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Ankle brachial pressure index but not brachial-ankle pulse wave velocity is a strong predictor of systemic atherosclerotic morbidity and mortality in patients on maintenance hemodialysis

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ABSTRACT

Background: Ankle brachial pressure index (ABPI) and pulse wave velocity (PWV) have been widely recognized as a marker of systemic atherosclerosis. We examined whether ABPI and brachial-ankle PWV (baPWV) predict individual cardiovascular events in patients on maintenance hemodialysis (HD).
Methods: We prospectively followed-up 445 HD patients undergoing both ABPI and baPWV measurements for up to 5 years. They were divided into 2 groups [group with ABPI >0.9 to ≤1.3 (n = 328) and group with ABPI ≤0.9 or >1.3 (n = 117)] and were also divided into tertiles according to the baPWV level ($T1$: <1850 cm/s; $T2$: 1850–2310 cm/s and $T3$: ≥2310 cm/s).
Results: During the follow-up period (mean 43 ± 17 months), 206 cardiovascular events [cardiac event: 125 (28.1%), cerebrovascular events: 39 (8.8%), and peripheral arterial events: 42 (9.4%)] occurred, and 36 (8.1%) and 42 (9.4%) patients experienced cardiovascular and non-cardiovascular deaths, respectively. Cox multivariable analysis showed that presence of ABPI ≤0.9 or >1.3 was a significant predictor of cardiac events [hazard ratio (HR) 1.78, 95% confidential interval (CI) 1.27–2.49, p = 0.0008], cerebrovascular event (HR 1.95, 95%CI 1.13–3.36, p = 0.017), peripheral arterial event (HR 3.64, 95%CI 2.10–6.29, p < 0.0001), composite endpoint of cardiovascular events (HR 2.22, 95%CI 1.64–2.99, p < 0.0001), cardiovascular mortality (HR 2.42, 95%CI 1.44–4.06, p = 0.0008) and all-cause mortality (HR 1.52, 95%CI 1.03–2.25, p = 0.037). However, baPWV did not predict cardiovascular events on multivariate analysis.
Conclusion: ABPI but not baPWV is useful for risk stratification of systemic atherosclerotic morbidity and mortality in HD patients. Furthermore, ABPI could predict not only individual peripheral arterial events but also cardiac and cerebrovascular events.

1. Introduction

The number of patients with end-stage renal failure who need hemodialysis (HD) keep increasing worldwide, and it is said to have reached two million people now [1]. The risk of cardiovascular disease in patients on HD is reportedly 20–30 higher times than the general person [2]. It is thus clear that patients on HD are at an extremely high risk of cardiovascular disease [3]. Moreover the increase in their medical treatment costs becomes a large problem [4], so it is important to predict the risk of cardiovascular disease in the treatment of such population. The number of HD patients also keeps increasing in Japan. Additionally, the high rate of diabetic nephropathy and high age at starting HD therapy are characteristics resulting in an increase in cardiovascular disease in Japan [5].

Ankle brachial pressure index (ABPI) is a useful inspection to diagnose peripheral artery disease (PAD) as recommended in guidelines of Trans Atlantic Inter-Society Consensus II and American College of Cardiology/American Heart Association [6,7]. PAD is an important manifestation of systemic atherosclerosis, and it is known that patients with ABPI < 0.9 have 3–4 times higher risk of cardiovascular mortality [8]. The utility of ABPI as a predictor of cardiovascular disease is reported not only in general population [9] but also in patients on HD [10–13]. Studies have shown that aortic pulse wave velocity (PWV) as the index of aortic stiffness is also a strong predictor of cardiovascular disease not only in general pop-

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ulation [13] but also in patients with renal insufficiency including HD patients [14–17]. Recently, it is reported that the measurement of handy brachial-ankle PWV (baPWV) is equally useful with that of aortic PWV [18,19]. Though ABPI and PWV are strong predictors of cardiovascular disease, studies which compare both modalities are still few. The purpose of the present prospective study was to examine which had stronger predictive power in patients on HD who are at high risk group of cardiovascular disease.

2. Methods

2.1. Study population

This prospective cohort study was conducted at the Nagoya Kyoritsu Hospital. A total of 445 outpatients on HD undergoing both ABPI and PWV measurement from January 2002 to June 2002 were enrolled in this study. To be eligible for the study, patients had to have received regular HD at least for 3 months just before entry. Moreover, patients had to be clinically stable for 6 months before entry and specifically lack of acute cardiovascular event, cerebrovascular event, infection, ulcer or gangrene in the lower limb, or other active diseases.

