Ankle–Brachial Index as a Prognostic Indicator in Patients with Atrial Fibrillation—A Subanalysis of the IMPACT–ABI Study

Yasutaka Oguchi¹(2)*, Tatsuya Saigusa¹, Soichiro Ebisawa¹, Ayako Okada¹, Hirohiko Motoki¹ and Koichiro Kuwahara¹

¹) Department of Cardiovascular Medicine, Shinshu University School of Medicine
²) Department of Cardiovascular Medicine, Aizawa Hospital

Introduction

The CHADS² (congestive heart failure, hypertension, age≥75 years, diabetes, stroke) score and CHA²DS²-VASc (congestive heart failure, hypertension, age≥75 years, diabetes, stroke) scores are well-established predictors for outcomes in patients with atrial fibrillation (AF). The ankle–brachial index (ABI) is a known tool for diagnosing peripheral artery disease and a predictor for cardiovascular diseases; however, it is unclear whether it can predict cardiovascular death, myocardial infarction (MI), and stroke (major adverse cardiovascular event (MACE)) in patients with AF. The aim of this study was to investigate whether ABI could predict the prognosis in AF patients.

Background: The CHADS₂ score is a well-established predictor for outcomes in patients with atrial fibrillation (AF). The ankle–brachial index (ABI) is a known tool for diagnosing peripheral artery disease and a predictor for cardiovascular diseases; however, it is unclear whether it can predict cardiovascular death, myocardial infarction (MI), and stroke (major adverse cardiovascular event (MACE)) in patients with AF. The aim of this study was to investigate whether ABI could predict the prognosis in AF patients.

Methods: We measured ABI in 3131 consecutive patients who visited our cardiovascular center from 2005 to 2015, of which 401 had AF, and they were enrolled in this study. Three patients were excluded because their ABI was inadequate or higher than 1.5. The mean age was 68.0±11.3 years old, and mean observation period was 4.6±2.7 years. We examined the relationship between ABI and MACE.

Results: Of 398 patients, 52 (13.1 %) had MACE. Patients with ABI <0.92 had an increased incidence of MACE over those with ABI>0.92 (17 of 66 vs. 35 of 332, HR 2.2, 95 % CI 1.3 to 3.6, p = 0.0056). Patients with CHADS₂ score>2 had a statistically insignificant increase in MACE over those with CHADS₂<1 (HR 1.6, 95 % CI 0.92 to 2.73, p = 0.12). Further, a CHA²DS²-VASc score>2 showed a significant difference from that<1 (HR 3.0, 95 % CI 1.2 to 7.4, p = 0.0079).

Conclusions: The ABI could predict the prognosis of patients with AF comparable to the CHADS₂ or CHA²DS²-VASc scores. Shinshu Med J 67 : 197―204, 2019

Key words: ABI, AF, CHADS₂ score, CHA²DS²-VASc score

Abbreviations: ABI, ankle brachial index; MACE, major adverse cardiovascular event; BMI, body mass index; CAD, coronary artery disease; CHF, congestive heart failure; HT, hypertension; DM, diabetes mellitus; SAS, sleep apnea syndrome; AF, atrial fibrillation; MI, myocardial infarction; ACE-I, angiotensin-converting enzyme inhibitor; ARB, angiotensin receptor blocker; CCB, calcium channel blocker; Hb, hemoglobin; Cr, creatinine; eGFR, estimated glomerular filtration rate; T-Chol, total cholesterol; HDL-Chol, high-density lipoprotein cholesterol; LDL-Chol, low-density lipoprotein cholesterol; HbA1c, hemoglobin A1c; CRP, C-reactive protein; BNP, B-type-natriuretic peptide; LVDd, left ventricular end diastolic diameter; LVDs, left ventricular end systolic diameter; LVEF, left ventricular ejection fraction; LADs, left atrial diameter of systole; LV, left ventricular; NYHA, New York Heart Association; RA, right atrial

* Corresponding author: Yasutaka Oguchi
Department of Cardiovascular Medicine,
Shinshu University School of Medicine,
3-1-1 Asahi, Matsumoto, Nagano 390-8621, Japan
E-mail: litatolad@yahoo.co.jp

