의학석사 학위논문

뇌사자 동종 이식편을 이용한 혈관 재건술의 장기간 예후와 형태학적 변화

Analysis of long-term outcomes and morphologic changes of cadaveric arterial allograft used as a vascular conduit

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이 논문을 의학석사 학위 논문으로 제출함

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Analysis of long-term outcomes and morphologic changes of cadaveric arterial allografts used as vascular conduits

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Abstracts

Background: This study's primary objective was to describe the long-term outcomes and morphologic changes of cadaveric arterial allografts (CAAs) used as vascular conduits, and the secondary objective was to determine the predictors of favorable outcomes of CAAs.

Methods: This retrospective, single-center study included 21 consecutive patients with peripheral artery disease who had undergone bypass surgery using cryopreserved CAAs (c-CAA group) and 34 liver-transplanted patients in whom fresh CAAs were used as aorto-hepatic conduits (f-CAA group). The primary outcomes were aneurysm or occlusion/stenosis (≥50%); the secondary outcomes included CAA dilatation, stenosis (<50%), or wall calcification. The morphologic changes were defined as aneurysm, aneurysmal dilatation, and wall calcification.

Results: During the median follow-up of 55 months (range, 3–137 months), the primary and secondary outcome incidences were 38.2% (21/55) and 16.4% (9/55), respectively, and the incidence of morphologic changes was 20.0% (11/55). There were significant differences in the incidences of the primary outcomes ($p = 0.02$) and morphologic changes ($p < 0.001$) between the c-CAA and f-CAA groups. Multivariable analysis indicated that the use of immunosuppressants was an important, protective, prognostic factor associated with the long-term morphologic changes of CAAs (hazard ratio, 0.07; 95% confidence interval, 0.01–0.42; $p = 0.004$).

Conclusions: Our analysis revealed that a substantial proportion of CAAs was associated with the long-term primary outcomes and morphological changes. Regular imaging studies to evaluate the gradual deterioration and degeneration of CAAs should be considered in these patients.

Keywords: cadaveric arterial allograft, liver transplantation, morphologic change, outcome, peripheral artery disease

Contents

Introduction

Cadaveric arterial allografts (CAAs) are no longer used in general clinical settings because their chronic rejection can result in arterial wall degeneration and deterioration, making them unsuitable for long-term arterial replacement in vascular surgery.[1] However, CAAs have been used for vascular reconstruction in selected conditions—for vascular reconstruction in an infected field, in the absence of suitable autologous vascular conduits, or in deceased-donor transplantation.[2-4] The rejection process of CAAs induces intense remodeling of the arterial wall, medial destruction being the main consequence of chronic rejection in CAA. Arterial walls become unable to counter the force exerted by the blood pressure, resulting in thinning of the media, dislocation of the elastic lamellae, progressive destruction of smooth muscle cells, and infiltration of inflammatory cells into the adventitia.[1] Therefore, medial cell loss, matrix degeneration, and adventitial inflammation indicate immune injury and response in CAAs.[1] Two strategies have been suggested to reduce these arterial changes during rejection: immunosuppression and cryopreservation.[1,5-11] However, there is a lack of evidence supporting the benefits of these management strategies for patients undergoing vascular reconstruction with CAAs.

Given the limited data on the durability of CAAs, the primary objective of this study was to describe the long-term outcomes and morphologic changes of CAAs used as vascular

conduits, and the secondary objective was to determine the predictors of the favorable outcomes of CAAs.

Methods

Study design and populations

This single-center, retrospective, observational study used data extracted from the medical records of patients who had undergone vascular reconstruction using cryopreserved CAAs (c-CAAs) for peripheral artery disease (PAD) or placement of an aorto-hepatic conduit using a fresh CAA (f-CAA) during deceased-donor liver transplantation (LT). Approval for data collection and publication was obtained from the institutional review board at our hospital (IRB No. 2018-0007), which waived the requirement for written informed consent because of the study's retrospective design. All methods were performed in accordance with the relevant guidelines and regulations.

Between January 2010 and December 2018, 79 consecutive patients—33 PAD patients with c-CAAs and 46 deceased-donor LT patients with f-CAAs—were screened for inclusion in this study. In the PAD patients with c-CAAs, ABO blood type compatibility was not an inclusion criterion, and no tissue matching was performed. Exclusion criteria for PAD patients with c-CAAs were: (1) follow-up loss (n = 6), (2) transfer to other hospitals after

operation (n = 3), and (3) no follow-up imaging studies (n = 3). Exclusion criteria for LT patients with f-CAAs were: (1) follow-up loss (n = 1), (2) transfer to other hospitals after operation (n = 2), and (3) early mortality within 1 month after LT (n = 9). A total of 55 patients who had undergone vascular reconstruction using CAAs at our hospital were included in the final analysis. These patients were assigned to the c-CAA group, including four patients with a previous history of organ transplantation (n = 21, 38.2%), or the f-CAA group (n = 34, 61.8%) (**Fig. 1**).

The demographics, risk factors of interest, and clinical outcomes, including the morphologic changes on follow-up imaging studies, of all consecutive patients, were recorded in an Excel database (Microsoft Corp., Redmond, WA, USA) and analyzed retrospectively. Risk factor variables were defined as described previously.[12]

Procurement of arterial allografts from deceased donors and preservation techniques

This study employed human arterial allografts from deceased multi-organ donors. All procedures for CAA procurement and processing were in compliance with Korean legislation (Law 5858/1999 and Law 11976/2013) and conformed to the ethical and safety concerns for therapeutic use.

The CAA was obtained aseptically from each anonymized donor diagnosed with brain death during the course of multi-organ procurement. The preservation techniques are

described in detail elsewhere[13-15] and summarized in **Fig. 2**.

Outcomes of interest and follow-up

The primary outcomes of interest were i) CAA aneurysm, defined as a dilatation or widening of the artery to at least 1.5 times larger than the normal arterial diameter; ii) stenosis, defined as a significant (≥50%) narrowing of the arterial diameter compared with the normal diameter; iii) occlusion. The secondary outcomes included CAA dilatation (<1.5 times larger than the normal diameter), stenosis (<50% narrowing of the arterial diameter), or CAA wall calcification, defined as calcification of the full diameter of the CAA wall. The morphologic changes were defined as aneurysm, aneurysmal dilatation, and CAA wall calcification (**Fig. 3**).