The endpoints were cardiovascular events (including cardiac event, cerebrovascular event and peripheral artery event), cardiovascular mortality and all-cause mortality. Cardiac event was defined as coronary revascularization, myocardial infarction, sudden death, and hospitalization or death due to heart failure and/or arrhythmia. A cerebrovascular event was defined as cerebral bleeding and infarction. Peripheral artery event was defined as any intervention due to lower limb ischemia including claudication, ulceration and/or gangrene, and/or amputation of lower limb. Cardiovascular mortality and all-cause mortality were also evaluated. All patients were prospectively followed-up for up to 5 years from the day when both ABPI and baPWV were measured.

2.2. Data collection

Enrolled subjects were divided into 2 groups according to ABPI [group with ABPI >0.9 to ≤1.3 ($n = 328$) and group with ABPI ≤0.9 or >1.3 ($n = 117$)] [7]. Also, they were divided into tertiles according to baPWV level ($T1$: <1850 cm/s; $T2$: ≥1850 to <2310 cm/s; and $T3$: ≥2310 cm/s). Demographic and medical data were obtained from medical records and interviews with patients and/or the patients' primary nephrologists.

2.3. Measurement of ABPI and baPWV

As previously reported [19,20], both ABPI and baPWV were non-invasively measured by trained technicians according to a standard protocol using a Colin Waveform analyzer (form PWV/ABI; Colin Medical Technology, Komaki, Japan), which simultaneously measures bilateral arm and ankle blood pressure and pulse volumes of the brachial and posterior tibial arteries using an oscillometric method, respectively. Briefly the participant was asked to lie flat on an examination table and after 5 min of rest, standard arm blood pressure cuffs were applied to the arm opposite the dialysis blood access, and to each ankle (with the lower end of the bladder within 3 cm of the malloli). These measurements were done at non-dialysis day.

ABPI was calculated as the ratio of ankle systolic pressure divided by arm systolic pressure, in which lower values of ankle pressure were used for the calculation.

In order to calculate spreading pulse wave velocity (PWV) in a tube, it is necessary to record pulse wave at 2 points of either of the tube. The path length between 2 points (L) and pulse transmission time (PTT) were measured. PWV = L/PTT that can be obtained.

In order to obtain brachial-ankle PWV (baPWV), pulse volume waveforms of the brachial and tibial arteries were recorded. Path length between aorta ostium and ankle (L_a), and path length between aorta ostium and brachial (L_b) is possible to be substituted by the calculating formula calculated from the height requested from the data of many example [20].

Each calculating formula is as follows:

$$L_a = 0.819 \times \text{height (cm)} + 12.318$$

$$L_b = 0.2195 \times \text{height (cm)} - 2.0734$$

Time interval between brachium and ankle (T_{ba}) is measured and baPWV can be obtained as follows [21]:

$$\text{baPWV} = \frac{L_a - L_b\,(\text{cm})}{T_{ba}\,(\text{s})}$$

In current study for patients on hemodialysis, we did not measure PWV with the blood access side arm. Not pressing one arm when measuring PWV seems more natural than pressing both arms, because it does not close the arterial system, so the influence of the reflected wave is weakened.

2.4. Statistical analysis

Continuous variables were presented as mean ± standard deviations.

Differences in frequencies were tested by means of chi-square test, and analysis of variance was used to compare distributed variables. Prognostic factors of survival were identified by use of the Cox proportional hazard regression model, and the multivariate model included all baseline valuables with $p < 0.05$ by univariate analysis. The assumption of proportional hazards over time was verified before the analysis was performed and was met by all covariates. The assumption concerning linearity of continuous covariates was also verified before analysis. Survival curves were estimated by Kaplan–Meier product-limit method and compared by the log-rank test.

Statistical significance is defined as $p < 0.05$. SPSS ver.18 (SPSS, Chicago, IL, USA) was used for all statistical analyses.

3. Results

The baseline characteristics of the study population at the time of inclusion are listed in Table 1. The age at inclusion was 63 ± 11 years old, and duration of HD was 6.4 ± 6.5 years. In a total of 30 first consecutive patients, ABI and baPWV were measured once again 1 week after the first analysis for evaluation of reproducibility. The variabilities of ABI and baPWV were well correlated [mean difference 0.04 ± 0.08, $r = 0.93$ ($p < 0.0001$) and mean difference 43 ± 224 cm/s, $r = 0.92$ ($p < 0.0001$), respectively].

