

[ ORIGINAL ARTICLE ]

# Association of Ankle Brachial Index with Clinical Outcomes Following Percutaneous Coronary Intervention in Patients with Aortic Aneurysm

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## Abstract:

**Objective** Since patients with thoracic aortic aneurysm (TAA)/abdominal aortic aneurysm (AAA) are often complicated with coronary artery disease, it is common for those patients to undergo percutaneous coronary intervention (PCI). The ankle brachial index (ABI) is usually measured in patients with TAA/AAA to screen the presence of peripheral arterial disease. The present study investigated the association between the ABI and clinical outcomes following PCI in patients with TAA/AAA.

**Methods** We divided 200 TAA/AAA patients who underwent PCI into a normal ABI group (n=137) and an abnormal ABI group (n=63) according to the ABI cut-off level of 1.00. The primary endpoint was one-year major adverse cardiovascular events (MACE), defined as the composite of cardiovascular death, non-fatal myocardial infarction, stroke, target vessel revascularization, and hospitalization for heart failure.

**Results** Mean ABIs in the normal and abnormal ABI groups were  $1.12 \pm 0.09$  and  $0.86 \pm 0.11$ , respectively ( $p < 0.01$ ). Kaplan-Meier curves showed MACE were more frequent in the abnormal ABI group than in the normal ABI group ( $p = 0.01$ ). A multivariate Cox hazard analysis revealed that an abnormal ABI was significantly associated with 1-year MACE (vs.  $ABI \geq 1.0$ : HR 3.02, 95% confidence interval 1.00-9.08,  $p = 0.049$ ).

**Conclusion** Among patients with TAA/AAA who underwent PCI, abnormal ABI was significantly associated with 1-year MACE, suggesting the utility of the ABI measurement in this high-risk population.

**Key words:** percutaneous coronary interventions, aortic aneurysm, ankle brachial index

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## Introduction

Thoracic aortic aneurysm (TAA) and abdominal aortic aneurysm (AAA) are major vascular diseases induced by systemic atherosclerosis. TAA and AAA are related to increased morbidity and mortality rates, especially due to the rupture of TAA/AAA (1, 2). Since the mortality of TAA/AAA rupture is still very high, open surgery or endovascular aortic repair should be performed before rupture occurs (2-4). Furthermore, patients with TAA/AAA often have coronary artery disease (CAD), which results in an even greater mortality rate (5, 6). Therefore, it is not uncommon for patients with TAA/AAA to undergo percutaneous coro-

nary intervention (PCI) (6, 7).

The ankle brachial index (ABI) has been reported as an indicator of clinical outcomes in patients who underwent PCI (8-10). ABI is widely performed in patients with TAA/AAA, as TAA/AAA is often complicated with peripheral artery disease (11). Thus, patients with TAA/AAA who undergo PCI usually have available data on their ABI, regardless of the presence of peripheral artery disease. However, the significance of the ABI in TAA/AAA patients who undergo PCI has not been established, as few reports have described the association between the ABI and clinical outcomes in TAA/AAA patients with CAD. We hypothesized that the ABI could be a simple and useful predictor of major adverse cardiovascular events (MACE) in TAA/AAA pa-

tients who underwent PCI. The present study investigated the association between the ABI and 1-year MACE in TAA/AAA patients who underwent PCI.

## Materials and Methods

### Study design

We reviewed consecutive PCI cases in our medical center between January 2016 and December 2018. The inclusion criteria were 1) patients who underwent PCI and 2) patients who had TAA/AAA at the day of PCI. The exclusion criteria were 1) PCI to graft lesions, and 2) a lack of ABI data (within one year before or one month after the index PCI). We divided the final study population into two groups according to the ABI. Cases with a preserved ABI (ABI  $\geq$  1.00) were considered the normal ABI group, while those with borderline (0.90 to  $<$ 1.00) or decreased values ( $<$ 0.90) were defined as the abnormal ABI group (8, 12, 13).

