermia and selective antegrade cerebral perfusion for cerebral protection during CPB.

Intraoperative changes in rScO2 evaluated by NIRS

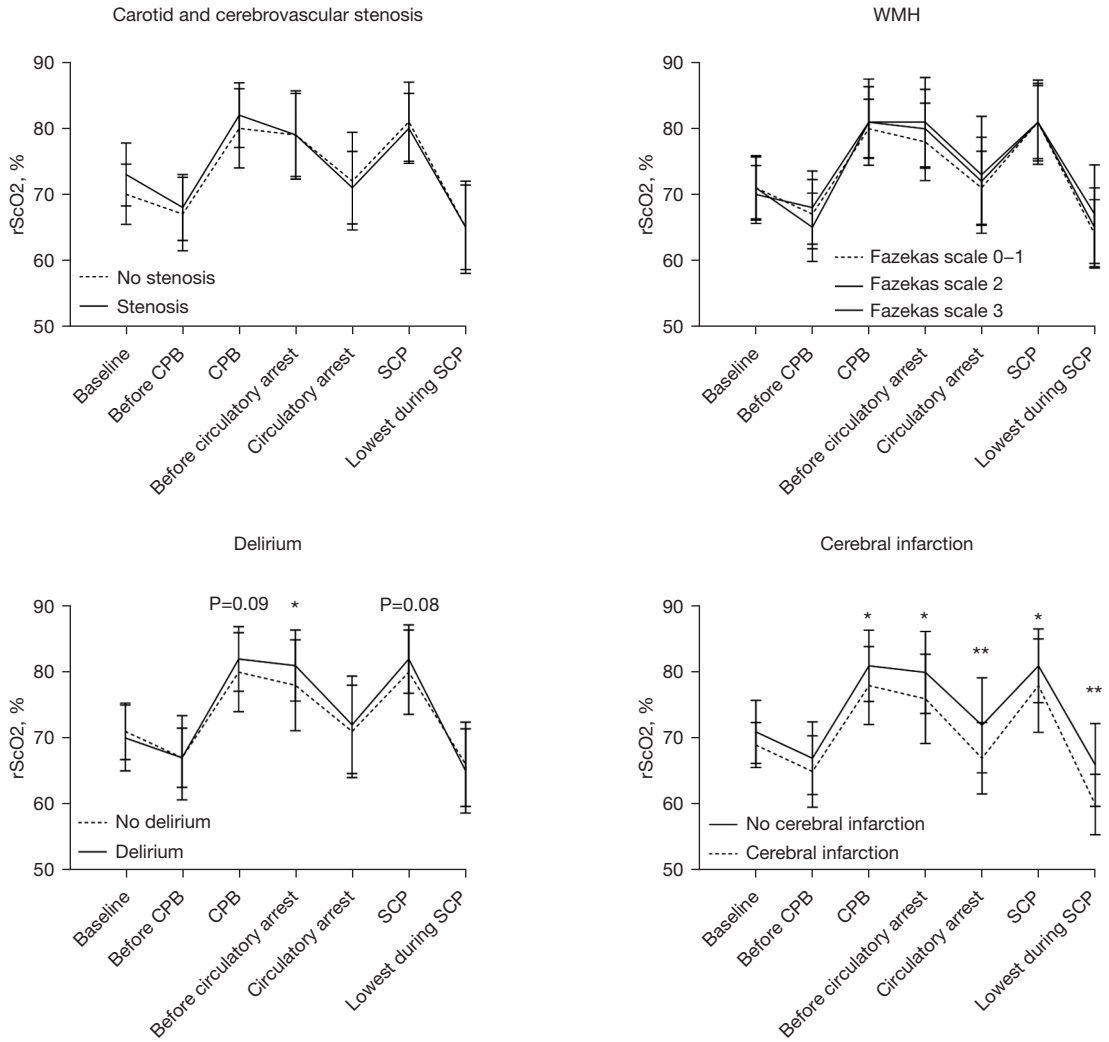

Figure 2 Changes in regional cerebral oxygen saturation measured by near infrared spectroscopy at different phase of surgery. Patients with cerebral infarction after surgery had significantly lower regional cerebral oxygen saturation after initiation of cardiopulmonary bypass. *, P<0.05; **, P<0.01. rScO2, regional cerebral oxygen saturation; CPB, cardiopulmonary bypass; SCP, selective cerebral perfusion; WMH, white matter hyperintensities.