The number of patients with ABPI >0.9 to ≤1.3 and those with ABPI ≤0.9 or >1.3 was 328 and 117, respectively. The group with ABPI ≤0.9 or >1.3 was found to be significantly associated with increased age, high prevalence of diabetes, and high rate of previous coronary artery disease (Table 1).

When enrolled patients were divided into tertiles according to baPWV level ($T1$: <1850 cm/s; $T2$: ≥1850 to <2310 cm/s and $T3$: ≥2310 cm/s), each number of patients was 148 (Table 1). Increased baPWV level was found to be significantly associated with increased age and diabetes. Duration of dialysis, smoking status, hypertension, previous coronary artery disease, previous cerebrovascular disease, and serum cholesterol levels had no relation to the level of baPWV.

During follow-up period (43 ± 17 months), cardiovascular events occurred in 206 (46.3%) patients [cardiac event: 125 (28.1%),

Table 1
Baseline characteristics among groups according to stratified ABPI and baPWV.

	All patients (n = 445)	ABPI >0.9 to ≤1.3 (n = 328)	ABPI ≤0.9 or >1.3 (n = 117)	p value	Tertile 1 baPWV < 1850 cm/s (n = 148)	Tertile 2 1850≦ baPWV < 2310 cm/s (n = 148)	Tertile 3 baPWV ≧ 2310 cm/s (n = 149)	p value
Male (%)	59.3	60.0	57.3	0.54	60.1	57.4	52.3	0.51
Age (years)	63 ± 11	62 ± 12	65 ± 11	0.0052	58 ± 12	63 ± 10	67 ± 10	< 0.0001
Duration of dialysis (years)	6.4 ± 6.5	6.4 ± 6.7	6.4 ± 6.3	0.97	6.4 ± 6.7	6.4 ± 6.1	6.4 ± 6.9	0.99
Diabetes (%)	47.5	43.9	57.2	0.016	35.1	45.9	51.7	0.039
Hypertension (%)	76.3	77.7	72.7	0.30	78.4	82.4	83.2	0.59
Smoking (%)	28.3	27.1	31.6	0.36	31.1	29.7	26.2	0.74
Body mass index (kg/m^2)	20.6 ± 2.8	20.6 ± 2.9	20.7 ± 3.2	0.79	20.8 ± 3.0	20.9 ± 2.9	20.1 ± 2.8	0.085
Previous coronary artery disease (%)	24.9	22.9	31.6	0.049	29.1	29.1	30.2	0.95
Previous cerebrovascular disease (%)	7.9	7.3	9.4	0.46	6.8	6.8	12.1	0.27
Hematocrit (%)	31.6 ± 3.6	30.8 ± 4.0	31.4 ± 3.6	0.17	31.5 ± 3.4	31.6 ± 4.0	31.6 ± 3.7	0.99
Albumin (g/dl)	3.5 ± 0.3	3.6 ± 0.4	3.5 ± 0.4	0.27	3.6 ± 0.3	3.6 ± 0.4	3.6 ± 0.3	0.42
Triglyceride (mg/dl)	117 ± 69	115 ± 66	123 ± 79	0.36	117 ± 82	115 ± 58	118 ± 68	0.95
Total cholesterol (mg/dl)	167 ± 39	165 ± 40	167 ± 36	0.71	171±37	162 ± 40	168 ± 43	0.17
High-density lipoprotein cholesterol (mg/dl)	44 ± 14	45±15	42 ± 12	0.053	101 ± 31	94 ± 32	99 ± 34	0.20
Low-density lipoprotein cholesterol (mg/dl)	98 ± 32	95 ± 32	99 ± 29	0.29	44 ± 14	44 ± 15	45 ± 14	0.90
C-reactive protein (mg/l)	6.9 ± 15.3	6.3 ± 10.9	6.9 ± 11.2	0.71	6.2 ± 13.6	7.6 ± 18.1	7.3 ± 14.3	0.79
Parathyroid hormone (ng/ml)	126 ± 129	132 ± 137	105 ± 103	0.067	110 ± 94	95 ± 86	108 ± 103	0.44