The primary endpoint was MACE at one year after PCI. MACE were defined as the composite of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, any target vessel revascularization, and hospitalization for acute de-compensated heart failure (ADHF). Non-fatal myocardial infarction was defined according to the universal definition (14). The index day was set as the day of PCI.

All therapeutic strategies related to TAA/AAA and CAD were considered and discussed with cardiovascular surgeons, and we decided not only the indication but also the order and timing of treatments.

### PCI procedure

We decided PCI indication and strategy at our daily conference except emergent PCI cases. In elective PCI for patients with TAA, we consult cardiovascular surgeons about the possibility of open thoracic surgery with simultaneous CABG, and about the timing of PCI (before or after surgery). In elective PCI for patients with AAA, we consult cardiovascular surgeons about the timing of PCI (before surgery or after surgery). The high-risk or complex cases in the present study were discussed at a weekly heart team conference consisting of cardiologists and cardiovascular surgeons. The radial artery was most frequently used, followed by the brachial artery, with the femoral artery being used the least, because of TAA/AAA. We maintained an activated clotting time of at least 250 seconds. The selection of devices was left to the discretion of each interventional cardiologist. We tried to avoid the insertion of mechanical support devices, such as an intra-aortic balloon pump (IABP) or veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Regarding complications, peri-procedural myocardial infarction in non-emergent cases was defined as cases where the creatine kinase values the day after PCI increased to more than twice the upper normal limit (15).

### Definitions

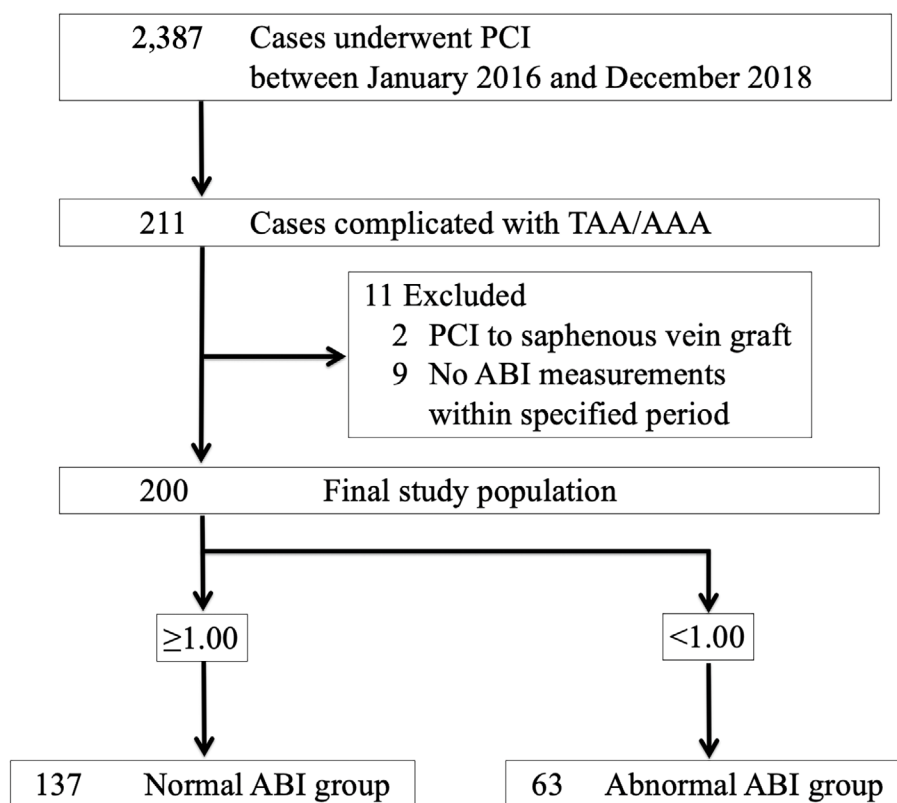
TAA/AAA was defined in cases with a history of open or endovascular surgeries or maximum aortic diameter  $\geq$ 45 mm for TAA and  $\geq$ 30 mm for AAA (16). Hypertension was defined as receiving treatment for hypertension before admission (17, 18). Dyslipidemia was defined as total cholesterol  $>$ 220 mg/dL, low-density lipoprotein cholesterol (LDL)  $>$  140 mg/dL, or medical treatment for dyslipidemia before admission (17-19). Diabetes mellitus (DM) was defined as hemoglobin A1c (HbA1c)  $>$ 6.5% [national glycol-hemoglobin standardization program (NGSP) value] or medical treatment for DM (17, 19, 20). The estimated glomerular filtration rate (eGFR) was calculated by the modification of diet in renal disease (MDRD) method, adjusted for a Japanese population (21, 22). Anemia was determined by the World Health Organization (WHO) criteria as a hemoglobin (Hb) value  $<$  13 g/dL in men and  $<$ 12 g/dL in women (23, 24). ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina pectoris (UAP) were defined according to the universal definitions (14). Acute coronary syndrome (ACS) was defined as a composite of STEMI, NSTEMI, and UAP.

