Determinants of Peripheral Arterial Stiffness in Patients With Chronic Kidney Disease in Southern Taiwan

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High prevalences of peripheral artery occlusive disease (PAOD) and increased arterial stiffness have been reported in patients with chronic kidney disease (CKD). However, these have not been assessed in Taiwan where the prevalence of CKD is high. The aim of this study was to investigate the determinants of PAOD and arterial stiffness in patients with CKD in southern Taiwan. We enrolled 169 patients with stage 3–5 CKD in one regional hospital. Ankle-brachial index (ABI) and brachial-ankle pulse wave velocity were measured using an ABI-form device (Colin VP1000). In multivariate analysis, ABI <0.9 was positively correlated with the presence of diabetes mellitus ($p=0.014$) and negatively correlated with the estimated glomerular filtration rate (eGFR) ($p=0.049$), and increased brachial-ankle pulse wave velocity was correlated with increased age, diabetes mellitus, increased systolic blood pressure, decreased pulse pressure and decreased eGFR. This study identified determinants of PAOD and arterial stiffness in patients with CKD in one hospital in southern Taiwan. In addition to the traditional atherosclerotic risk factors, decreased eGFR was also correlated with PAOD and increased arterial stiffness in these patients.

Key Words: ankle-brachial index, arterial stiffness, brachial-ankle pulse wave velocity, chronic kidney disease, peripheral artery occlusive disease

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Cold extremities, peripheral numbness and frequent muscle cramps are very commonly reported by patients with chronic kidney disease (CKD), and these symptoms can be caused by peripheral ischemia related to peripheral artery occlusive disease (PAOD). This problem is becoming more severe because of the increasing number of diabetic and elderly patients within the CKD and the dialysis populations. Previous studies have reported the high prevalence of PAOD and increased arterial stiffness in patients with renal insufficiency [1–6]. Therefore, appropriate evaluation of PAOD is important in the management of those ischemic symptoms. The definition of PAOD included the presence of obstructive lesions in the lower extremities. The ankle-brachial index (ABI) was reported to be a good marker for atherosclerosis and useful in the diagnosis of PAOD, and an ABI <0.9 has been used to identify this condition in clinical practice and in epidemiologic studies [7–9]. Brachial-ankle pulse wave velocity (baPWV) has also been reported to be a good marker for atherosclerosis or arterial stiffness [10,11]. Careful evaluation using both techniques may rationally provide more accurate and better evaluation

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of peripheral vascular damage. We have recently reported the associated risk factors for abnormal ABI in hemodialysis patients [12], but a similar study in CKD patients has not been done in Taiwan. The aim of this study was to evaluate the determinants of arterial stiffness using ABI and baPWV in CKD patients in southern Taiwan. Our results show that the presence of decreased glomerular filtration rate (GFR), besides old age and diabetes, is strongly correlated with both ABI and baPWV and is an important determinant of arterial stiffness in our CKD patients.

METHODS

Study patients and design

The study was conducted in a regional hospital in southern Taiwan. We enrolled 169 (114 males and 55 females) outpatients with stage 3–5 CKD according to the National Kidney Foundation-Kidney Disease Outcomes Quality Initiative guidelines [13]. The protocol was approved by our Institutional Review Board and all enrolled patients gave written, informed consent.

ABI and baPWV measurement

ABI and baPWV values were measured using an ABI-form device (VP1000; Colin Co. Ltd., Komaki, Japan), which automatically and simultaneously measures the blood pressure (BP) in both arms and ankles using an oscillometric method [14–16]. An ABI <0.9 is 95% sensitive and 100% specific for angiographically documented PAOD in identifying healthy individuals [17]. Meanwhile, baPWV is a known marker for arterial stiffness, and was developed to screen large populations [10,11]. ABI was calculated as the ratio of the ankle systolic BP divided by the arm systolic BP, and the lowest ankle systolic BP value was used in the calculation. To measure baPWV, pulse waves obtained from the brachial and tibial arteries were recorded simultaneously, and the transmission time was determined as the time interval between the initial increase in brachial and tibial waveforms. The transmission distance from the arm to each ankle was calculated according to body height. The baPWV value was automatically calculated as the transmission distance divided by the transmission time. After obtaining bilateral baPWV values, the highest value was considered representative for each subject. The ABI and baPWV measurements were done once in each patient.