In the c-CAA group, follow-up computed tomographic (CT) scans were scheduled during the postoperative hospital stay, at 6 and 12 months and annually thereafter. In the f-CAA group, postoperative Doppler ultrasonography (DUS) was performed daily in the first week and on a weekly basis during the hospital stay. CT scans were scheduled annually after discharge. Three of four patients with previous history of organ transplantation in the c-CAA group and all patients in the f-CAA group were treated with conventional immunosuppressive therapy after transplantation.[16] For study purposes, all the images were re-reviewed by an experienced vascular surgeon and a dedicated, board-certified radiologist

who were blinded to the original CT scan reports.

Statistical analysis

Categorical variables were analyzed using the chi-squared test or Fisher's exact test, and continuous variables were analyzed by Student's *t*-test or Wilcoxon rank-sum test. Factors associated with clinical outcomes and morphologic changes were analyzed using a Cox proportional hazards model. Variables yielding p-values < 0.1 from the univariable analysis and other clinically important variables were subjected to multivariable analysis, and hazard ratios (HRs) with 95% confidence intervals (CIs) were calculated. Using the Kaplan-Meier method and the log-rank test, we compared the incidence of primary and secondary outcomes, as well as of morphologic changes, between the c-CAA and f-CAA groups. All statistical analysis was performed using the IBM SPSS Statistics for Windows, version 28.0 (IBM Corp., Armonk, NY, USA), and $p < 0.05$ was considered statistically significant.

Results

After all exclusions based on the predetermined criteria, 21 patients in the c-CAA group and 34 patients in the f-CAA group were consecutively enrolled in this study. The baseline and

clinical characteristics of the included patients are presented in **Table 1**. The percentage of females was significantly higher in the f-CAA group than in the c-CAA group ($p = 0.002$). Patients in the c-CAA group were significantly more likely to have hypertension ($p = 0.04$), dyslipidemia ($p = 0.05$), and a history of smoking ($p = 0.04$) than those in the f-CAA group. In contrast, patients in the f-CAA group had a higher prevalence of chronic kidney disease than those in the c-CAA group ($p = 0.09$). With regard to medication, all patients in the f-CAA group received an immunosuppressant ($p < 0.001$), and the proportion of statin use was higher in the c-CAA group ($p = 0.02$).

Table 2 shows the indications and anatomic locations of the CAA. Previous operation site infection and mycotic aneurysms were identified as the common cause of CAA use in 71.4% (15/21) of cases in the c-CAA group, and the anatomic locations of vascular reconstruction were above-knee (47.6%) and the aorto-iliac (33.3%) segments. In the f-CAA group, all patients had undergone placement of an aorto-hepatic conduit using a CAA during deceased-donor LT due to inadequate recipient hepatic artery.

During the median follow-up of 55 months (range, 3–137 months), the primary and secondary outcome incidences were 38.2% (21/55) and 16.4% (9/55), respectively, and the incidence of morphologic changes was 20.0% (11/55) (**Table 3**). During the mean follow-up periods of 50.7 ± 40.0 and 63.4 ± 41.2 months in the c-CAA and f-CAA groups, respectively, the primary outcome incidence was significantly higher in the c-CAA group (57.1% vs. 26.5%, $p = 0.02$),

whereas the secondary outcome incidence was similar between the two groups (23.8% vs. 11.8%, $p = 0.28$). Secondary intervention was more frequently performed in the c-CAA group (33.3% vs. 0%, $p < 0.001$). The rates of long-term morphologic changes were significantly higher among patients in the c-CAA group than in the f-CAA group (42.9% vs. 5.9%, $p < 0.001$). **Supplementary Fig. 1** shows the operative findings and the serial CT scans of a 65-year-old man without morphologic changes of the c-CAA, after aneurysm resection and c-CAA interposition due to a mycotic abdominal aortic aneurysm. **Supplementary Fig. 2** shows the CT scan, the pathologic specimen of the resected aneurysm, and histopathological findings in a 67-year-old man with aneurysm of the CAA at 12 years after femoro-popliteal arterial bypass with a c-CAA due to recurrent prosthetic graft infection.

Kaplan–Meier survival analysis showed that there were significant differences in the incidences of primary outcomes ($p = 0.01$) and morphologic changes ($p < 0.001$) between the c-CAA and f-CAA groups (**Fig. 4**). The cumulative incidences of the primary outcomes at 6 months and 1, 3, and 5 years were 23.8%, 38.1%, 61.9%, and 81.0%, respectively, in the c-CAA group and 2.9%, 20.6%, 35.3%, and 67.6%, respectively, in the f-CAA group. The cumulative incidences of the morphologic changes at 6 months and 1, 3, and 5 years were 4.8%, 4.8%, 47.6%, and 66.7%, respectively, in the c-CAA group and 2.9%, 14.7%, 23.5%, and 58.8%, respectively, in the f-CAA group.

There was no significant factor associated with an increased risk of primary and secondary

outcome incidence (**Table 4**). After the adjustment for potential confounding variables, multivariable analysis indicated that the use of immunosuppressants was an important protective prognostic factor associated with the long-term morphologic changes of CAA (HR, 0.07; 95% CI, 0.01–0.42; p = 0.004) (**Table 5**).

Discussion

In the current study, we aimed to report the long-term clinical outcomes of CAAs used as vascular conduits in various indications and anatomic locations to address the unavailability of data on the durability of CAA. In addition, given that the occurrence of early occlusion (within 30 days) of the CAA varies between 0 and 17%[17–21] due to multiple factors, we also evaluated the long-term morphologic changes of CAA, excluding stenosis or thrombotic occlusion due to technical factors, to ensure valid assessments of long-term gradual deterioration and degeneration of CAAs. The results of our current study showed that the incidences of the primary outcome and morphologic changes were remarkably high among patients who used CAAs as vascular conduits. Additionally, we found that the use of immunosuppressants was the only protective prognostic factor associated with the long-term morphologic changes of CAA. However, because of the small heterogeneous study sample consisting of both c-CAA and f-CAA, we cannot draw conclusions, and our results should

be considered as hypothesis-generating rather than definitive.