### Statistical analyses

Categorical data were presented as number and percentage, and continuous data were presented as the mean  $\pm$  standard deviation (SD). Normally distributed continuous variables were compared using an unpaired Student's *t*-test. Other continuous variables were compared using the Mann-Whitney *U*-test. Categorical data were compared using the chi-square test or Fisher's exact test. A Kaplan-Meier survival analysis was performed to compare 1-year MACE between the normal and abnormal ABI groups, and difference were assessed by the log-rank test. We also performed a multivariate Cox hazard analysis (stepwise method: backward elimination using likelihood ratio) to examine the associations between an abnormal ABI and 1-year MACE after controlling confounding factors. In this model, MACE were used as a dependent variable, and an abnormal ABI was the independent variable. We applied confounding factors, defined as variables with a P value  $<$ 0.10 in the univariate analyses. Although there were significant differences between the normal and abnormal ABI groups, variables that had missing values were not included in the multivariate analysis. Hazard ratios (HRs) and 95% confidence intervals (CIs) were calculated. A p value  $<$ 0.05 was considered statistically significant. All analyses were performed with IBM SPSS statistics version 25 (Chicago, USA).

## Results

We screened 2,387 consecutive PCI cases encountered at our center between January 2016 and December 2018. Of those, 211 were diagnosed with or treated for TAA/AAA. Eleven cases were excluded because of PCI to a saphenous



**Figure 1.** Flowchart of patient selection and how patients were divided into two groups. AAA: abdominal aortic aneurysm, ABI: ankle brachial index, PCI: percutaneous coronary intervention, TAA: thoracic aortic aneurysm

vein graft or a lack of ABI data (within one year before or one month after the index PCI). A total of 200 PCI cases were thus included as the final study population and divided into the normal ABI group (n=137) and abnormal ABI group (n=63) (Fig. 1).

Table 1 shows the comparison of the baseline characteristics between the two groups. As defined, the ABI was significantly higher in the normal ABI group than in the abnormal group ( $1.12 \pm 0.09$  vs.  $0.86 \pm 0.11$ ,  $p < 0.01$ ). The eGFR was also higher in the normal ABI group than in the abnormal group ( $59.5 \pm 20.1$  mL/min/1.73 m<sup>2</sup> vs.  $53.4 \pm 23.6$  mL/min/1.73 m<sup>2</sup>,  $p = 0.04$ ). BNP values were lower in the normal ABI group than in the abnormal ABI group ( $211 \pm 409$  pg/mL vs.  $336 \pm 480$  pg/mL,  $p = 0.01$ ). The use of diuretics at discharge were more frequently observed in the abnormal ABI group than in the normal ABI group (25.5% vs. 47.6%,  $p < 0.01$ ).

Table 2 shows the comparison of the procedural characteristics between the two groups. The prevalence of ACS was lower in the normal ABI group than in the abnormal ABI group (29.2% vs. 46.0%,  $p = 0.03$ ). VA-ECMO was more frequently inserted in the abnormal ABI group than in the normal ABI group (0% vs. 6.3%,  $p < 0.01$ ).