Collection of demographic, medical and laboratory data

Demographic and medical data including age, sex, smoking history (ever *vs.* never) and comorbid conditions were obtained from medical records and interviews with patients. The study subjects were defined as having diabetes mellitus (DM) if the subject's medical data included an International Classification of Diseases (9^{th} edition) code of 250.00 to 250.90, or if the fasting blood glucose level was >126 mg/dL, or if hypoglycemic agents were used to control blood glucose levels. Hypertension was defined as an International Classification of Diseases code of 401.9, diagnosis by a physician, systolic BP ≥140 mmHg or diastolic BP ≥90 mmHg, or the subject was using antihypertensive medications irrespective of BP. Cerebrovascular disease was defined as a history of cerebrovascular accident including cerebral bleeding and infarction. Coronary artery disease was defined as a history of angina, ischemic electrocardiogram change, history of myocardial infarction, or having undergone coronary bypass surgery or angioplasty. Body mass index (BMI) was calculated as the ratio of weight in kilograms divided by the square of height in meters. Obesity was defined as BMI >27 kg/m^2 according to criteria modified for Taiwanese subjects [18]. Laboratory data were measured from fasting blood samples using an autoanalyzer (COBAS Integra 400; Roche Diagnostics GmbH, Mannheim, Germany). The estimated GFR (eGFR) was calculated using the simplified formula developed in the Modification of Diet in Renal Disease study [19]. Serum intact parathyroid hormone concentration was evaluated using a commercially available two-site immunoradiometric assay (CIS Bio International, Gif Sur Yvette, France). Blood samples were obtained within 1 month of enrolment. Urine albumin and creatinine were measured on a spot urine sample by an autoanalyzer (COBAS Integra 400 Plus; Roche Diagnostics, North America) and albuminuria was defined as the ratio of urine albumin to creatinine of ≥30 mg/g.

Statistical analysis

Statistical analysis was performed using SPSS version 12.0 (SPSS Inc., Chicago, IL, USA) for Windows. Data are expressed as percentages or mean ± standard

deviation. Logistic regression analysis and linear regression analysis were used to identify the major determinants of ABI < 0.9 and baPWV, respectively. Age, sex and independent variables with a p value < 0.2 in the univariate analysis were selected for multivariate analysis. A significant difference was considered when the p value was < 0.05.

Results

The clinical characteristics of the patients in this study are shown in Table 1. A total of 169 non-dialyzed CKD patients were included. The mean age was 67.6 ± 12.4 years and there were 114 males and 55 females.

Table 1. Characteristics of all 169 study patients

Age (yr)	67.6 ± 12.4
Male sex (%)	67.5
Smoking history (%)	56.0
Diabetes mellitus (%)	51.5
Hypertension (%)	84.6
Coronary artery disease (%)	20.7
Cerebrovascular disease (%)	20.1
CKD stage	
Stage 3 (%)	47.3
Stage 4 (%)	29.6
Stage 5 (%)	23.1
Systolic BP (mmHg)	142.7 ± 21.8
Diastolic BP (mmHg)	79.2 ± 12.3
Pulse pressure (mmHg)	62.8 ± 18.3
Body mass index > 27 (kg/m^2) (%)	33.1
Laboratory parameters	
Albumin (g/dL)	3.9 ± 0.4
Fasting glucose (mg/dL)	122.7 ± 47.5
Triglyceride (mg/dL)	167.9 ± 100.1
Cholesterol (mg/dL)	197.1 ± 47.4
eGFR (mL/min/1.73 m^2)	28.2 ± 14.8
Hematocrit (%)	35.9 ± 6.9
Calcium (mg/dL)	9.5 ± 0.8
Phosphate (mg/dL)	4.0 ± 1.0
Ca × P (mg^2/dL^2)	36.7 ± 10.5
Uric acid (mg/dL)	8.3 ± 2.2
PTH (pg/mL)	75.6 ± 117.3
ABI < 0.9 (%)	8.3
baPWV (cm/s)	2,040.3 ± 507.2

CKD = chronic kidney disease; BP = blood pressure; eGFR = estimated glomerular filtration rate; PTH = parathyroid hormone; ABI = ankle-brachial index; baPWV = brachial-ankle pulse wave velocity.