CAAs were the first material to be widely used as vascular conduits[22] but are no longer used in general clinical settings because their chronic rejection can induce arterial wall degeneration and deterioration, resulting in arterial wall dilation and rupture. Therefore, they are considered unsuitable for long-term arterial replacement in vascular surgery.[1] However, CAAs can be an alternative option for vascular reconstruction in infected fields, in the absence of a suitable autologous vascular conduit, or in deceased-donor transplantation.[2-4]

Two strategies have been suggested to reduce both morphological and biomechanical changes in CAAs in case of rejection: immunosuppression to reduce the immunologic response of the host and cryopreservation to reduce the allograft antigenicity.[1,5-8,15] Animal experiments suggest that the use of a low-maintenance dose of immunosuppressants prevents aneurysmal changes in arterial allografts. In our study, the use of immunosuppressants was a protective prognostic factor associated with the long-term morphologic changes of CAA. However, immunosuppression can also induce potentially serious adverse effects in non-transplanted elderly and critically ill patients.[5,15] Various preservation techniques have been introduced,[9-11] but there is a lack of evidence supporting the benefits of these management strategies for patients undergoing vascular reconstruction using CAA.[1,5] Cryopreservation can reduce allograft antigenicity by decellularization and improve the permanence of allografts compared to fresh allografts, which are known to have

an even higher risk for complications.[7,23,24] Although costs limit the more widespread use of CAAs, c-CAAs are more easily available than f-CAAs. Allograft decellularization can result in the qualitative and quantitative preservation of the medial elastin network, as well as in the suppression of adventitial inflammatory cell infiltration into allografts.[1,5-8] Moriyama et al.[11] reported that cryopreservation treatment did not suppress the antigenicity of animal tissue, and the extent of immunologic response was similar to that found in fresh tissue. However, it is not certain whether cryopreservation suppresses the antigenicity of tissue; further studies on larger cohorts are warranted.

With regard to cryopreservation, limited data are available on the relationship between the quality of allografts and their cryopreservation storage duration.[9] Fiala et al.[10] examined the mechanical and structural properties of human aortic and pulmonary allografts during the first 10 years of cryopreservation and storage and reported that elasticity, stiffness, solidity, and morphology of aortic and pulmonary allografts did not change noticeably with cryopreservation in the first 10 years of cryopreservation and storage. In our tissue bank, the cryopreservation protocol sets the expiration period of CAA to be 10 years. Although we did not analyze the effects of cryopreservation and the duration of storage on clinical outcomes and morphologic changes of CAA, Hidi et al.[9] evaluated whether cryopreservation moderates the thrombogenicity of arterial allografts during storage and found that the hemostatic potential of cryopreserved allografts was retained, although their thrombogenic

potential declined during the 6-month storage. Further studies with larger sample sizes are needed to better understand the impact of cryopreservation period on the outcomes and morphologic changes of cryopreserved allografts.

Study limitations

Several limitations of this study should be noted. Owing to its retrospective and observational design, using single-center registry data from a small number of patients, this study is potentially affected by selection and information biases. A substantial number of critically ill patients with limited life span and short follow-up period who could not be evaluated for the long-term morphologic changes of CAAs were excluded from this study, and detailed clinical information was not available for some of the included patients. Furthermore, several key variables, such as the interval from the procurement of CAAs from deceased donors to the use of cryopreserved CAA (cryopreservation period) and ABO blood type compatibility in the c-CAA group, were not available in our data sources. Although there has been controversy over the impact of ABO blood type compatibility, some authors reported that use of the cryopreserved allograft with donor–recipient ABO compatibility for peripheral arterial bypass had significantly better patency rates.[25,26] We could not include these variables in our analysis. Furthermore, the decisions regarding the use of CAA were mainly made without randomization by physicians, based on the expected level of the

efficacy of the management strategies.

Conclusion

Despite all these limitations and the debate regarding durability of CAA and preservation techniques, CAAs can be an alternative option for vascular reconstruction in selected cases. Considering that aneurysm, aneurysmal dilatation, occlusion/stenosis, and wall calcification are observed in a substantial proportion of CAAs, regular imaging studies should be considered in these patients to evaluate gradual deterioration and degeneration of CAAs.

Tables

Table 1. Baseline and clinical characteristics of the study sample

	c-CAA group* (n = 21)	f-CAA group (n = 34)	p value
Original disease	PAD	ESLD	
Preservation of CAA	Cryopreservation	Fresh	
ABO compatibility	Unknown	Compatible	
Mean age, years	53.6 ± 22.9 (1–75)	52.5 ± 10.9 (18–69)	0.84
Female sex	1 (4.8)	15 (44.1)	0.002
Body mass index, kg/m^2	22.7 ± 4.6	21.5 ± 3.5	0.27
Diabetes mellitus	3 (3.1)	5 (14.7)	1.00
Hypertension	8 (38.1)	4 (11.8)	0.04
Dyslipidemia	3 (14.3)	0 (0)	0.05
History of smoking	12 (57.1)	10 (29.4)	0.04
Infection	11 (52.4)	3 (8.8)	<0.001
CAD	4 (19.0)	3 (8.8)	0.41
CKD	3 (14.3)	12 (35.3)	0.09
Medication			
antithrombotic agent	15 (71.4)	21 (61.8)	0.46
statin	9 (42.9)	4 (11.8)	0.02
immunosuppressant	3 (14.3)	34 (100.0)	<0.001
Follow-up duration (months)	50.7 ± 40.0	63.4 ± 41.2	0.26

Continuous data are presented as means ± standard deviations (range); categorical data are given as n (%).

CAA, cadaveric arterial allograft; CAD, coronary artery disease; c-CAA, cryopreserved

cadaveric arterial allograft; CKD, chronic kidney disease; ESLD, end-stage liver disease; f-CAA, fresh cadaveric arterial allograft; PAD, peripheral artery disease

*Included four patients with previous history of organ transplantation.

Table 2. Indications and anatomic locations of use of CAA

	c-CAA group (n = 21)	f-CAA group (n = 34)
Indication		
Infection	9 (42.9)	-
Mycotic	6 (28.6)	-
Inadequate autologous conduit	6 (28.6)	-
Inadequate recipient HA	-	34 (100)
Anatomic location		
Aorto-iliac	7 (33.3)	-
Above-knee	10 (47.6)	-
Below-knee	4 (19.0)	-
Aorto-hepatic	-	34 (100)

Data are expressed as numbers (%).