cerebrovascular event: 39 (8.8%), and peripheral artery event: 42 (9.4%)]. A total of 78 patients (17.5%) died during the follow-up period, 36 patients (8.1% of total patients) of them with fatal cardiovascular events: 8 deaths attributed to congestive heart failure, 6 to fatal arrhythmia, 5 to myocardial infarction, 3 to sudden death, 1 to valve disease, 1 to aortic aneurysm, 10 to cerebrovascular disease and 2 to peripheral artery disease. The 42 died by non-cardiovascular events: 21 deaths attributed to infection, 8 to malignancy, 7 to digestive disorder, 4 to respiratory disorder and 2 to cachexia. Upon Kaplan–Meier analysis, the frequency of cardiac events for 5 years was 25.5% in patients with ABPI >0.9 to ≤1.3 and 46.4% in those with ABPI ≤0.9 or >1.3 (p < 0.0001) (Fig. 1A). Similarly, rates of other endpoints for 5 years were significantly different between the two groups according to the ABPI levels (Fig. 1B–F).

Table 2 shows Cox proportional hazards analysis of the covariates using the model includes ABPI to predict the endpoint of all cardiovascular event (including cardiac event, cerebrovascular event and peripheral artery event), cardiovascular mortality and all-cause mortality. The group with ABPI ≤0.9 or >1.3 was significantly associated with higher incidence of each endpoint. Interestingly, statistical significance was only seen in incidence of cerebral infarction when the etiology of cerebrovascular event was divided into cerebral infarction and hemorrhage (cerebral infarction: hazard ratio 3.08, 95% confidential interval 1.47–6.47, p = 0.0029 and hemorrhage: hazard ratio 2.04, 95% confidential interval 0.58–7.24, p = 0.26).

Table 3 shows Cox proportional hazards analysis of the covariates using the model includes baPWV to predict cardiovascular events (including cardiac event, cerebrovascular event and peripheral artery event), cardiovascular mortality and all-cause mortality. baPWV was an only predictor of all-cause mortality on univariate. However PWV did not reach statistical significance in multivariate analysis.

4. Discussion

Previous studies have reported that atherosclerosis such as peripheral artery disease has been reported as an independent predictor of all-cause mortality and cardiovascular events in patients with end-stage renal failure [22,23]. In addition, such patients have a higher prevalence of coronary artery disease without symptoms [24]. Moreover, HD influences limb amputation and mortality [25,26]. In such situation, a predictive value has been warranted and we hypothesized that measures such as ABPI and/or baPWV might be effective to predict clinical prognosis in HD patients.

The main finding of the present study was that ABPI ≤0.9 or >1.3 which was considered as abnormal levels was an independent predictor of not only worsening peripheral artery disease but also other cardiovascular events including cardiac events and cerebrovascular events in patients on maintenance HD. In an earlier large-scale study of non-HD population, abnormal ABPI level did not predict either cardiac events or cerebrovascular events [8]. On the contrast, ABPI was a stronger predictor of all cardiovascular events in patients on HD who have advanced atherosclerosis in the current study. Another study showed that ABPI is also a strong predictor of cardiovascular mortality and all-cause mortality in patients on HD [11]. We clarified that ABPI was a strong predictor not only

Table 2
Predictive values of ABPI ≤0.9 or >1.3 for each endpoint in the Cox proportional hazard analysis.

	Non-adjusted		Adjusted	
	HR (95%CI)	p value	HR (95%CI)	p value
Cardiac events	2.00 (1.43–2.78)	<0.0001	1.78 (1.27–2.49)	0.0008
Cerebrovascular events	2.04 (1.19–3.50)	0.0097	1.95 (1.13–3.36)	0.017
Peripheral arterial events	4.08 (2.37–7.01)	<0.0001	3.64 (2.10–6.29)	<0.0001
All cardiovascular events	2.34 (1.76–3.12)	<0.0001	2.22 (1.64–2.99)	<0.0001
Cardiovascular mortality	2.53 (1.59–4.04)	0.0001	2.42 (1.44–4.06)	0.0008
All-cause mortality	1.76 (1.22–2.54)	0.0024	1.52 (1.03–2.25)	0.037

Adjusted for all baseline variables with p < 0.05 on Cox univariate analysis.

