Original
Article

Prolonged Antegrade Cerebral Perfusion via Right Axillary Artery (≥ 60 min) Does Not Affect Early Outcomes in a Repair of Type A Acute Aortic Dissection

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Purpose: We aim to investigate whether the duration of antegrade cerebral perfusion (ACP) via right axillary artery with an 8-mm prosthetic graft affects early outcomes in a repair of type A acute aortic dissection (AAD).

Methods: Over the 24 months from April 2010, a repair of AAD under ACP via the right axillary artery and mild hypothermic circulatory arrest (rectum temperature, $28\text{--}30^{\circ}\text{C}$) was performed in 34 patients. Mean age was 64.5 ± 13.7 years of age. Preoperative shock status was in three due to cardiac tamponade. Organ malperfusion occurred in 11 patients preoperatively. Mean follow-up period was 9.6 ± 8.4 months and follow-up rate was 100%.

Results: Hospital mortality rate was 8.8%. No newly required hemodialysis and new onset of temporary or permanent neurologic deficits were present in survivors. There were no statistically significant differences of mortality rate, new onset of permanent or temporary neurologic deficits and distal organ dysfunction between ACP duration <60 min and ≥ 60 min. The 12-month survival was $84.4\% \pm 6.4\%$. And, freedom from aorta-related events at 12 and 18 months were $100\% \pm 0.0\%$ and $88.9\% \pm 10.5\%$, respectively.

Conclusions: The duration of ACP via right axillary artery does not affect early outcomes following a repair of AAD.

Keywords: antegrade cerebral perfusion, type A acute aortic dissection, right axillary artery perfusion

Introduction

Type A acute aortic dissection (AAD) is still a devastating disease, which mortality rate following medical managements reaches to 50% within the first 48 h.¹⁾ The

mortality rate associated with AAD after surgery has decreased, but it reached to roughly 20% worldwide.²⁾ In Japan, hospital mortality rate after an emergency surgery for AAD was 9.2% in 2010.³⁾

Antegrade cerebral perfusion (ACP) is one of the techniques for cerebral protection during proximal aortic surgery. This technique has been applied to a repair of AAD and effective circulation management could be achieved by Comas et al.²⁾ Controversy that which arterial cannulation site is optimal in a repair of AAD exists.^{4,5)} Femoral artery is the usual cannulation site for cardiopulmonary bypass in a repair of AAD,⁶⁾ on the other hand, the retrograde perfusion via the femoral artery causes retrograde thromboembolism from atheromatous debris in the thoracic and

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abdominal aorta or intimal dissection associated with malperfusion.^{4,6} In contrast, the use of the right axillary artery can provide not only antegrade aortic perfusion during cardiopulmonary bypass but also continuous antegrade cerebral perfusion during circulatory arrest.^{4,6,7} Our current strategy of arterial perfusion is the right axillary artery perfusion with an 8-mm prosthetic graft in a repair of AAD. In this study, we aim to investigate operative outcomes in our strategy of arterial perfusion in a repair of AAD and whether the duration of ACP affects early outcomes.

Materials and Methods

This study was approved by the institutional review board at our hospital.

Over the 24 months from April 2010, a repair of AAD under ACP via the right axillary artery and mild hypothermic circulatory arrest (rectum temperature, 28°C–30°C) was performed in 34 patients. What is important point in our surgical strategy for a repair of AAD included is perfusion via right axillary artery with an 8-mm prosthesis because this technique can easily establish ACP with clamping the brachicephalic artery. Other routine strategy includes (1) ACP using cold blood; (2) mild hypothermic circulatory arrest at rectal temperature 28°C (total aortic arch replacement) or 30°C (hemiarch replacement).