More than half of our patients (51.5%) were diabetic and 84.6% received medications for high blood pressure. Pre-existing and documented coronary arterial and cerebrovascular diseases were noted in 20.7% and 20.1% of patients, respectively. Overall, 47.3% of patients had mildly impaired renal function (CKD stage 3) and the other patients had more severe disease (29.6% in stage 4, 23.1% in stage 5). Obesity (BMI > 27 kg/m^2) was noted in 33.1% of patients. The biochemical data are presented in Table 1. All of the patients underwent ABI and baPWV measurement and the prevalence of ABI < 0.9 was 8.3%. The average baPWV value was 2,040.3 ± 507.2 cm/s.

Table 2 shows the logistic regression analysis for clinical determinants of ABI < 0.9 in our patients. In univariate analyses, ABI < 0.9 was significantly associated with decreased eGFR, older age, a history of coronary artery disease or cerebrovascular disease, decreased serum albumin, decreased cholesterol and decreased hematocrit level. However, multiple logistic regression analysis showed that ABI < 0.9 was only positively correlated with the presence of DM and negatively correlated with eGFR.

The analysis of determinants of baPWV in our patients is presented in Table 3. In the univariate analyses, baPWV was positively correlated with age, DM, history of cerebrovascular disease, systolic and diastolic BP, pulse pressure and albuminuria, and negatively correlated with eGFR, hematocrit and obesity. Stepwise multivariate analysis showed that baPWV was correlated with older age, DM, increased systolic BP, decreased pulse pressure and decreased eGFR.

Discussion

PAOD is prevalent in CKD patients and contributes to multiple clinical ischemic symptoms and may affect the rate of mortality of these patients [1,5,20]. Besides clinical evaluation, ABI and baPWV are the two most commonly used surrogate markers for peripheral arterial stiffness—an advocate of PAOD in this study. ABI < 0.9 has been shown to indicate PAOD, as documented by angiography, with high sensitivity and specificity [17]. On the other hand, the measurement of baPWV was found to be a simple and reliable marker for arterial stiffness and was used as a screening tool in population studies [10,11]. In the present study, we measured ABI and baPWV in CKD patients

Table 2. Determinants of ankle-brachial index < 0.9 in study patients

Parameter	Univariate		Multivariate	
	Odds ratio (95% CI)	*p*	Odds ratio (95% CI)	*p*
Age (per 1 yr)	1.060 (1.003–1.122)	0.040	1.020 (0.923–1.126)	0.701
Male *vs.* female	1.851 (0.495–6.926)	0.360	2.993 (0.290–30.880)	0.357
Smoking (ever *vs.* never)	0.406 (0.130–1.026)	0.121	0.206 (0.023–1.863)	0.160
Diabetes mellitus	2.532 (0.762–8.421)	0.130	12.088 (1.653–88.397)	0.014
Hypertension	1.099 (0.231–5.225)	0.905	–	–
Coronary artery disease	3.259 (1.050–10.116)	0.041	2.163 (0.435–10.743)	0.346
Cerebrovascular disease	3.402 (1.094–10.582)	0.034	1.501 (0.287–7.858)	0.631
Systolic BP (per 1 mmHg)	1.002 (0.976–1.028)	0.878	–	–
Diastolic BP (per 1 mmHg)	0.955 (0.910–1.002)	0.059	0.939 (0.879–1.004)	0.064
Pulse pressure (per 1 mmHg)	1.009 (0.979–1.040)	0.565	–	–
Body mass index > 27 (kg/m^2)	0.312 (0.067–1.444)	0.136	0.125 (0.014–1.116)	0.063
Laboratory parameters				
Albumin (per 1 g/dL)	0.205 (0.075–0.559)	0.002	0.174 (0.028–1.060)	0.058
Fasting glucose (mg/dL)	1.003 (0.993–1.014)	0.509	–	–
Triglyceride (per 1 mg/dL)	1.001 (0.996–1.006)	0.734	–	–
Cholesterol (per 1 mg/dL)	0.983 (0.968–0.998)	0.028	0.989 (0.972–1.007)	0.220
eGFR (per 1 mL/min/1.73 m^2)	0.952 (0.911–0.995)	0.028	0.922 (0.850–1.000)	0.049
Hematocrit (per 1%)	0.918 (0.845–0.998)	0.045	1.109 (0.917–1.342)	0.285
Calcium (per 1 mg/dL)	1.026 (0.506–2.080)	0.944	–	–
Phosphate (per 1 mg/dL)	0.869 (0.476–1.587)	0.648	–	–
Ca × P (per 1 mg^2/dL^2)	1.006 (0.955–1.061)	0.811	–	–
Uric acid (per 1 mg/dL)	1.108 (0.876–1.401)	0.392	–	–
PTH (per 1 pg/mL)	1.001 (0.996–1.005)	0.799	–	–
Albuminuria	2.071 (0.444–9.665)	0.354	–	–