We constructed Kaplan-Meier curves, and 1-year MACE were significantly better in the normal ABI group than in the abnormal ABI group (Fig. 2) ( $p = 0.01$ ). We also performed Fisher's exact tests regarding individual outcomes,

including MACE, between the two groups (Table 3). The causes of five cardiovascular deaths were as follows: acute myocardial infarction in one, HF in one, sudden death just after thoracic aortic replacement (without rupture) in one, and sudden death due to unknown causes in two.

Table 4 shows the results of a multivariate Cox hazard analysis. Although the initial model included an abnormal ABI, history of hypertension, eGFR  $< 30$  mL/min/1.73 m<sup>2</sup>, moderate or severe AR, diuretics user, insulin user, ACS, multi-vessel disease, and VA-ECMO use, the final model included only 5 variables (ABI, multi-vessel disease, ACS, moderate/severe AR, and insulin user) according to the stepwise method (backward elimination and likelihood ratio). An abnormal ABI  $< 1.0$  (HR 3.02, 95% CI 1.00-9.08,  $p = 0.049$ ) was significantly associated with 1-year MACE after controlling for confounding factors.

## Discussion

We divided 200 patients with TAA/AAA who underwent PCI into the normal ABI group (n=137) and abnormal ABI group (n=63). The Kaplan-Meier curves showed that the incidence of MACE was higher in the abnormal ABI group than in the normal ABI group. The multivariate Cox hazard analysis revealed that an abnormal ABI was significantly associated with 1-year MACE (HR 3.02, 95% CI 1.00-9.08,  $p = 0.049$ ) after controlling for confounding factors. Several re-

**Table 1. Comparison of Patient Characteristics between the Normal ABI Group and Abnormal ABI Group.**

	Overall (n=200)	Normal ABI group (n=137)	Abnormal ABI group (n=63)	p value
Age (years old)	74.3±7.7	74.4±8.0	74.1±7.3	0.79
Female gender (No.) (%)	26 (13.0)	16 (11.7)	10 (15.9)	0.50
Body mass index (kg/m <sup>2</sup> )	23.1±3.6	23.2±3.9	22.7±2.7	0.39
Smoker (No.) (%)	160 (80.0)	109 (79.6)	51 (81.0)	1.00
Hypertension (No.) (%)	181 (90.5)	128 (93.4)	53 (84.1)	0.07
Dyslipidemia (No.) (%)	167 (83.5)	117 (85.4)	50 (79.4)	0.31
Diabetes mellitus (No.) (%)	54 (27.0)	38 (27.7)	16 (25.4)	0.86
History of hospitalization of ADHF (No.) (%)	8 (4.0)	5 (3.6)	3 (4.8)	0.71
AAA (No.) (%)	174 (87.0)	120 (87.6)	54 (85.7)	0.82
TAA (No.) (%)	51 (25.5)	36 (26.3)	15 (23.8)	0.86
Post intervention to aneurysms (No.) (%)	71 (35.5)	51 (37.2)	20 (31.7)	0.53
ABI	1.04±0.15	1.12±0.09	0.86±0.11	<0.01
Laboratory data				
HbA1c (NGSP) (%) (n=198)	6.1±0.9	6.1±0.6	6.2±1.3	0.23
Creatinine (mg/dL)	1.30±1.47	1.17±1.12	1.58±2.01	0.09
eGFR (mL/min/1.73 m <sup>2</sup> )	57.6±21.4	59.5±20.1	53.4±23.6	0.04
LDL cholesterol (mg/dL) (n=191)	94±28	97±29	89±25	0.16
Hemoglobin (g/dL)	12.8±2.9	13.0±3.2	12.4±1.7	0.13
Anemia (WHO criteria) (No.) (%)	109 (54.5)	74 (54.0)	35 (55.6)	0.88
Prothrombin time-INR (n=186)	1.08±0.19	1.06±0.15	1.14±0.24	0.02
APTT (s) (n=190)	39.6±28.7	36.6±22.0	45.9±38.7	0.01
D-dimer (n=123)	4.16±7.32	4.39±8.73	3.70±3.23	0.63
BNP (pg/mL) (n=189)	251±436	211±409	336±480	0.01
Echo cardiography				
Left ventricular EF (modified Simpson's methods) (%) (n=124)	52.6±17.5	54.5±15.8	48.0±20.6	0.18
Moderate/severe AR (No.) (%)	8 (4.0)	8 (5.8)	0 (0.0)	0.06
Medication at discharge				
Aspirin (No.) (%)	200 (100)	137 (100)	63 (100)	-
P2Y <sub>12</sub> inhibitors (No.) (%)				0.13
Clopidogrel	147 (73.5)	100 (73.0)	47 (74.6)	
Prasugrel	49 (24.5)	36 (26.3)	13 (20.6)	
Oral anti-coagulants (No.) (%)	29 (14.5)	19 (13.9)	10 (15.9)	0.83
Statins (No.) (%)	194 (97.0)	132 (96.4)	62 (98.4)	0.67
ACEI and/or ARB (No.) (%)	137 (68.5)	92 (67.2)	45 (71.4)	0.62
β blockers (No.) (%)	157 (78.5)	105 (76.6)	52 (82.5)	0.46
Diuretics (No.) (%)	65 (32.5)	35 (25.5)	30 (47.6)	<0.01
Oral hypoglycemic agents (No.) (%)	32 (16.0)	19 (13.9)	13 (20.6)	0.30
Insulin (No.) (%)	4 (2.0)	1 (0.7)	3 (4.8)	0.09