We retrospectively analyzed these 34 patients. Mean age was 64.5 ± 13.7 years of age and 21 males (62%) were present. Five patients (15%) were aged greater than 80 years of age. Preoperative characteristics are summarized in **Table 1**. Twenty-one patients (71%) had a history of hypertension. Coronary artery disease was present in three patients (9%). The patients who underwent prior cardiovascular surgery were not recognized in this study. We did not experience a repair of AAD for Marfan syndrome patients.

The details of AAD are summarized in **Table 1**. Thrombosed type was present in six patients (18%). Preoperative shock status was in three due to cardiac tamponade. Despite hemodynamic instability, right axillary artery perfusion was performed for these three patients. Organ malperfusion occurred in 11 patients preoperatively. For malperfusion of lower extremity, femoral artery perfusion with 8-mm prosthesis was performed in addition to axillary artery perfusion. Regarding neurologic deficits, we encountered hemiplegia in four and dysphasia in one preoperatively. The cause of these deficits was carotid artery occlusion associated with extensive dissection. Transthoracic echocardiography showed

Table 1 Preoperative profile of patients (n = 34)

Variables	N (%) or Mean $\pm$ SD
Age	64.5 $\pm$ 13.7
Hypertension	24 (71)
Diabetes mellitus	4 (12)
Hyperlipidemia	7 (21)
COPD	0 (0)
Previous stroke	4 (12)
Creatinine $\geq$ 1.5 mg/dL	4 (12)
Hemodialysis	2 (6)
CAD	3 (9)
PAD	1 (3)
Atrial fibrillation	3 (9)
Congestive heart failure	2 (6)
Prior cardiac surgery	0 (0)
Prior aortic surgery	0 (0)
Details of acute type A aortic dissection	
De Bakey Type I	27 (79)
Type II	7 (21)
Thrombosed type	6 (18)
Shock status	3 (9)
Organ malperfusion	
AA occlusion	1 (3)
Unilateral RA occlusion	2 (6)
Bilateral EIA occlusion	3 (9)
Unilateral EIA occlusion	5 (15)
Neurologic deficit	
Hemiplegia	4 (12)
Dysphasia	1 (3)
Echocardiographic data	
LVEF (%)	60.1 $\pm$ 9.6
Aortic regurgitation	
Moderate	5 (15)
Severe	3 (9)

AA: axillary artery; CAD: coronary artery disease; COPD: chronic obstructive pulmonary disease; EIA: external iliac artery; LVEF: left ventricular ejection fraction; PAD: peripheral artery disease; RA: renal artery; SD: standard deviation

the degree of aortic regurgitation more than mild in five (15%) and more than moderate in three (9%). A left ventricular ejection fraction rate was normal.

Mean follow-up period was 9.6 ± 8.4 months and follow-up rate was 100%. All patients are followed up at our outpatient clinic.

Surgical techniques

An emergency surgery is to be performed after a diagnosis of acute type A aortic dissection confirmed by computed tomography. Cardiologists assess cardiac function such as left ventricular function, degree of aortic regurgitation or cardiac tamponade on transthoracic echocardiography. We, cardiac surgeons, prepare an

emergency surgery and assess if the patients remain hemodynamically unstable. If not, we choose the right axillary artery which is anastomosed an 8-mm prosthetic graft as an arterial cannulation. On the other hand, if so, we alternatively do femoral artery or left ventricular apex as the perfusion site. General anesthesia is introduced and two arterial monitorings are placed routinely in both the right radial artery and dorsalis pedis artery. Swan-Ganz catheter and transesophageal cardiographic probe are placed. To brain oxygenation, near infrared spectroscopy is applied in all patients. At first, we dissect the right axillary artery via deltoideopectoral approach⁶⁾ and tape up it. Heparin is given intravenously and right axillary artery is clamped after activated clotting time longer than 250 s. We confirm that the right axillary artery is pulsatile and not dissected and an 8-mm prosthetic graft (Triplex; Terumo Corp, Tokyo, Japan) is anastomosed in an end-to-side fashion. If the artery dissection is present, we change an arterial cannulation site (femoral artery or left ventricular apex). In case of malperfusion of lower extremity, femoral artery perfusion is made in addition to axillary artery perfusion.