showed a similar pattern in patients with and without significant stenosis of the carotid and intracranial arteries. The initial increase in rScO2 after CPB initiation suggested the effect of an increase in arterial blood oxygen tension and systemic flow from the CPB (8-10). The increase in rScO2 after selective antegrade cerebral perfusion suggested cerebral perfusion recovery with perfusion pressure of 100–150 mmHg. The slight decline after selective cerebral perfusion suggested flow adjustment by the cerebral autoregulation mechanism. This has been reported in previous studies, which showed a positive correlation between rScO2 and cerebral blood flow (11,12). Slightly higher rScO2 after CPB initiation may suggest the reason for the development of delirium, which may be due to hyperperfusion of the cerebral tissue associated with dysregulation in the cerebral autoregulation mechanism (13,14).

rScO2 was significantly lower intraoperatively in patients with postoperative cerebral infarction. Since many of the cerebral infarctions showed multiple lesions on MRI, lower rScO2 in patients with cerebral infarction may suggest hypoperfusion or multiple emboli intraoperatively. Since rScO2 measurements were not significantly

Table 3 Changes in regional cerebral oxygen saturation at different phase of surgery

Phase of surgery	Carotid/cerebrovascular stenosis			WMH			Delirium			Cerebral infarction		
	No	Yes	P	Fazekas 0–1	Fazekas 2	Fazekas 3	P	No	Yes	P	No	Yes	P
Baseline (%)	70±4.6	73±4.8	0.97	71±4.9	70±4.4	71±4.7	0.46	71±4.3	70±5.0	0.57	71±74.8	69±3.4	0.04
Before CPB (%)	67±5.6	68±5.0	0.83	67±5.3	68±5.6	65±5.2	0.09	67±6.4	67±4.5	0.63	67±5.5	65±5.4	0.04
After CPB (%)	80±5.6	82±4.9	0.92	80±4.5	81±6.6	81±5.4	0.66	80±6.0	82±4.9	0.09	81±5.4	78±5.9	0.02
Before circulatory arrest (%)	79±6.3	79±6.7	0.52	78±5.9	81±6.8	80±6.0	0.10	78±6.9	81±5.4	0.01	80±6.2	76±6.8	0.02
Circulatory arrest (%)	72±7.4	71±5.5	0.24	71±5.6	73±8.9	72±6.7	0.51	71±7.0	72±7.4	0.70	71±4.8	69±3.4	0.003
Initiation of SCP (%)	81±6.3	80±5.3	0.32	80±5.6	81±6.4	81±5.9	0.90	80±6.4	82±5.2	0.08	81±5.6	78±7.1	0.02
Lowest during SCP (%)	65±6.4	65±7.0	0.56	64±5.2	67±7.5	65±6.0	0.02	66±6.4	65±6.4	0.43	66±6.3	60±4.6	<0.001

Data are presented as mean ± standard deviation. WMH, white matter hyperintensities; CPB, cardiopulmonary bypass; SCP, selective cerebral perfusion.

different between patients with and without preoperative cerebrovascular lesions, it may be suggested that hemodynamic management based on NIRS may be more important than preoperative evaluation of cerebrovascular lesions in preventing cerebral infarction. Similarly, a previous report from Holmgaards *et al.* reported that time below 10% from baseline rScO2 was significantly longer for patients with new cerebral lesions compared to patients without cerebral infarction during cardiac surgery (5). Olsson *et al.* have also reported lower rScO2 in the affected hemisphere during selective cerebral perfusion in patients who developed stroke postoperatively (15). The use of NIRS in detecting malposition of the cannula has also been suggested previously; thus, the correction of NIRS-guided cerebral perfusion catheters may be a preventative measure for cerebral hypoperfusion in patients undergoing aortic arch replacement (16).