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[doubled], vascular disease, sex category [if female, counted]) score are well-established predictors of stroke in patients with atrial fibrillation (AF)\(^1\). Moreover, they are known to be predictors of adverse cardiovascular events\(^2\) -\(^4\). On the other hand, the ankle-brachial index (ABI) is known as a tool for detecting peripheral artery disease (PAD) with high specificity (low ABI (ABI≤0.9), a strong predictor of future cardiovascular events and mortality\(^5\) -\(^7\). and as a predictor for cardiovascular diseases and all-cause mortality\(^8\) -\(^9\). However, the influence of the ABI in predicting the disease outcome of AF patients has not been well researched. We think that ABI measurement in AF patients has two clinical meanings. First, we wanted to evaluate their prognosis using only ABI, which is simple and non-invasive. Also, no other study has clarified this hitherto. The aim of the study was to investigate whether the ABI could predict the prognosis in AF patients.

II Methods

A Study design and patient population

This study was a sub-analysis of the impressive predictive value of ABI for clinical long-term outcome in patients with cardiovascular disease examined by the ABI (IMPACT-ABI) study. The IMPACT-ABI study was a retrospective, single-center, observational study. We retrospectively identified 4619 consecutive patients admitted to Shinshu University for any cardiovascular disease between January 2005 and December 2012. All patients had their ABI measured upon admission. Of 4619 patients, 1488 were excluded because of missing data on ABI, and the remaining 3131 patients were enrolled for our study. We obtained the clinical, demographic, and laboratory data from the electronic medical records. The follow-up data were collected from the medical records or by contacting the patients. This study was performed in accordance with the Declaration of Helsinki and was approved by the ethics committee of Shinshu University School of Medicine. As this study was performed retrospectively without written informed consent, the data were analyzed anonymously. This study was registered with the University Hospital Medical Information Network Clinical Trials Registry (UMIN-CTR), as accepted by the International Committee of Medical Journal Editors (UMIN-1D: 000020276). Of the initial 3,131 patients, 2,730 were excluded because of the presence of sinus rhythm on admission, 2 subjects owing to ABI>1.5, and 1 with inadequate ABI data. Thus, we evaluated 398 patients who presented with atrial fibrillation on admission. We divided the study cohort into 2 groups based on the ABI: a low ABI group (ABI<0.92, n = 66), and high ABI group (ABI≥0.92, n = 332). We also analyzed the relation between ABI and prognosis (Fig. 1) and analyzed between CHADS\(_2\)/CHA2DS\(_2\)-VASC score and prognosis. The mean observation period was 4.6±2.7 years.

B Definitions

Body mass index (BMI) was calculated as weight (kg) divided by height squared (m\(^2\)). Smoking habit was defined as previous/current smoking status, as obtained by an interview. Coronary artery disease was defined as a history of angina and/or previous MI. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≤90 mmHg, or the current use of anti-hypertensive agents. Dyslipidemia was defined as serum total cholesterol levels ≥220 mg/dL, low-density lipoprotein cholesterol levels ≥140 mg/dL, high-density lipoprotein cholesterol levels ≤40 mg/dL, triglyceride levels ≥150 mg/dL, or the use of lipid lowering agents. Diabetes mellitus (DM) was defined as fasting blood glucose levels ≥126 mg/dL, casual plasma glucose levels ≥200 mg/dL, HbA1c ≥6.5 %, or the use of insulin or oral hypoglycemic agents. Stroke was defined as ischemic stroke that persisted for ≥24 hours or evidence of infarction on magnetic resonance imaging in accordance with the statement from the American Heart Association/American Stroke Association\(^{10}\). Previous heart failure was defined as a prior diagnosis of heart failure according to the Framingham criteria\(^{11}\) or current treatment for heart failure. Sleep apnea syndrome was defined as apnea hypopnea index (AHI) ≥15 with or without symptoms\(^12\). The echocardiographic parameters included the left ventricular end diastolic diameter (LVDd), left ventricular
end systolic diameter (LVDs), and left ventricular ejection fraction (LVEF), as estimated using the Teichholz method. The estimated glomerular filtration rate (eGFR) was calculated using the Japanese equation to estimate kidney function as follows:

\[ eGFR \text{ (ml/min/1.73 m}^2) = 194 \times \text{serum creatinine}^{-1.094} \times \text{age}^{-0.287} \]

for male patients, and the same formula multiplied by 0.739 for female patients. The all-cause death was defined as any death recorded during the follow-up period. Cardiovascular death was defined as death resulting from acute myocardial infarction (MI), significant cardiac arrhythmia, congestive heart failure (HF), stroke, or other cardiovascular causes.