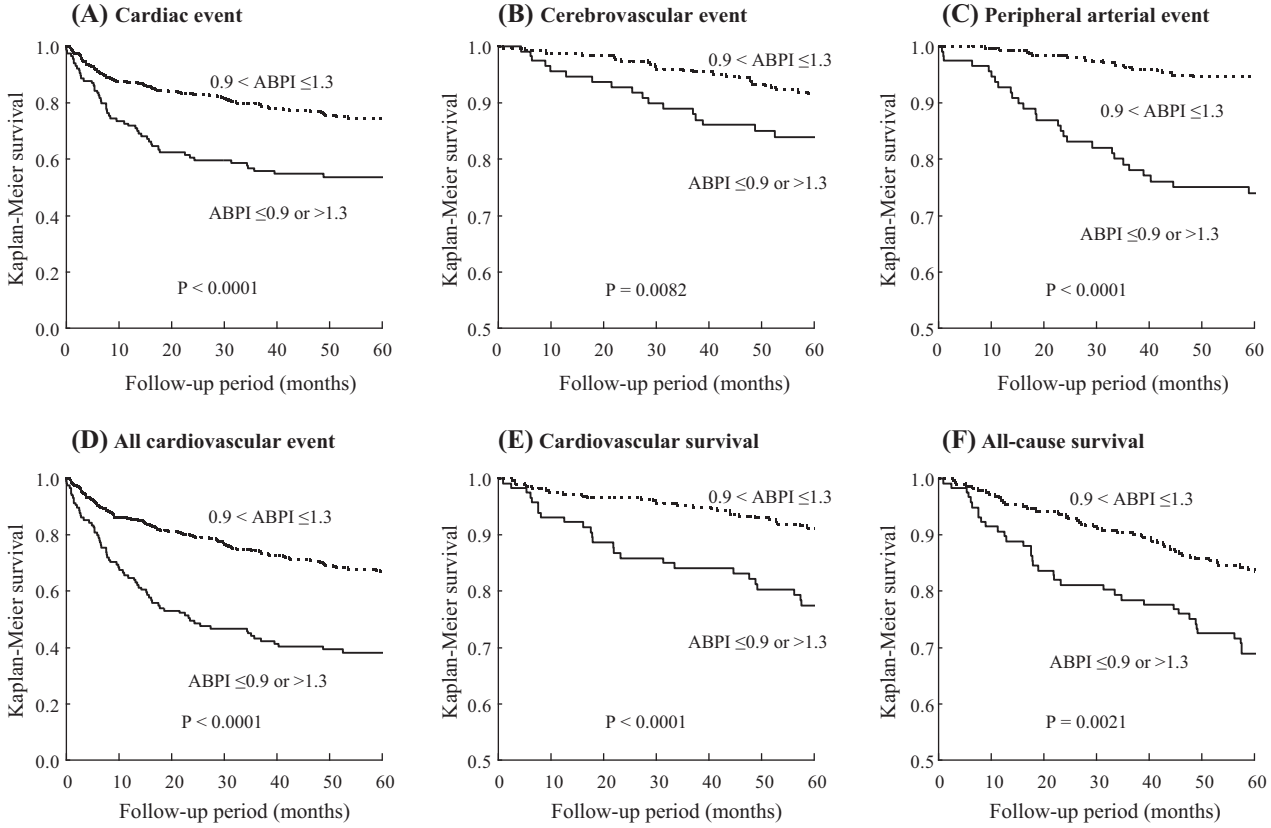

Fig. 1. (A) Kaplan–Meier curves of freedom from cardiac event. In 5-year follow-up period, incidences of cardiac events in HD patients with ABPI >0.9 to ≤1.3 and those with ABPI ≤0.9 or >1.3 were 25.5% and 46.4%, respectively ($p < 0.0001$). (B) Kaplan–Meier curves of freedom from cerebrovascular event. In 5-year follow-up period, incidences of cerebrovascular events in HD patients with ABPI >0.9 to ≤1.3 and those with ABPI ≤0.9 or >1.3 were 9.3% and 16.1% ($p = 0.0082$). (C) Kaplan–Meier curves of freedom from peripheral arterial event. In 5-year follow-up period, incidences of peripheral artery events in HD patients with ABPI >0.9 to ≤1.3 and those with ABPI ≤0.9 or >1.3 were 5.3% and 26.1% ($p < 0.0001$). (D) Kaplan–Meier curves of freedom from all cardiovascular event. In 5-year follow-up period, incidences of cardiovascular events in HD patients with ABPI > 0.9 to ≤1.3 and those with ABPI ≤0.9 or >1.3 were 32.9% and 61.9% ($p < 0.0001$). (E) Kaplan–Meier curves of cardiovascular survival. In 5-year follow-up period, cardiovascular death in HD patients with ABPI >0.9 to ≤1.3 and those with ABPI ≤0.9 or >1.3 were 8.8% and 22.7% ($p < 0.0001$). (F) Kaplan–Meier curves of all-cause survival. In 5-year follow-up period, all-cause death in HD patients with ABPI >0.9 to ≤1.3 and those with ABPI ≤0.9 or >1.3 were 16.1% and 31.1% ($p = 0.0021$).