WHO criteria: the hemoglobin of the male<13 g/dL and the female<12 g/dL

ABI: ankle brachial index, ADHF: acute decompensated heart failure, AAA: abdominal aortic aneurysm, TAA: thoracic aortic aneurysm, HbA1c: hemoglobin A1c, NGSP: national glycohemoglobin standardization program, eGFR: estimated glomerular filtration rate, LDL: low density lipoprotein, WHO: World Health Organization, INR: international normalized ratio, APTT: activated partial thromboplastin time, BNP: brain natriuretic peptide, EF: ejection fraction, AR: aortic valve regurgitation, ACEI: angiotensin converting enzyme inhibitors, ARB: angiotensin II receptor blockers

ports concerning members of the general population who underwent PCI have described the relationship between the ABI and MACE (8-10,12,25). Sasaki et al. (2020) reported that a lower ABI was a good predictor of MACE after drug-eluting stent implantation (9). Furthermore, Nishimura et al. (2017) mentioned that an ABI <1.00 predicted future inci-

dent of hospitalization for ADHF (25). However, our study, which focused on TAA/AAA cases, is unique in elucidating the relationship between ABI and MACE.

Regarding why an abnormal ABI was associated with MACE after PCI in patients with TAA/AAA, when we considered each parameter of MACE, the incidence of hospitali-