CI = confidence interval; BP = blood pressure; eGFR = estimated glomerular filtration rate; PTH = parathyroid hormone.

and found that DM and low eGFR were closely correlated with these two parameters, as markers for arterial stiffness. It has been stated that decreased eGFR may predispose to atherosclerosis and arteriosclerosis, and multiple pathogenic mechanisms are involved in this process, including an imbalance between calcium and phosphate, secondary hyperparathyroidism, elevated homocysteine levels, lipoprotein(a) metabolism and alterations in inflammatory and coagulation pathways [21]. Other factors such as fluid overload, alterations in the angiotensin and endothelin systems, malnutrition, elevated uremic toxins, oxidative stress and insulin resistance have also been proposed [22]. A decrease in eGFR may, by itself, be considered as a marker for atherosclerosis and arteriosclerosis contributing to arterial stiffness.

In our multivariate analysis of baPWV and arterial stiffness (Table 2), increased age, DM, increased systolic BP, decreased pulse pressure and eGFR were found to be closely correlated. Most of our findings were consistent with previous reports, except for pulse pressure, which is reported differently in various papers [2,4,5,20,23–26]. Stancanelli et al evaluated the associated risk factors for aortic stiffness in 31 patients with CKD and found that increased pulse pressure was an independent risk factor for aortic stiffness [27]. Kawamoto et al also used aortic PWV to evaluate arterial stiffness, and found that systolic and diastolic BP, but not pulse pressure, were correlated with increased aortic PWV [2]. In Nakagawa et al's study, the results also showed no significant correlation between baPWV and aortic stiffness [4]. Based on our own results, the increased pulse pressure might negatively influence arterial stiffness. The discrepancy between our own and other studies cannot be explained in detail at the present time, but

Table 3. Determinants of brachial-ankle pulse wave velocity in the study patients

Parameter	Univariate		Multivariate (stepwise)	
	Standardized coefficient β	*p*	Standardized coefficient β	*p*
Age (per 1 yr)	0.433	<0.001	0.566	<0.001
Male *vs.* female	–0.085	0.275	–	–
Smoking (ever *vs.* never)	–0.018	0.813	–	–
Diabetes mellitus	0.271	<0.001	0.223	0.001
Hypertension	0.144	0.062	–	–
Coronary artery disease	0.087	0.261	–	–
Cerebrovascular disease	0.155	0.045	–	–
Systolic BP (per 1 mmHg)	0.466	<0.001	0.826	<0.001
Diastolic BP (per 1 mmHg)	0.299	<0.001	–	–
Pulse pressure (per 1 mmHg)	0.357	<0.001	–0.606	<0.001
Body mass index >27 (kg/m^2)	–0.222	0.004	–	–
Laboratory parameters				
Albumin (per 1 g/dL)	–0.083	0.292	–	–
Fasting glucose (mg/dL)	0.035	0.659	–	–
Triglyceride (per 1 mg/dL)	0.077	0.322	–	–
Cholesterol (per 1 mg/dL)	–0.126	0.105	–	–
eGFR (per 1 mL/min/1.73 m^2)	–0.184	0.017	–0.136	0.030
Hematocrit (per 1%)	–0.229	0.003	–	–
Calcium (per 1 mg/dL)	0.030	0.707	–	–
Phosphate (per 1 mg/dL)	–0.025	0.755	–	–
Ca × P (per 1 mg^2/dL^2)	0.022	0.766	–	–
Uric acid (per 1 mg/dL)	0.100	0.204	–	–
PTH (per 1 pg/mL)	0.058	0.471	–	–
Albuminuria	0.156	0.046	–	–