After a standard median sternotomy and opening of pericardium, the right atrium is cannulated with a standard two-staged venous cannula. Cardiopulmonary bypass is established and the patients are gradually cooled down to a rectal temperature of 30°C (a case of hemiarch replacement) or 28°C (a case of total aortic arch replacement). Left ventricular venting tube and retrograde cardioplegic cannula are inserted via the right pulmonary vein and coronary sinus, respectively. During the cooling time, the ascending aorta is dissected and is cross-clamped, followed initiation of retrograde perfusion of cold blood cardioplegia. Cardioplegia perfuses every 20 min via coronary sinus. The ascending aorta is transected. We eliminate fresh thrombus in the false lumen as we can as possible and biological glue is directly injected in the false lumen to fix the aortic wall. Finally, ascending aorta is reinforced and reconstructed using inner and outer Teflon felt strips. If needed, we added the commissural resuspension for aortic valve detachment causing aortic regurgitation. After the completion of proximal aortic reconstruction and achievement of targeted rectal temperature, circulatory arrest is initiated and the cross-clamped aorta is opened. For cold antegrade cerebral perfusion (20°C), brachiocephalic artery is cross-clamped and additional selective antegrade cerebral perfusion cannulas (12 Fr) are inserted via the

left carotid artery and the left subclavian artery. At inserting them, we snare the neck vessels to secure the cannulas during surgery. Cold cerebral perfusion flow is adjusted at 10 to 15 ml/kg/min and total flow is less than 1000 ml/min. Our surgical strategy is to perform hemiarch replacement to exclude intimal tear. When intimal tear cannot be detected in the ascending aorta or younger patients age 65 years or less, we perform total aortic arch replacement. In both procedures, the descending aorta is reinforced using inner and outer Teflon felt strips. The open distal anastomosis is completed using side-branched prosthesis (Triplex; Terumo Corp, Tokyo, Japan) in hemiarch replacement. In total aortic arch replacement, the elephant trunk technique is applied in all patients, subsequently 4-branched prosthesis (Triplex; Terumo Corp, Tokyo, Japan) is anastomosed to an elephant trunk. The lower body circulation is restarted via side branch of the prosthesis and whole body rewarming is also started in hemiarch replacement. In total arch replacement, body rewarming is started following completion of the left carotid artery reconstruction. After the proximal anastomosis is performed, the graft clamp is released and coronary perfusion is restarted in a retrograde fashion. Then, the patients are weaned from cardiopulmonary bypass.

Definition of complications

We defined the postoperative neurologic deficits and renal dysfunction as follows in accordance with a previous report. The permanent neurologic deficits were defined as the new and persisted deficits at discharge confirmed on computed tomography or magnetic resonance imaging. The temporary neurologic deficits were defined as postoperative confusion, delirium, obtundation, or transit focal deficits with negative evidence on computed tomography or magnetic resonance imaging.⁸⁾ The permanent or temporary neurologic deficits caused by preoperative brain malperfusion, hemodynamic instability, or atrial fibrillation were excluded in this study.

Renal dysfunction was defined as postoperative elevated creatinine level greater than 1.5 mg/dL. Patients with preoperative elevated creatinine level or a history of hemodialysis were excluded.

Statistical analysis

All statistical analyses were performed with StatView version 5.0 software (SAS Institute, Cary, NC). Categorical variables were analyzed using a χ^2 test and are expressed as percentages. Continuous variables were

analyzed by Student's paired or unpaired *t* tests and are expressed as the mean $\pm$ standard deviation. The Kaplan-Meier method was used to estimate survival rates.

All *p* values ≤ 0.05 were considered statistically significant.