**Table 2. Comparison of Interventions between the Normal ABI Group and the Abnormal ABI Group.**

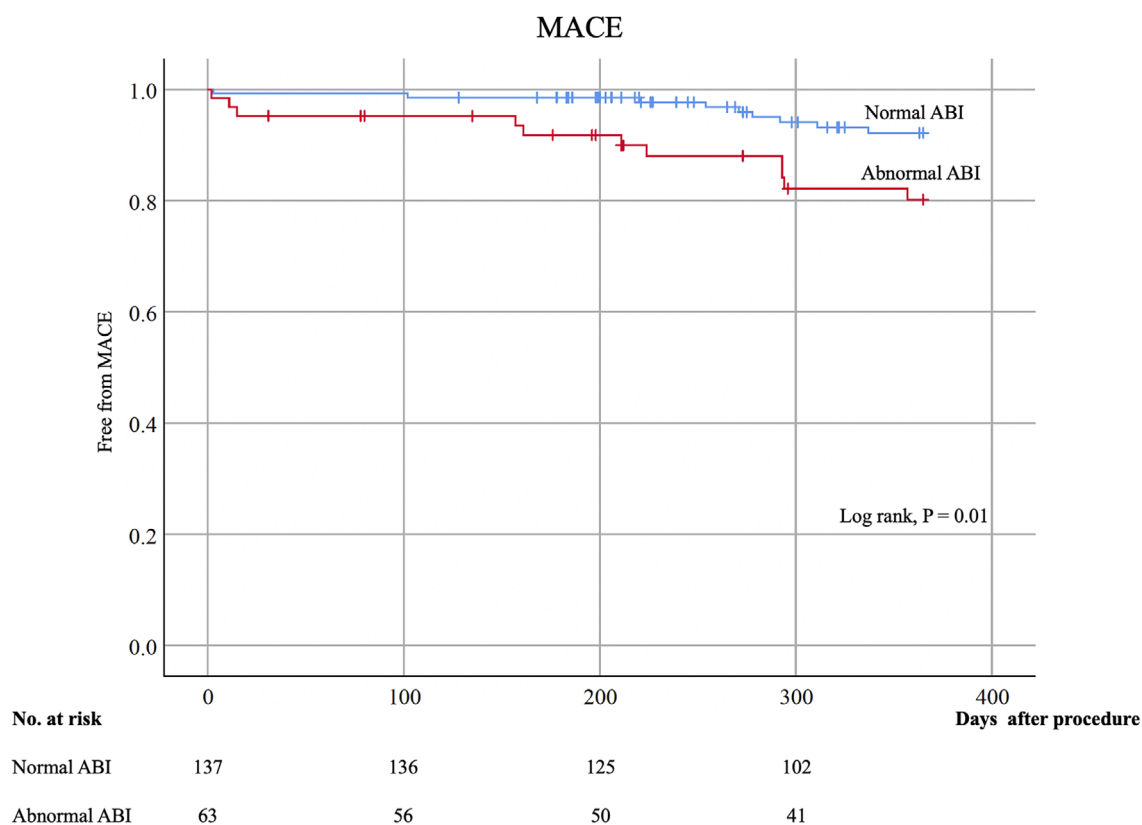
	Overall (n=200)	Normal ABI group (n=137)	Abnormal ABI group (n=63)	p value
Emergent PCI (No.) (%)	37 (18.5)	23 (16.8)	14 (22.2)	0.43
Acute coronary syndrome (No.) (%)	69 (34.5)	40 (29.2)	29 (46.0)	0.03
Approach site				0.23
Right radial or brachial (No.) (%)	114 (57.0)	79 (57.7)	35 (55.6)	
Left radial or brachial (No.) (%)	48 (24.0)	36 (26.3)	12 (19.0)	
Femoral (No.) (%)	38 (19.0)	22 (16.1)	16 (25.4)	
Sheath size (No.) (%)				0.20
6 Fr	104 (52.0)	77 (56.2)	27 (42.9)	
7 Fr	89 (44.5)	56 (40.9)	33 (52.4)	
≥8 Fr	7 (3.5)	4 (2.9)	3 (4.8)	
Culprit lesions				0.26
Right coronary (No.) (%)	77 (38.5)	53 (38.7)	24 (38.1)	
Left main (No.) (%)	10 (5.0)	4 (2.9)	6 (9.5)	
Left anterior descending (No.) (%)	86 (43.0)	61 (44.5)	25 (39.7)	
Left circumflex (No.) (%)	27 (13.5)	19 (13.9)	8 (12.7)	
Multi-vessel disease (No.) (%)	110 (55.0)	69 (50.4)	41 (65.1)	0.07
Lesion types and strategies				
In-stent restenosis (No.) (%)	13 (6.5)	6 (4.4)	7 (11.1)	0.12
Chronic total obstruction (No.) (%)	20 (10.0)	15 (10.9)	5 (7.9)	0.62
Rotablation (No.) (%)	24 (12.0)	16 (11.7)	8 (12.7)	0.82
Jailed micro-catheter (No.) (%)	3 (1.5)	2 (1.5)	1 (1.6)	1.00
Post optimized technique (No.) (%)	15 (7.5)	8 (5.8)	7 (11.1)	0.25
Kissing balloon technique (No.) (%)	5 (2.5)	3 (2.2)	2 (3.2)	0.65
Micro-catheters (No.) (%)	66 (33.0)	41 (29.9)	25 (39.7)	0.20
Extension catheters (No.) (%)	30 (15.0)	24 (17.5)	6 (9.5)	0.20
POBA alone (No.) (%)	8 (4.0)	3 (2.2)	5 (7.9)	0.11
DCB alone (No.) (%)	16 (8.0)	10 (7.3)	6 (9.5)	0.58
Stenting (No.) (%)	177 (88.5)	124 (90.5)	53 (84.1)	0.23
Drug eluting stents (n=177) (No.) (%)	161 (91.0)	112 (90.3)	49 (92.5)	0.78
Mechanical support devices				
IABP (No.) (%)	5 (2.5)	2 (1.5)	3 (4.8)	0.18
VA-ECMO (No.) (%)	4 (2.0)	0 (0)	4 (6.3)	<0.01
Successful PCI (No.) (%)	198 (99.0)	136 (99.3)	62 (98.4)	0.53
Contrast volume (mL)	112±58	112±49	114±76	0.46
Exposure time (min)	24±18	23±16	25±20	0.93
Peri-procedural MI (No.) (%)	8 (4.0)	5 (3.6)	3 (4.8)	0.71