Retrograde cerebral perfusion is another method often used for cerebral protection (17). It may be an effective method, especially for patients with significant arterial stenosis, in which antegrade perfusion may be insufficient to perfuse the cerebral tissue. However, as reported by Tanaka *et al.*, the safe duration of retrograde cerebral perfusion with deep hypothermic cardiac arrest was up to 30 min, and other options were to be considered if circulatory arrest time was expected to exceed 60 min (18). In our institution, reconstruction of the aortic arch branches was performed after distal and proximal anastomosis; thus, an alternative option rather than retrograde cerebral perfusion was considered appropriate. Furthermore, the present study showed that the preoperative existence of carotid and intracranial lesions did not affect changes in rScO2. The incidence of postoperative delirium and cerebral infarction was also not affected in patients undergoing selective antegrade cerebral perfusion, suggesting selective antegrade cerebral perfusion is an effective and acceptable method for cerebral protection in patients with cerebrovascular lesions.

This study had some limitations. rScO2 can be affected by multiple factors, including skin color, hypercapnia, hemoglobin level, and the placement of NIRS probes. Although PaCO2 and hemoglobin levels were not significantly different in patients with and without neurological deficits, further analysis is needed to confirm the result. Second, the definition of cerebral infarction in this study was a symptomatic stroke; thus, a silent stroke, which can only be detected on MRI, was not included. However, symptomatic stroke has a more clinical impact on the patients; thus, the present classification may be

acceptable for the evaluation of clinical outcomes. Third, preoperative cognitive function may be an important confounding factor in the present study. We started the evaluation of preoperative cognitive function in 2021; thus, we only have data for 70 patients. Based on the data from these 70 patients, the Mini-Mental State Examination (MMSE) was not significantly different in patients with and without cerebral infarction {cerebral infarction: 27 [24–30] *vs.* no cerebral infarction: 29 [26–30] *vs.* P=0.22}. However, MMSE was significantly different in patients with and without delirium {delirium: 27 [25–30] *vs.* no delirium: 29 [27–30], P=0.049}. Further studies are needed to confirm this result. Fourth, due to the enrollment period, data on long-term outcomes were unavailable. Moreover, due to the study design, this study only showed the association between rScO2 and postoperative cerebral infarction. Further studies are needed to confirm whether NIRS-guided management of perioperative hemodynamics can reduce the risk of postoperative neurological dysfunction.

Conclusions

A high incidence of cerebral and carotid artery lesions was observed in patients with aortic arch aneurysm. Although preoperative lesions were not significantly associated with the incidence of neurological dysfunction and changes in rScO2, significantly lower rScO2 was observed intraoperatively in patients with postoperative cerebral infarction.

Acknowledgments

We would like to thank Editage (www.editage.jp) for English language editing.
Funding: This study was funded by JSPS KAKENHI [JP21K16500 (DH)] from the Ministry of Education, Culture, Sports, Science and Technology, Japan.

Footnote

Reporting Checklist: The authors have completed the STROBE reporting checklist. Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-78/rc

Data Sharing Statement: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-78/dss

Peer Review File: Available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-78/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jtd.amegroups.com/article/view/10.21037/jtd-24-78/coif). A.Y. received consulting fee from Japan Lifeline Co. Ltd. The other authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved. This study was conducted in accordance with the Declaration of Helsinki (as revised in 2013). It was approved by the institutional ethics board of Saitama Medical Center, Jichi Medical University (No. S23-040), and individual consent for this retrospective analysis was waived.

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Cite this article as: Hori D, Yamamoto T, Kimura N, Yamaguchi A. Prevalence of carotid artery stenosis and intracranial lesions in patients with aortic arch aneurysm and its association with intraoperative regional cerebral oxygen saturation and postoperative neurological outcomes. J Thorac Dis 2024;16(5):2713-2722. doi: 10.21037/jtd-24-78