CAA, cadaveric arterial allograft; c-CAA, cryopreserved cadaveric arterial allograft; HA, hepatic artery; f-CAA, fresh cadaveric arterial allograft

Table 3. Comparison of clinical outcomes and morphologic changes between c-CAA and f-CAA groups

	Total (n = 55)	c-CAA group (n = 21)	f-CAA group (n = 34)	p value
Primary outcomes	21 (38.2)	12 (57.1)	9 (26.5)	0.02
aneurysm	5 (9.1)	4 (19.0)	1 (2.9)	0.06
occlusion	9 (16.4)	5 (23.8)	4 (11.8)	0.28
stenosis (≥50%)	10 (18.2)	3 (14.3)	7 (20.6)	0.73
Secondary intervention	7 (12.7)	7 (33.3)	0 (0)	<0.001
Secondary outcomes	9 (16.4)	5 (23.8)	4 (11.8)	0.28
CAA dilatation	2 (3.6)	1 (4.8)	1 (2.9)	1.00
Stenosis (<50%)	3 (5.5)	0 (0)	3 (8.8)	0.28
CAA wall calcification	4 (7.3)	4 (19.0)	0 (0)	0.02
Morphologic changes*	11 (20.0)	9 (42.9)	2 (5.9)	<0.001

Data are expressed as numbers (%).

CAA, cadaveric arterial allograft; c-CAA, cryopreserved cadaveric arterial allograft; f-CAA, fresh cadaveric arterial allograft

*Morphologic changes include aneurysm, CAA dilatation, and wall calcification.

Table 4. Factors associated with clinical outcomes (primary and secondary outcomes) of the study sample (n = 55)

	Univariable analysis		Multivariable analysis	
	HR (95% CI)	p value	HR (95% CI)	p value
Hypertension	1.26 (0.35–4.55)	0.72		
Dyslipidemia	0.58 (0.05–6.84)	0.67		
CAD	0.89 (0.18–4.40)	0.88		
Smoking	1.00 (0.34–2.96)	1.00		
Infection	2.81 (0.80–9.92)	0.11	2.04 (0.14–1.93)	0.33
Antithrombotic agent	1.71 (0.55–5.35)	0.35		
Statin	1.04 (0.30–3.62)	0.95		
Immunosuppressant	0.39 (0.12–1.23)	0.11	0.52 (0.14–1.93)	0.33

CAD, coronary artery disease; CI, confidence interval; HR, hazard ratio

Table 5. Factors associated with morphologic changes of the study sample (n = 55)

	Univariable analysis		Multivariable analysis	
	HR (95% CI)	p value	HR (95% CI)	p value
Hypertension	1.46 (0.32–6.64)	0.63		
Dyslipidemia	9.56 (0.78–117.07)	0.08	1.82 (0.12–27.56)	0.67
CAD	0.63 (0.07–5.88)	0.69		
Smoking	3.38 (0.85–13.42)	0.08	1.65 (0.31–8.82)	0.56
Infection	3.24 (0.80–13.07)	0.10		
Antiplatelet/Anticoagulant	1.52 (0.35–6.58)	0.57		
Statin	1.28 (0.28–5.73)	0.75		
Immunosuppressant	0.06 (0.01–0.31)	<0.01	0.07 (0.01–0.42)	0.004

CAD, coronary artery disease; CI, confidence interval; HR, hazard ratio

Figures and Figure Legends

Fig. 1. Flow chart of patient inclusion.

c-CAA, cryopreserved cadaveric arterial allograft; f-CAA, fresh cadaveric arterial allograft; LT, liver transplantation; PAD, peripheral artery disease

*Included four patients with previous history of organ transplantation

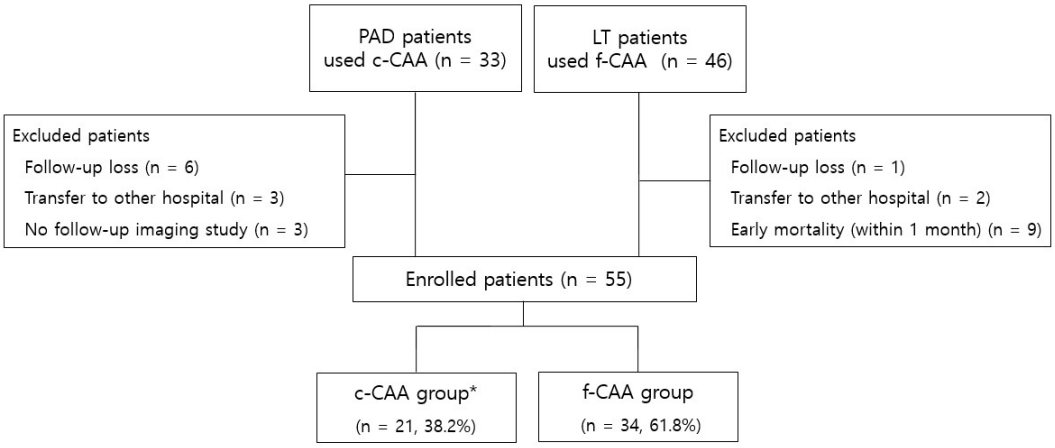
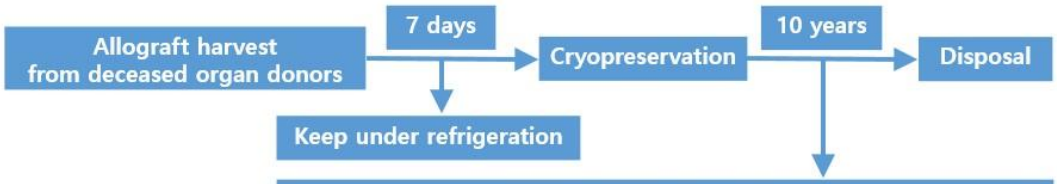


Fig. 2. Preservation techniques.