ABI: ankle brachial index, PCI: percutaneous coronary intervention, POBA: plane old balloon angioplasty, DCB: drug coated balloon, IABP: intra-aortic balloon pumping, VA-ECMO: veno-arterial extracorporeal membrane oxygenation, MI: myocardial infarction

zation caused by ADHF tended to be greater in the abnormal ABI group than in the normal ABI group, albeit without reaching statistical significance (2.9% vs. 7.9%,  $p=0.14$ ). Several groups reported the relationship between an abnormal ABI and ADHF (12, 25). Gupta et al. (2014) described the significant association between an abnormal ABI and HF in a middle-age community cohort ( $n=1,315$ ) and speculated that vascular stiffness and atherosclerotic microvascular dysfunction might provoke HF in patients with an abnormal ABI (12). An impaired vascular compliance was shown to be associated with a low ABI (26). Vascular stiffness increases the left ventricular afterload, which may impair the coronary blood flow and induce myocardial ischemia, even without significant coronary artery stenosis (27).

Furthermore, an abnormal ABI may indicate systemic atherosclerosis or poly-vascular disease (8). Indeed, CAD is often complicated with peripheral arterial diseases or cerebral vascular disease (28), which have been associated with MACE after PCI (29). Since the presence of TAA/AAA and severe CAD was an inclusion criterion in the present study, all study patients already had at least two major atherosclerotic risk factors. In the present study population, abnormal ABI referred to the presence of another atherosclerotic risk factor, suggesting more advanced atherosclerosis in the abnormal ABI group than in the normal ABI group. Therefore, the abnormal ABI cases was associated with one-year MACE.

Our results suggest that ABI measurement is useful in pa-



**Figure 2.** The comparison of the Kaplan-Meier curves between the normal and abnormal ABI groups. Major adverse cardiovascular events (MACE) at one year after percutaneous coronary intervention (PCI) were defined as the composite of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, any target vessel revascularization, and hospitalization for acute de-compensated heart failure. ABI: ankle brachial index