C ABI measurements

The ABI was determined with the patient at rest in the supine position for at least 10 minutes using the form pulse wave velocity (PWV)/ABI (Omron Colin, Tokyo, Japan), which is an automated oscillometric device with four cuffs that can measure the blood pressure in both upper and lower extremities simultaneously. The ABI was obtained as a ratio of the systolic blood pressure measured in the lower extremity divided by the higher of the two systolic blood pressures measured in the upper extremities. The lower of the two results was used as the patient ABI for all analyses.

D Endpoint

The primary endpoint of this study was the composite of major adverse cardiovascular events (MACE), including cardiovascular death, myocardial infarction, and stroke. The secondary endpoint was all-cause death and cardiovascular death.

E Statistical analysis

Statistical analyses were performed using JMP 13.0 (SAS Institute, Cary, NC, USA). Continuous variables were summarized as mean ± standard deviation if normally distributed and as median and interquartile range if not normally distributed. Normal data distribution was assessed using the Shapiro–Wilk test. Categorical variables were reported as numbers and percentages and were compared using the Chi-square test and the Mann–Whitney U test for continuous variables. Kaplan–Meier plots were calculated from baseline to the time of occurrence of cardiac event and were compared using the log-rank test. Adjusted odd ratios (OR) and 95% confidence interval (CI) for high ABI were obtained by
multiple logistic regression analysis. Univariate Cox proportional hazard analyses were performed to identify the independent predictors of MACE. The optimal Receiver–Operating–Characteristic (ROC) curve cutoff value for prediction of MACE was considered as the value maximizing sensitivity plus specificity. The proportional hazards assumption was verified with log (time) vs. log [–log (survival)] plots. The variables that exhibited a P-value of <0.05 in the univariate analysis were included in the multivariate model. The magnitude of the relationship between the variables and MACE was expressed as the hazard ratio (HR) and 95 % CI. P-values of < 0.05 were considered statistically significant.

Ⅲ Results

A Baseline characteristics

Baseline characteristics are summarized in Table 1. This study enrolled 398 patients, with a median age of 68.0±11.3 years; the majority of patients were male (76.9 %). The causes of admission among participants included acute myocardial infarction (1.5 %), unstable angina pectoris (0.5 %), stable angina pectoris (17.3 %), other atherosclerotic diseases (14.1 %), arrhythmia (38.2 %), heart failure (8.8 %), and others (19.3 %). On the basis of the ABI, all patients were divided into two groups: 66 patients (16.6 %) had low ABI, 332 patients (83.4 %) had high ABI. No significant differences were found in BMI, coronary artery disease, hypertension, dyslipidemia, stroke, and sleep apnea syndrome between each group. However, low ABI values were significantly associated with higher smoking status and higher CHA2DS2-VASc scores. An increased number of patients exhibited diabetes mellitus and congestive heart failure in the low ABI group. In the echocardiographic findings, there were no significant differences in median LVDd, LVDs, and LADs; however, patients with high ABI showed significantly higher LVEF than those with low ABI. A lower ABI was significantly associated with greater levels of creatinine and B-type natriuretic peptide (BNP) and lower levels of hemoglobin, estimated GFR, and HDL–Cho. At discharge, the prescription rates of drugs such as statins, angiotensin converting enzyme inhibitors (ACEIs), and/or angiotensin receptor blockers (ARBs), and amiodarone showed no difference; however, the use of calcium channel blockers was significantly higher in patients with low ABI.

B Outcomes

During the follow-up period (mean 4.6 years), a total of 52 cases (13.1 %) of MACE were recorded, including 37 of cardiovascular death, 4 of MI, and 11 of stroke. Between the two groups, the low ABI group exhibited significantly higher values of MACE, with significant differences in MI; however, no significant differences in cardiovascular death and stroke were observed.

C Predictors of MACE

The univariate Cox proportional hazards analysis showed that low ABI as well as higher CHA2DS2-VASc score (≥2) were independent predictors of MACE. Multivariate Cox proportional hazards analysis shows that adjusted for low ABI (<0.92), CHA2DS2-VASc score ≥2, LVEF, diabetes mellitus, congestive heart failure, and prescription of ACEIs, low ABI independently predicted a poorer prognosis (OR, 2.21; 95 % CI, 1.03–4.66; P = 0.039). (Table 2). The Kaplan–Meier analysis showed that the cumulative event-free survival rate of MACE was markedly lower in patients with a low ABI (Fig. 2).