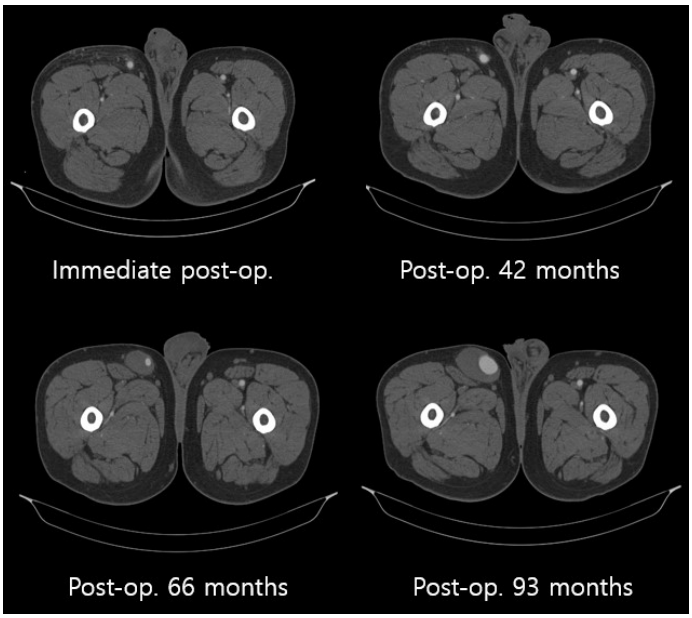
DMSO, dimethylsulfoxide; RPMI, Roswell Park Memorial institute



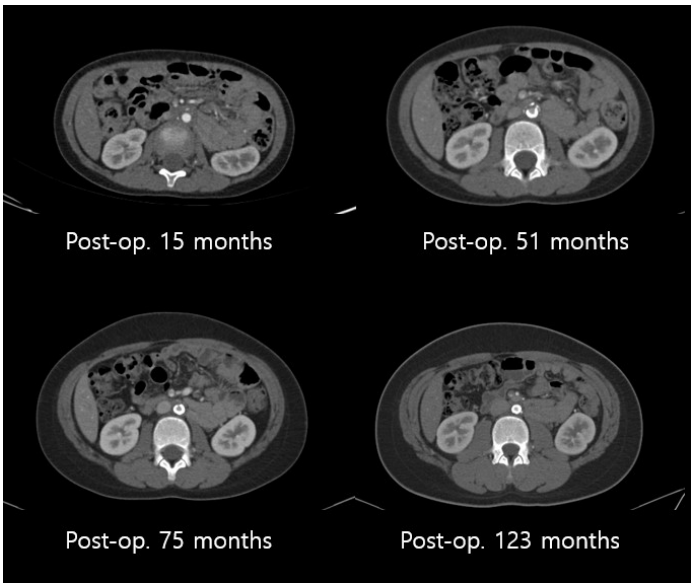
Cryopreservation techniques	
Procedure	**Description**
Antibiotics	Vancomycin, Gentamicin, Cefazolin
Cryoprotectant	RPMI 1640 media with 10% DMSO
Cryopreservation	Rate controlled freezing to -70℃ Storage in the vapor of liquid nitrogen for a maximum of 10 years at the temperature of -100℃
Thawing before use	Rapid thawing in 37–42℃ saline
Rinsing	Rinse DMSO four times with saline for 2 min each time Do not shake frozen cryopreserved arterial allografts during manipulation to avoid microfracture of crystallized tissue

Fig. 3. Representative figures of serial computed tomographic images of morphologic changes of CAA. **(A)** Aneurysm, and **(B)** wall calcification with patent flow.

;CAA, cadaveric arterial allograft



(A)

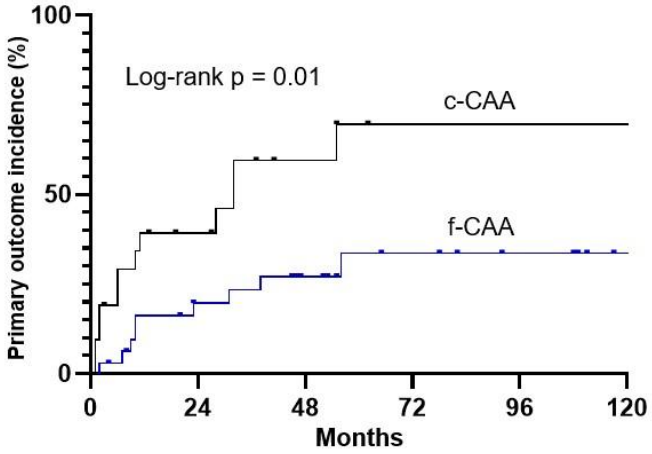


(B)

Fig. 4. Kaplan–Meier analyses of the cumulative incidences of clinical outcomes and morphologic changes.

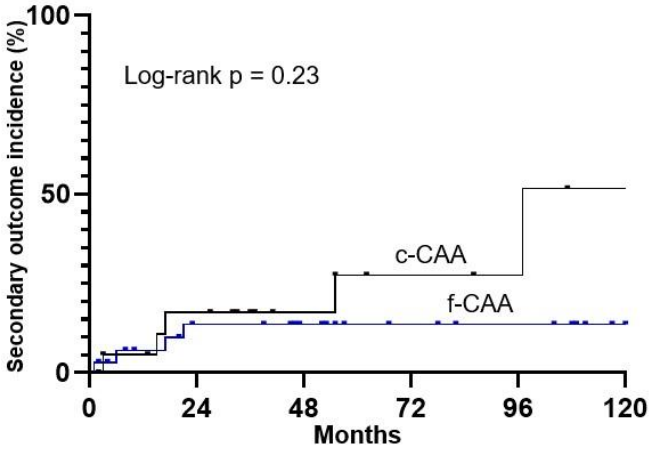
(A) Primary outcome, **(B)** secondary outcome, and **(C)** morphologic change incidences between the c-CAA and f-CAA groups

;c-CAA, cryopreserved cadaveric arterial allograft; f-CAA, fresh cadaveric arterial allograft