Table 3
Cox proportional hazard analysis using the model includes baPWV.

	Non-adjusted		Adjusted	
	HR (95%CI)	*p* value	HR (95%CI)	*p* value
Cardiac events				
baPWV (versus *T*1)	Reference	0.26		
*T*2	1.06 (0.65–1.75)		–	
*T*3	1.43 (0.89–2.29)			
Celebrovascular events				
PWV (versus *T*1)	Reference	0.68		
*T*2	1.27 (0.56–2.84)		–	
*T*3	1.43 (0.61–3.36)			
Peripheral artery events				
baPWV (versus *T*1)	Reference	0.74		
*T*2	1.31 (0.57–2.99)		–	
*T*3	1.34 (0.59–3.06)			
All cardiovascular events				
baPWV (versus *T*1)	Reference	0.089		0.84
*T*2	1.07 (0.69–1.66)		1.16 (0.62–1.74)	
*T*3	1.52 (1.01–2.29)		1.51 (0.68–1.97)	
Cardiovascular death				
PWV (versus *T*1)	Reference	0.089		0.15
*T*2	2.15 (0.88–5.19)		3.22 (0.83–12.44)	
*T*3	2.39 (0.92–5.54)		3.61 (0.97–13.45)	
All-cause death				
PWV (versus *T*1)	Reference	0.0073		0.21
*T*2	1.76 (0.89–3.48)		2.02 (0.69–5.91)	
*T*3	2.73 (1.43–5.16)		2.63 (0.89–7.80)	

Adjusted for all baseline variables with $p < 0.05$ on Cox univariate analysis.

of both cardiovascular mortality and all-cause mortality but also of all cardiovascular events in patients on HD. It is natural that abnormal ABPI level would predict worsening of peripheral artery disease, but we first found that the group with ABPI ≤0.9 or >1.3 was associated with higher incidence of cardiac disease and cerebrovascular disease, too. Moreover, the hazard ratio of presence of ABPI ≤0.9 or >1.3 for any endpoint was higher than any other risk factors of atherosclerosis. In addition, in line with a previous report [14] which indicated a significant relation between PWV and cardiovascular mortality, baPWV was an only predictor of all-cause mortality on univariate Cox analysis in the present study. We found that baPWV was not an independent predictor for any endpoint in patients on HD when analyzed in combination with ABPI. Thus, baPWV was less useful for risk stratification of systemic atherosclerotic morbidity and mortality in patients on maintenance HD, compared to ABPI.

PWV is measured by various parts besides aortic in recent years, and those comparison researches are performed [15,17,18]. Any PWV index other than aortic did not show a strong relation to the risk. Ito et al. reported that baPWV could underestimate arterial stiffness in hypertensive patients with a history of cardiovascular events [18]. The blood pressure of a patient on HD changes intensely during a day (especially, before and after dialysis), and the systolic arterial pressure influences PWV. Cardiac valvular disease in patients on HD keeps increasing, however, PWW of patients with heart valve disease, (especially aortic valvular disease) is not so useful as the index of stiffness of the artery. Also, PWV of patients with myocardial infarction is likely to be represented lower than the actual. These factors might explain the reason why baPWV was less useful for risk stratification of systemic atherosclerotic morbidity and mortality in patient on HD.

Some limitations should be discussed. We used data on measurement of the ABPI/baPWV performed once at the start of the study. This issue was the main limitation. This study consisted of only 445 patients. Thus, lower statistical power may exist. Moreover, all enrolled patients were Japanese. They have better prognosis compared to subjects in other countries [22]. As to the baPWV analysis, we used tertiles but not two groups because there has been no appropriate cut-off point in the baPWV. However, the arbitrary cut-off might be used for baPWV and this could affect the results.

In conclusion, we clarified that ABPI but not baPWV was a strong predictor of systemic atherosclerotic morbidity and mortality in HD patients. Furthermore, ABPI could predict not only deterioration of peripheral artery disease but also cardiac and cerebrovascular events individually.

ABPI is an extremely handy examination, and screening is also easy. Thus, we can easily perform risk stratification to predict clinical prognosis in HD patients with an extremely high risk of cardiovascular disease by measuring ABPI. It seems that regularly understanding HD patient's risk of cardiovascular disease leads to not only the improvement of patient's prognosis but also to decrease the medical treatment cost by preventing coexisting disease.

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