Ⅳ Discussion

To the best of our knowledge, this is the first report demonstrating the predictive power of low ABI on the prognosis of patients with AF. The major finding of this study was that low ABI could predict cardiovascular events as well as a high (≥2) CHADS2 or CHA2DS2-VASc score. In patients with AF, higher CHADS2 or CHA2DS2-VASc score (≥2) is known to be a predictor of stroke1. On the other hand, the prognosis of AF patients with heart failure is poor15, and it is known that their risk for stroke, hospitalization for heart failure, and death is high16–19. In such patients, higher CHADS2 or CHA2DS2-VASc score (≥2) is also known to be a predictor of hospitalization for worsening of heart failure and mortality2. Ostergren and Sleight20, demonstrated that low ABI predicted
cardiovascular mortality and development of future heart failure.

We showed that low ABI was associated with increased incidence of MACE and all-cause mortality as well as higher CHADS2 or CHA2DS2-VASc score (≥2) in patients with AF. AF and atherosclerosis are known to be a chronic inflammatory disease and inflammation is a triggering factor of atherosclerotic plaque rupture. AF is also associated with the inflammatory state such as obesity, diabetes mellitus, hypertension, heart failure, metabolic syndrome and sedentary lifestyle. In this regard, ABI serves as a measure of atherosclerosis, and thus, lower ABI can indicate vascular abnormalities causing adverse car-

### Table 1 Baseline Characteristics

<table>
<thead>
<tr>
<th>Variables</th>
<th>Overall Population</th>
<th>ABI ≥0.92</th>
<th>ABI &lt;0.92</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean age (years)</td>
<td>68 ± 11</td>
<td>67 ± 11</td>
<td>72 ± 11</td>
<td>0.0031</td>
</tr>
<tr>
<td>Male gender, n (%)</td>
<td>308 (77)</td>
<td>254 (77)</td>
<td>54 (82)</td>
<td>0.420</td>
</tr>
<tr>
<td>Smoking, n (%)</td>
<td>161 (38)</td>
<td>126 (38)</td>
<td>35 (53)</td>
<td>0.0283</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>23 ± 4</td>
<td>23 ± 4</td>
<td>23 ± 4</td>
<td>0.161</td>
</tr>
<tr>
<td>CHADS2 score</td>
<td>1.6 ± 1.2</td>
<td>1.5 ± 1.2</td>
<td>2.2 ± 0.2</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>CHA2DS2-VASc score</td>
<td>2.7 ± 1.7</td>
<td>2.5 ± 1.6</td>
<td>3.8 ± 0.2</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>

### Comorbidities

- Hypertension, n (%) 229 (58) 184 (55) 45 (68) 0.058
- Diabetes mellitus, n (%) 98 (25) 70 (21) 28 (42) 0.0005
- Coronary artery disease, n (%) 130 (33) 103 (31) 27 (41) 0.150
- Dyslipidemia, n (%) 131 (33) 114 (34) 17 (26) 0.198
- Stroke, n (%) 51 (13) 41 (12) 10 (15) 0.546
- Congestive heart failure, n (%) 82 (21) 62 (19) 20 (30) 0.045
- Sleep apnea syndrome, n (%) 12 (3) 10 (4) 2 (4) 1.00

### Medications

- ACEIs, n (%) 80 (20) 64 (20) 16 (24) 0.408
- ARBs, n (%) 143 (36) 117 (36) 26 (39) 0.679
- β-blockers, n (%) 155 (39) 124 (38) 31 (47) 0.215
- CCB, n (%) 116 (29) 87 (27) 29 (44) 0.0079
- Statin, n (%) 113 (28) 91 (28) 22 (33) 0.457
- Amiodarone, n (%) 22 (6) 19 (6) 3 (5) 1.00