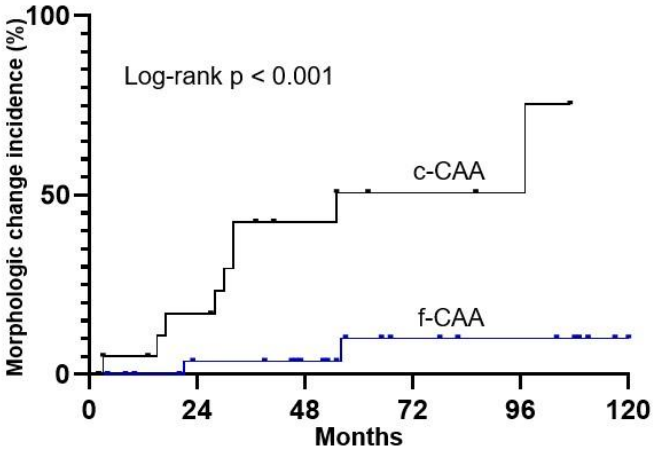
No. at risk	0 month	24 month	48 month	72 month	96 month	120 month
c-CAA	21	11	5	2	2	2
f-CAA	34	24	18	10	7	3

(A)



No. at risk	0 month	24 month	48 month	72 month	96 month	120 month
c-CAA	21	15	9	6	4	2
f-CAA	34	23	19	11	9	3

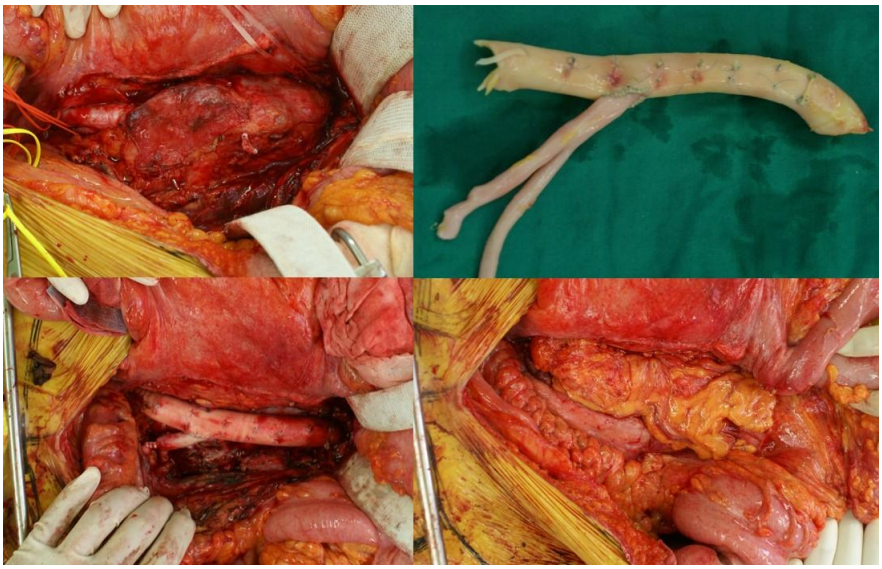
(B)



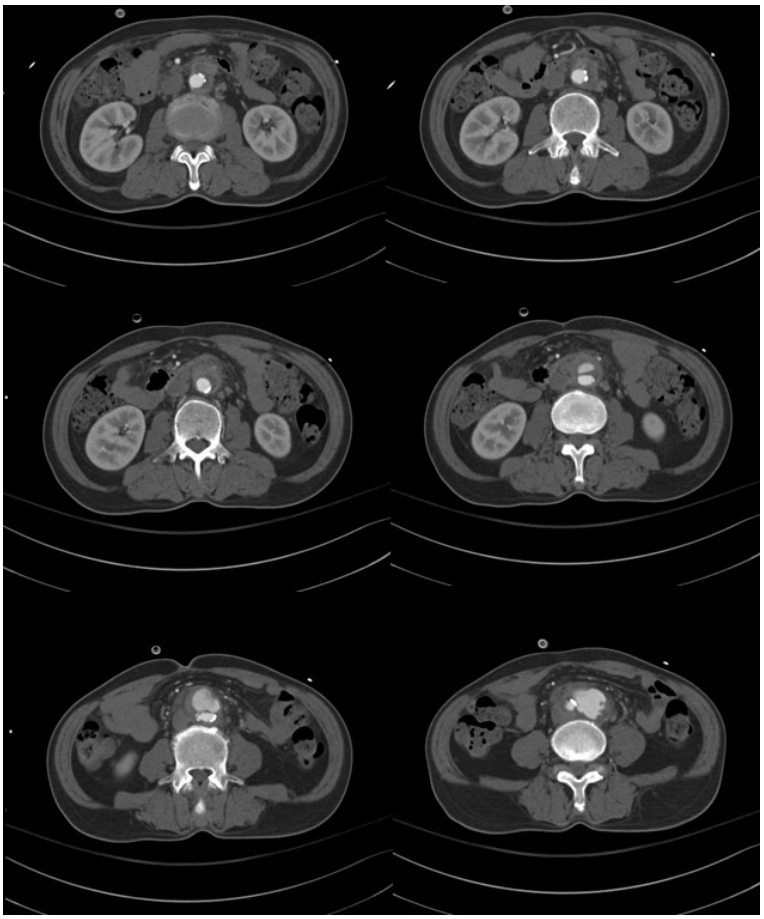
No. at risk	0 month	24 month	48 month	72 month	96 month	120 month
c-CAA	21	15	8	5	3	1
f-CAA	34	26	22	12	10	4

(C)

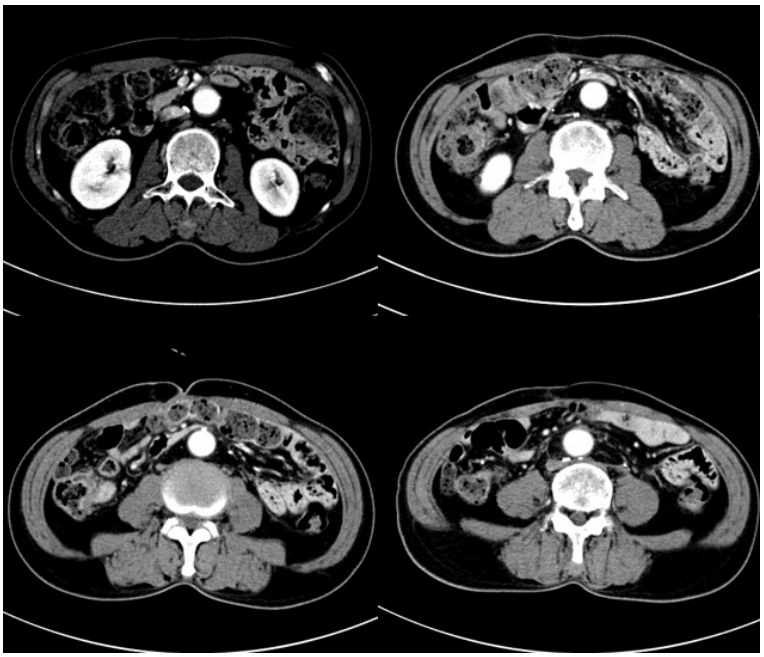
Supplementary Fig. 1 Operative findings and serial computed tomography images in a 65-year-old man without morphologic changes of the c-CAA after aneurysm resection and c-CAA interposition due to mycotic abdominal aortic aneurysm. **(A)** Operative finding of mycotic abdominal aortic aneurysm (upper-left panel). Preparation of c-CAA (upper-right panel). Aneurysm resection and c-CAA interposition (lower-left panel). Omentopexy (lower-right panel). **(B)** Serial images of computed tomography scans at preoperative, 18 months, 42 months, and 54 months after operation.