Results

All patients underwent a repair of AAD in emergency status under ACP and mild hypothermic circulatory arrest. The right axillary artery perfusion with an 8-mm prosthesis was chosen in 24 patients (71%), and axillary artery plus femoral artery perfusions in 10 (29%) because of presence of lower extremity malperfusion ($n = 8$) and lack of total cardiopulmonary bypass flow ($n = 2$). We performed hemiarch replacement in 19 patients (56%) and total aortic arch replacement in 15 (44%) based on patient's age and location of intimal tear. Concomitant surgical procedures are summarized in **Table 2**. Aortic valve suspension was performed in 11 patients for detachment of aortic valves. The mean durations of ACP were 79.2 ± 48.0 . The minimum rectal and nasopharyngeal temperatures were $28.0^\circ\text{C} \pm 0.8^\circ\text{C}$ and $26.6^\circ\text{C} \pm 1.2^\circ\text{C}$, respectively. At my hospital, esophageal or tympanic temperature intraoperatively was not measured. There were no patients who suffered from complications associated with axillary perfusion.

Early outcomes

Hospital mortality rate was 8.8% (3/34). Postoperative morbidities are summarized in **Table 3**. Re-exploration for bleeding was required in two patients (5.9%). Two patients had elevation of creatinine level ≥ 1.5 mg/dL postoperatively, but no newly required hemodialysis was present. New onset of permanent or temporary neurologic deficits was also not recognized in survivors. In eight patients who suffered from lower extremity malperfusion, we have not experience amputation of lower extremity or paraplegia/paraparesis. Seven survivors were discharged on foots, meanwhile one with preoperative right hemiplegia due to brain malperfusion was referred to a private hospital postoperatively.

Next, to investigate whether right axillary artery perfusion is safe for neurologic protection and distal organ perfusion in a repair of AAD, we analyzed early outcomes by dividing subjects into two subgroups as follows; ACP duration < 60 min and ≥ 60 min. The detailed information is summarized in **Table 4**. There was no statistically significant difference of mortality rate between two groups ($p = 0.4525$). In two subgroups, new onset of permanent

Table 2 Intraoperative findings (n = 34)

Variables	N (%) or Mean $\pm$ SD
Aortic perfusion site	
AA with graft	24 (71)
AA with graft + FA cannulation	10 (29)
Surgical procedures	
Hemiarch replacement	19 (56)
Total aortic arch replacement	15 (44)
Concomitant procedures	
Aortic valve suspension	11 (32)
Valve-sparing operation	3 (9)
Bentall operation	1 (3)
Tricuspid annuloplasty	1 (3)
ACP	34 (100)
ACP time (min)	79.2 ± 48.0
Cardiopulmonary bypass time (min)	214.2 ± 64.3
Aorta cross clamp time (min)	158.2 ± 58.1
Minimum rectum temp ($^\circ\text{C}$)	28.0 ± 0.8
Minimum nasopharygeal temp ($^\circ\text{C}$)	26.6 ± 1.2

AA: axillary artery; FA: femoral artery; ACP: antegrade cerebral perfusion; SD: standard deviation; temp: temperature

Table 3 Early outcomes (n = 34)

Variables	N (%) or Mean $\pm$ SD
Mortality rate	3 (8.8)
ICU stay (days)	11.2 ± 18.3
Ventilator (hrs)	48.9 ± 64.8
Morbidity	
Neurologic deficits	0
Creatinine ≥ 1.5 mg/dL	2 (5.9)
Newly required HD	0
Re-exploration for bleeding	2 (5.9)
Pneumonia	1 (2.9)
Gastrointestinal bleeding	1 (2.9)

HD: hemodialysis; ICU: intensive care unit; SD: standard deviation

or temporary neurologic deficit did not occur. With regard to deterioration of renal function, which was a sign of distal organ perfusion, there was no statistical difference in two subgroups.