**Table 3.** Comparison of One-year MACE between the Normal ABI Group and the Abnormal ABI Group.

	Overall (n=200)	Normal ABI group (n=137)	Abnormal ABI group (n=63)	p value
One-year MACE (No.) (%)	20 (10.0)	9 (6.6)	11 (17.5)	0.02
Cardiovascular death (No.) (%)	5 (2.5)	2 (1.5)	3 (4.8)	0.18
Non-fatal myocardial infarction (No.) (%)	0 (0)	0 (0)	0 (0)	-
Non-fatal stroke (No.) (%)	1 (0.5)	0 (0)	1 (1.6)	0.32
Target vessel revascularization (No.) (%)	5 (2.5)	3 (2.2)	2 (3.2)	0.65
Hospitalization due to ADHF (No.) (%)	9 (4.5)	4 (2.9)	5 (7.9)	0.14

ABI: ankle brachial index, ADHF: acute decompensated heart failure, MACE: major adverse cardiovascular event (composite of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, target vessel revascularization, and hospitalization due to ADHF)

tients with TAA/AAA who undergo PCI, as the ABI can be a marker predicting the clinical outcomes after PCI. Furthermore, an exercise stress test, which is an established predictor of clinical outcomes after PCI (30), tends to be avoided among patients with TAA/AAA due to concerns about the rupture of TAA/AAA. The ABI thus has a great advantage over exercise stress tests with regard to its non-invasiveness.

The present study revealed that ACS (HR 3.90, 95% CI 1.48-10.22,  $p=0.006$ ), moderate/severe AR (HR 8.31, 95% CI 1.96-35.29,  $p=0.004$ ) and insulin users (HR 8.88, 95% CI 2.14-36.87,  $p=0.003$ ) were associated with 1-year MACE. It is well known that patients with ACS have worse

clinical outcomes than those with stable angina (31). Although moderate/severe AR has been shown to be associated with MACE, there have been few reports describing the relationship between AR and MACE after PCI. However, severe AR can be a cause of HF. Since MACE included hospitalization for ADHF in the present study, AR might be associated with MACE. Insulin use was also associated with MACE. It is known that, among patients after PCI, the incidence of MACE is greater in insulin users than in non-users (32).

Several limitations associated with the present study warrant mention. Since this study was a retrospective study,

**Table 4. Multivariate COX Hazard Analysis.**

Independent variables for "one-year MACE"	HR	95% CI	p value
Peri-procedural ABI<1.0	3.02	1.00-9.08	0.049
Multi-vessel disease	2.60	0.85-7.98	0.096
Acute coronary syndrome	3.90	1.48-10.22	0.006
Moderate/severe AR	8.31	1.96-35.29	0.004
Insulin users	8.88	2.14-36.87	0.003

Covariates were ABI<1.0, history of hypertension, eGFR<30 mL/min/1.73 m<sup>2</sup>, moderate or severe AR, diuretics user, insulin user, acute coronary syndrome, multi-vessel disease, and VA-ECMO use.

Analysis was performed by stepwise method (backward elimination and likelihood ratio) and fifth step was final.

ABI: ankle brachial index, AR: aortic valve regurgitation, MACE: major adverse cardiovascular event (composite of cardiovascular death, non-fatal myocardial infarction, non-fatal stroke, target vessel revascularization, and hospitalization due to ADHF)

there was a potential selection bias. Because the sample size was limited, the statistical analysis carried an inherent risk of beta error (33). Since the number of 1-year MACE was small (n=20, 10.0% of the sample volume), the number of independent variables was limited in the multivariate Cox hazard analysis. Further studies including a sufficient study population are warranted to confirm the relationship between an abnormal ABI and clinical outcomes after PCI among patients with TAA/AAA.

## Conclusion

Among patients with TAA/AAA who underwent PCI, an abnormal ABI was significantly associated with 1-year MACE, suggesting the utility of measuring the ABI in this high-risk population.

### Author's disclosure of potential Conflicts of Interest (COI).

Kenichi Sakakura: Employment, Boston Scientific and Abbott Vascular; Honoraria, Abbott Vascular, Boston Scientific, Medtronic Cardiovascular, Terumo, OrbusNeich, Japan Lifeline and NIPRO. Hideo Fujita: Employment, Mehergen Group Holdings.

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