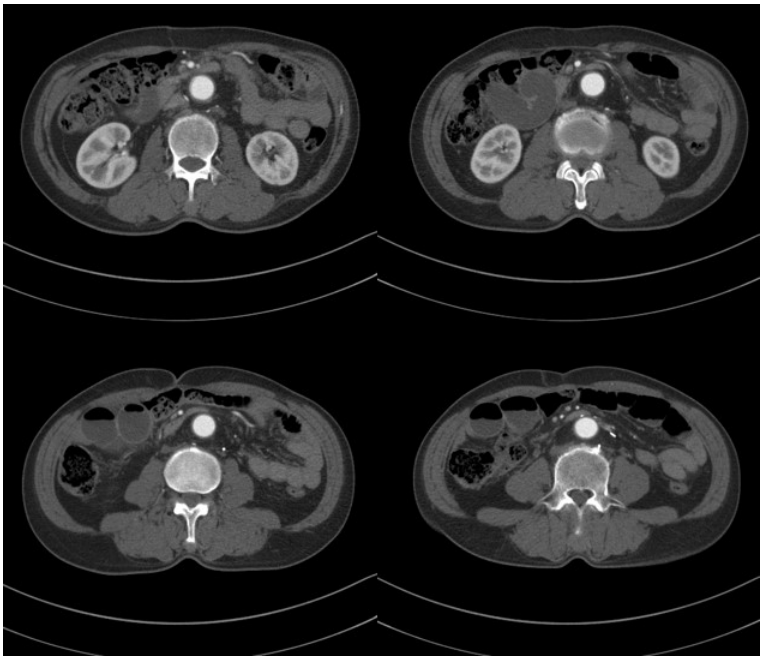
(A)