Short-term outcomes

The 1-year survival was $84.4\% \pm 6.4\%$ (**Fig. 1**). We experienced two deaths and the causes of deaths included disseminated intravascular coagulopathy and massive cerebral hemorrhage. No aorta-related death was present during follow-up period. These events occurred about

Table 4 Postoperative outcomes between antegrade cerebral perfusion <60 and ≥60 minutes

Variables	N (%) or Mean ± SD		p value
	ACP <60 (n = 19)	ACP ≥60 (n = 15)	
Mortality (%)	1 (5.3)	2 (13.3)	0.4525
Ventilator (hrs)	38.7 ± 52.7	61.7 ± 77.6	0.1726
ICU stay (days)	10.4 ± 13.7	12.2 ± 23.4	0.7770
Neurologic deficits	0	0	0.5000
Renal dysfunction	0	2 (13.3)	0.1239
Newly required HD	0	0	0.5000

HD: hemodialysis; ICU: intensive care unit; ACP: antegrade cerebral perfusion; SD: standard deviation

Fig. 1 (A) Kaplan-Meier curve showing survival. (B) Freedom from aorta-related events.

three months later following discharge on foot. Freedom from aorta-related events at 12 and 18 months were $100\% \pm 0.0\%$ and $88.9\% \pm 10.5\%$, respectively (**Fig. 1**). Sixty-nine male suffered from aorta-esophageal fistula, which occurred about 18 months later after discharge. He underwent at an initial surgery total aortic arch replacement. This patient underwent thoracic endovascular aortic repair for the descending aorta communicating esophageal, following resection of esophageal and reconstruction of that at two-staged surgery. However, stent graft infection occurred, which was refractory to antibiotics treatment. Therefore, we performed redo total aortic arch and descending aorta replacement after removal of infected stent graft. The patient is now under antibiotics treatment in a good condition.

Discussion

We described our surgical strategy in a repair of AAD and operative outcomes. The important point of our

policy was arterial perfusion via the right axillary artery with an 8-mm prosthesis excepting hemodynamic instability cases or dissection of right axillary artery. With usage of the right axillary arterial perfusion, we can initiate ACP easily by clamping the brachiocephalic artery in addition to establishment of systemic antegrade arterial perfusion. We do not perform the direct cannulation to the artery because we prevent intimal dissection associated with manipulation or achieve appropriate systemic flow sewing an 8-mm prosthetic graft to an axillary artery. In emergency situation, it took about 15 min to complete the graft anastomosis to artery. We did not experience any complications-related to axillary perfusion such as dissection or malperfusion in right upper extremity intraoperatively and postoperatively. Two patients need additional femoral perfusion. It was because body surface area was very large in these two. Mortality rate in the group of axillary arterial perfusion was excellent despite 25% of preoperative malperfusion.⁶⁾

As to antegrade aortic perfusion, an alternative perfusion site is transapical one. Wada and coworkers showed the utility of transapical cannulation in a repair of AAD. There was no case in which conversion case to other arterial cannulation sites, and in all 138 patients sufficient flow in cardiopulmonary bypass could be achieved.⁹⁾ This is the attractive outcome. However, there is a critical complication, bleeding at apex. In particular, in Japanese older female, the bleeding severely affects patients' lives because their apex condition often is very fragile and bleeding cannot be repaired. We have a few cases of experiences in transapical approach in patients with dissected axillary artery confirmed on preoperative computed tomography. Retrograde perfusion via femoral artery offers quick establishment of cardiopulmonary bypass in an emergency status. We sometimes apply this technique to hemodynamically unstable patients. However, we concern the possible complications of retrograde perfusion such as retrograde cerebral embolization or organ malperfusion.^{4,6)} Our policy is that we manage to establish antegrade aortic perfusion as possible as we can because of establishment of antegrade aortic perfusion and prevention of concerned complications.