### Echocardiographic data

- LVEF (%) 61 ± 16 62 ± 16 58 ± 16 0.044
- LV end-diastolic dimension (cm) 5.0 ± 0.8 5.0 ± 0.8 5.1 ± 1.0 0.110
- LV end-systolic dimension (cm) 3.4 ± 1.4 3.3 ± 1.5 3.4 ± 1.1 0.549
- LA end-systolic dimension (cm) 4.7 ± 1.0 4.7 ± 1.1 4.7 ± 1.0 0.704

### Laboratory data

- Hemoglobin (g/dL) 14.1 ± 2.0 14.2 ± 2.3 13.5 ± 2.3 0.0129
- eGFR (mL/min/1.73 m²) 56 ± 23 58 ± 22 45 ± 24 0.0016
- Total cholesterol (mg/dL) 187 ± 41 187 ± 40 186 ± 44 0.963
- HDL-cholesterol (mg/dL) 51 ± 15 52 ± 14 45 ± 15 0.0007
- LDL-cholesterol (mg/dL) 107 ± 30 106 ± 30 112 ± 34 0.185
- Hemoglobin A₁c (%) 6.27 ± 0.9 6.2 ± 1.0 6.3 ± 0.8 0.317
- BNP (pg/mL) 253 ± 352 232 ± 317 357 ± 479 0.0088
diovascular events and heart failure in the future. Thus, we should be cautious in AF patients with low ABI, because this patient population is more likely to experience cardiovascular events in the future than those with higher ABI. There is a possibility that early intervention in AF patients with low ABI can help in preventing the progression of the cardio­vascular disease.

### A Limitations

While our study included data from a large sample to detect a sufficient number of cardiovascular events, this study has several potential limitations. First, it was a single center, retrospective analysis and involved patients who were hospitalized for cardiovascular disease. Thus, our study population may not be representative of the general population. Second, the patients who were enrolled in this registry had heterogeneous baseline cardiovascular diseases, so sample bias might have affected the results of the study. Third, we measured ABI only once at the

<table>
<thead>
<tr>
<th>Variables</th>
<th>Unadjusted HR (95% CI)</th>
<th>P</th>
<th>Adjusted HR (95% CI)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>0.58 (0.30-1.15)</td>
<td>0.117</td>
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<tr>
<td>Age (years)</td>
<td>1.03 (1.00-1.07)</td>
<td>0.026</td>
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<tr>
<td>BMI (kg/m²)</td>
<td>1.00 (0.93-1.08)</td>
<td>0.999</td>
<td></td>
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<tr>
<td>Smoking</td>
<td>1.05 (0.63-1.73)</td>
<td>0.858</td>
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<tr>
<td>ABI&lt;0.92</td>
<td>2.76 (1.37-5.38)</td>
<td>0.0054</td>
<td>2.21 (1.03-4.66)</td>
<td>0.039</td>
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<tr>
<td>CHADS₂ ≥2</td>
<td>1.69 (0.92-3.16)</td>
<td>0.094</td>
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<tr>
<td>CHA₂DS₂-VASc ≥2</td>
<td>3.35 (1.41-9.90)</td>
<td>0.0047</td>
<td>1.91 (0.78-5.41)</td>
<td>0.184</td>
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<tr>
<td>Coronary artery disease</td>
<td>0.62 (0.34-1.08)</td>
<td>0.090</td>
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<tr>
<td>Hypertension</td>
<td>0.98 (0.59-1.63)</td>
<td>0.938</td>
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<tr>
<td>Dyslipidemia</td>
<td>0.61 (0.34-1.06)</td>
<td>0.081</td>
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<tr>
<td>Diabetes mellitus</td>
<td>1.76 (1.02-3.01)</td>
<td>0.043</td>
<td>0.94 (0.43-1.98)</td>
<td>0.883</td>
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<tr>
<td>Stroke</td>
<td>1.34 (0.64-2.62)</td>
<td>0.428</td>
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<tr>
<td>Congestive heart failure</td>
<td>1.89 (1.06-3.30)</td>
<td>0.031</td>
<td>1.04 (0.46-2.23)</td>
<td>0.923</td>
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**Echocardiographic data**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Unadjusted HR (95% CI)</th>
<th>P</th>
<th>Adjusted HR (95% CI)</th>
<th>P</th>
</tr>
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<tbody>
<tr>
<td>LVEF (%)</td>
<td>0.97 (0.96-0.99)</td>
<td>0.0048</td>
<td>0.97 (0.95-1.00)</td>
<td>0.015</td>
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<tr>
<td>LVDD (cm)</td>
<td>1.00 (0.99-1.01)</td>
<td>0.889</td>
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<tr>
<td>LVDS (cm)</td>
<td>1.01 (0.99-1.03)</td>
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**Medication**