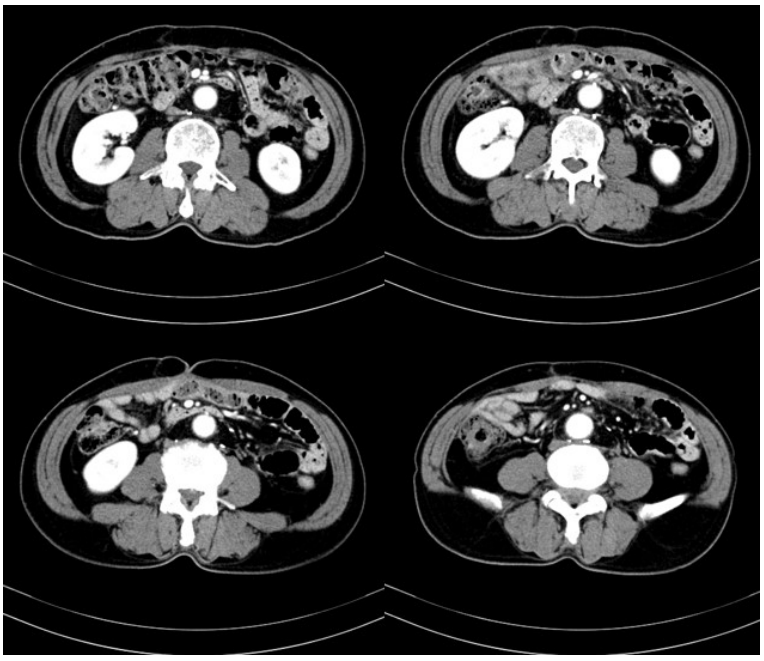
Preoperative CT scan



Postoperative 18 months



Postoperative 42 months

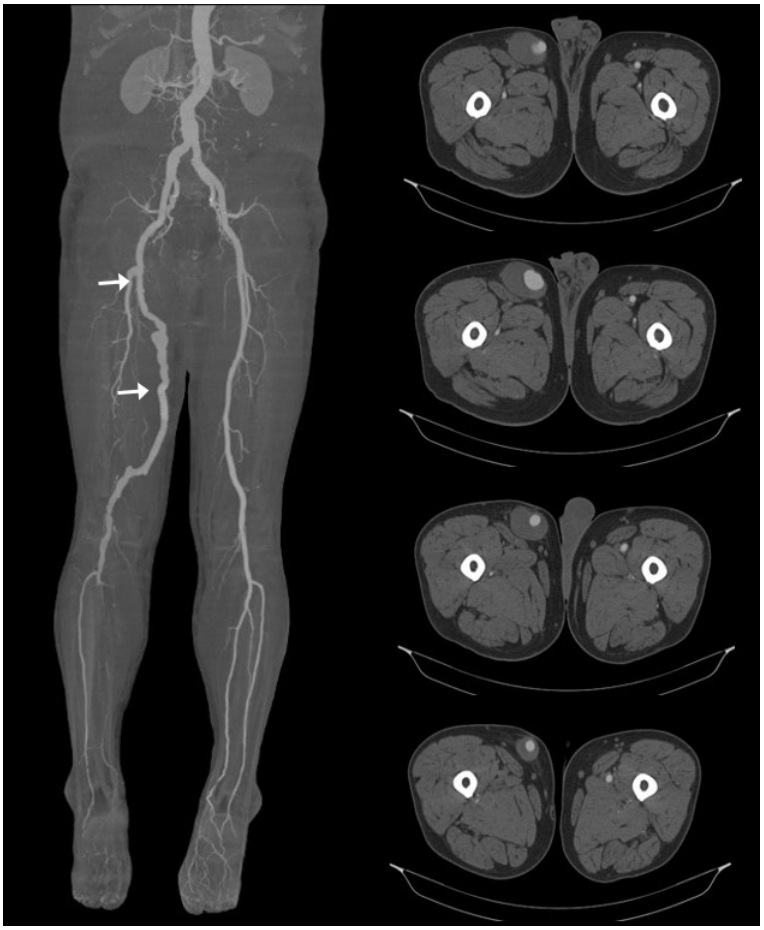


Postoperative 54 months

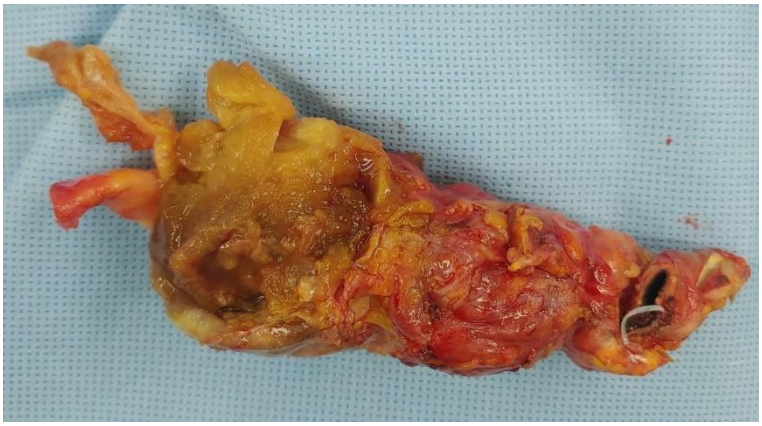
(B)

Supplementary Fig. 2. A 67-year-old man with chronic deep venous thrombosis involving inferior vena cava and both lower extremity deep veins developed an aneurysm of the CAA at 12 years after femoro-popliteal arterial bypass with c-CAA due to recurrent prosthetic graft infection.

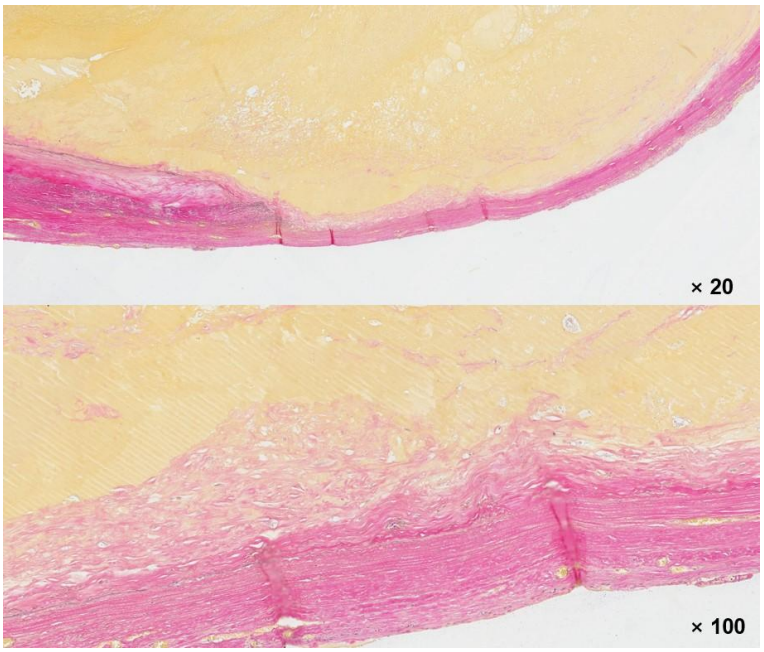
(A) Computed tomography angiography images showed a fusiform aneurysm (26 mm) (arrows) with increased perigraft fluid in the proximal segment of the femoro-popliteal arterial bypass. **(B)** Pathologic specimen of the resected aneurysm. **(C)** The histopathologic examination of the aneurysm sac showed marked thinning of medial layer with partial loss of medial elastic laminae (van Gieson Elastic stain). However, there was no definite evidence of chromic rejection.



(A)



(B)



(C)

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국문 요약

연구배경

본 논문의 목적은 뇌사자 동종 동맥 이식편을 이용한 혈관 재건술의 장기간 예후와 형태학적 변화를 분석하는 것이며, 이를 통해 뇌사자 동종 이식편의 예후와 관련된 인자를 제시하는 것입니다.

연구방법

본 논문은 단일 기관 기반의 후향적 연구이며 동결 보존 뇌사자 동종 동맥 이식편을 이용한 혈관 재건술을 받은 21명의 말초 동맥 혈관 질환 환자(c-CAA 군)과 신선 뇌사자 동종 동맥 이식편을 대동맥-간 도관으로 사용한 34명의 간이식 환자(f-CAA 군)이 포함하였습니다. 1차 결과로 동맥류 또는 폐색/50% 이상의 협착을 포함시켰으며 이차 결과로 뇌사자 동종 동맥 이식편의 확장, 50% 미만의 협착, 그리고 혈관벽의 석회화를 포함시켰습니다. 그리고 형태학적 변화는 동맥류, 동맥류성 확장, 그리고 혈관 석회화로 정의하였습니다.

연구결과

중앙값 55개월(3-137개월)의 추적 기간 동안 1차 및 2차 결과 발생률은 각각 38.2% (21/55), 16.4% (9/55) 였으며 형태학적 변화의 발생률은 20% (11/55) 였습니다. c-CAA 군과 f-CAA군 사이에 1차 결과 ($p=0.02$)와 형태학적 변화 ($p<0.001$)의 발생률에 유의한 차이가 있었습니다. 다변수 분석을 통해 면역억제제의 사용 (비

례위험도, 0.07; 95% 신뢰구간, 0.01-0.42; p=0.004) 이 뇌사자 동종 동맥 이식편의 장기적 형태학적 변화의 좋은 예후와 관련된 중요한 인자임이 나타났습니다.

결론

뇌사자 동종 동맥 이식편을 사용한 재건술을 받은 환자에서 장기적으로 이식편에 동맥류, 협착/폐색, 혈관 석회화 등이 관찰되는 점을 볼 때 이러한 환자에서 이식편의 점진적 악화 및 퇴화를 평가하기 위해 정기적 영상 검진을 고려해야 한다.