Cerebral protection we applied in a repair of AAD included ACP as mentioned above and mild hypothermic circulatory arrest (rectum temperature: 28°C–30°C). With regard to ACP, comas et al. described their surgical techniques and results in a repair of AAD. They commented that unilateral ACP with only right axillary artery same as ours was an effective circulatory management for permanent neurologic deficits. They did not apply selective perfusion for left carotid artery and left subclavian artery. Zierer et al. also described that unilateral ACP offered a reduced permanent neurologic deficit rate compared to bilateral ACP.⁵⁾ Meanwhile, Okita et al.¹⁰⁾ used three cannulas for selective ACP into all arch vessels without snaring them because more than 20% of the circle of Willis has been reported to be incomplete in healthy population.¹¹⁾ Merrkola et al. suggested that in case of marked problems in cervical arteries or circle of Willis by preoperative imagings, additional perfusion should be considered.¹¹⁾ However, all patients who suffered from AAD could not perform the preoperative head imagings such as magnetic resonance image. Therefore, we inserted two cannulas to the left carotid artery and left subclavian artery and snared them in a repair of AAD described here and aortic arch aneurysm. Although some authors pointed out the embolism related

to arch vessels manipulation, we did not experience cerebral infarction associated with embolism by manipulation not only in a repair of AAD but also aortic arch aneurysm.^{5,10,12)} Certainly, in a degenerative disease, aortic arch aneurysm, aortic arch wall inside was very atherosclerotic. In that case, we carefully perform the deridement for ostium of aortic arch vessels and washed out the debris around the ositum, following inserting cannulas to each vessel.

In this study, we analyzed the impact of ACP duration on not only neurologic function but also postoperative organ functions during circulatory arrest (**Table 4**). **Table 4** shows that between ACP duration < and ≥60 min, there was no statistically significant difference in postoperative neurologic and organ functions after a repair of AAD. Previous report showed that no significant difference was seen regarding the incidence of postoperative acute renal failure among three ACP duration groups.⁵⁾ The incidence rate of renal failure in duration of ACP ≥60 to 90 min was 8%. Incidence in our study was higher than that by Zierer et al.⁵⁾ However, a case of AAD was excluded in their result. Other reported that ACP duration longer than 90 min was not associated with an increased risk of hospital death or permanent neurologic deficit during a surgery on thoracic aorta including AAD.¹²⁾ Before application of ACP, hypothermic circulatory arrest of 25 min or more was associated with memory and fine motor deficit.¹³⁾ Svensson et al. showed that incidence rate of stroke and hospital mortality markedly recognized after 40 min and 65 min or more of hypothermic circulatory arrest, respectively.¹⁴⁾ Postoperative outcomes strongly depended on the duration of hypothermic circulatory arrest, which was shorter than that reported recently. In view of cerebral metabolism, a predicted safe duration of hypothermic circulatory arrest at esophageal temperature of 15°C was only 29 min.¹⁵⁾ To be short, in hypothermic circulatory arrest alone, the deeper hypothermia could lead to prolonged and safe duration of circulatory arrest. However, deep hypothermia was associated with coagulopathy or lung injury intraoperatively or postoperatively. With ACP, hypothermic circulatory arrest temperature up to 28°C can be increased for safe aortic arch repair for 60 min or less.¹⁶⁾ As to prolonged duration up to 60 min for circulatory arrest under same situation, they could not answer it.

As to temperature during circulatory arrest, we measured temperature at rectum because we understood that it reflect the temperature of spinal cord.

Study Limitations

Present study was retrospective in nature and not a randomized trial. Additionally, study size was relatively smaller than those reported previously. However, we described comparative data based on ACP duration in a repair of AAD by our present approach, and it did not have a negative impact on postoperative outcomes. This finding strongly emphasized importance and safety of ACP with axillary arterial perfusion.

Conclusions

Operative outcomes in a repair of AAD in a current fashion were acceptable. This study described that the duration of ACP via right axillary artery with an 8-mm prosthetic graft did not affect early outcomes in a repair of AAD.

Disclosure Statement

Naoto Fukunaga and co-workers have no conflict of interest.

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