<table>
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<th>P</th>
<th>Adjusted HR (95% CI)</th>
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<tbody>
<tr>
<td>Statin</td>
<td>1.12 (0.56-2.15)</td>
<td>0.747</td>
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</tr>
<tr>
<td>ACEIs</td>
<td>2.41 (1.22-4.66)</td>
<td>0.0126</td>
<td>1.76 (0.81-3.70)</td>
<td>0.140</td>
</tr>
<tr>
<td>ARBs</td>
<td>0.94 (0.48-1.78)</td>
<td>0.858</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beta-blockers</td>
<td>1.01 (0.53-1.89)</td>
<td>0.982</td>
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</tr>
<tr>
<td>CCB</td>
<td>1.50 (0.77-2.83)</td>
<td>0.228</td>
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<tr>
<td>Amiodarone</td>
<td>1.22 (0.28-3.78)</td>
<td>0.760</td>
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**Laboratory data**

<table>
<thead>
<tr>
<th>Variable</th>
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<th>P</th>
<th>Adjusted HR (95% CI)</th>
<th>P</th>
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</thead>
<tbody>
<tr>
<td>Hemoglobin (g/dL)</td>
<td>0.89 (0.77-1.03)</td>
<td>0.124</td>
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<tr>
<td>eGFR (mL/min/1.73 m²)</td>
<td>0.99 (0.87-1.01)</td>
<td>0.397</td>
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</tr>
<tr>
<td>Total cholesterol (mg/dL)</td>
<td>1.00 (0.99-1.01)</td>
<td>0.630</td>
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</tr>
<tr>
<td>LDL-cholesterol (mg/dL)</td>
<td>1.00 (0.98-1.02)</td>
<td>0.512</td>
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<tr>
<td>Hemoglobin A₁ (g/dL)</td>
<td>1.05 (0.75-1.43)</td>
<td>0.771</td>
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<tr>
<td>BNP (pg/mL)</td>
<td>1.00 (0.99-1.00)</td>
<td>0.098</td>
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BMI, body mass index; CHADS₂, congestive heart failure/hypertension/age ≥75 years old/diabetes/stroke [doubled]; CHA₂DS₂-VASc, congestive heart failure/hypertension/age ≥75 years old [doubled]/diabetes/stroke [doubled]/vascular disease/sex category [if female, counted]; ACEIs, angiotensin-converting enzyme inhibitor; ARBs, angiotensin receptor blocker; CCB, calcium channel blocker; LVEF, left ventricular ejection fraction; LVDD, left ventricular end diastolic diameter; LVDS, left ventricular end systolic diameter; LA, left atrial; eGFR, estimated glomerular filtration rate; HDL, high-density lipoprotein; LDL, low-density lipoprotein; BNP, B-type-natriuretic peptide.
time of hospitalization, so the data might not be right in patients with AF. Reports suggest that the accuracy of ABI in patients with atrial fibrillation decreases; however, other reports suggest that atrial fibrillation does not affect ABI data. Finally, we have no data regarding changes of medication or habits that could affect the prognosis during the observation period.

V Conclusion

We found that ABI measured at the time of admission can predict the prognosis of patients with AF, similar to the CHADS2 or CHA2DS2-VASc scores and can be useful to predict cardiovascular events and prevent progression of cardiovascular diseases.

VI Acknowledgement

The authors acknowledge the secretarial and technical assistance of Minako Aono.

References

2) Chao TF, Lin YJ: CHADS (2) and CHA (2)-VASc score in the prediction of clinical outcomes in patients with atrial fibrillation after catheter ablation. Am J Cardiol 58: 2380-2383, 2011
4) Larsen TB, Lip GY: Added predictive ability of the CHA2DS2VASc risk score for stroke and death in patients with


12) Westchester IL: American Academy of Sleep Medicine. The International Classification of Sleep Disorders: Diagnostic and Coding Manual. 2nd ed, American Academy of Sleep Medicine, 2005


22) Dabrowski M, Lewandowski J: Atrial fibrillation does not affect ankle–brachial index measured using the Doppler method. Hypertens Res 41: 60–65, 2018

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