BP = blood pressure; eGFR = estimated glomerular filtration rate; PTH = parathyroid hormone.

may be related to the different study designs, different patients recruited and the diverse associated diseases in these patients.

Albuminuria is a known risk factor for atherosclerosis and the subsequent development of cardiovascular diseases [28,29]. Some studies had noted increased baPWV in subjects with hypertension or DM along with proteinuria or albuminuria [30,31]. Ohya et al [6] examined the relationship between proteinuria and arterial stiffness in 3,387 people attending a health program in Japan, and found that proteinuria and low creatinine clearance were independently associated with increased baPWV. Smith et al [32] found that albuminuria and decreased renal function in patients with type 2 diabetes were significantly associated with baPWV. In our study, albuminuria was not correlated with baPWV; thus, albuminuria was not a critical determinant of increased baPWV in our patients.

The prevalence rate of peripheral arterial stiffness was 8.3% in our study, which is lower than the 19.2–38% in other reports [1,3,5,20]. ABI <0.9 was reported to be a good surrogate marker for angiographically confirmed PAOD [33]. However, falsely high values were common. Another source of uncertainty is the presence of incompressible "stiff" arteries (detected by the measuring cuff) at the ankle level, which are common in patients with extensive vascular calcification, such as patients with diabetes or being treated with hemodialysis [26,34]. In the study by Fishbane et al, the prevalence of ABI <0.9 was 38% in 144 hemodialysis patients and, of these, 25.4% were diabetic [7]. The marked difference between our and their data (8.3% *vs.* 38%) may be explained by the different patient populations (undialyzed patients in our study *vs.* patients undergoing hemodialysis in their study), and the different prevalence of DM (51.5% *vs.* 25.4%,

respectively), which predispose to stiff lower limb vessels. Our own previous report revealed a higher prevalence rate of low ABI in dialysis patients compared with CKD patients [35]. In a study by de Vinuesa et al, 32% of 102 CKD patients had ABI < 0.9, but only 26% of them were diabetic [20]. Therefore, the ABI values in CKD patients with diabetes may, in fact, be different from other patient populations, but this assumption needs further observation and consideration.

There are several limitations in our study. Our study included patients at only one hospital and the selection of patients might be biased; thus, our conclusion should not be extrapolated. In addition, this was a cross-sectional study and we were unable to predict the likelihood of PAOD or arterial stiffness. Further studies are needed to measure the association between baseline renal insufficiency with the progression of peripheral artery disease and future peripheral artery disease events. Finally, we did not measure parameters associated with atherosclerosis, such as C-reactive protein levels.

In conclusion, our study revealed the determinants of peripheral arterial stiffness in patients with CKD in Taiwan. Besides the well-known traditional atherosclerotic risk factors, a decreased eGFR was strongly correlated with increased arterial stiffness in such patients.

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探討南台灣慢性腎臟病人周邊動脈硬化的相關危險因子

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在慢性腎臟病病人報告指出有高盛行率的周邊動脈阻塞疾病及動脈硬化增加的情形。但在高慢性腎臟病盛行率的台灣卻很少探討。此篇文章即探討南台灣慢性腎臟病病人周邊動脈疾病及動脈硬化的相關危險因子。我們收集了一間區域醫院 **169** 位慢性腎臟病第 **3** 期到第 **5** 期病患。我們以 **ABI-form** (**Colin VP 1000**) 的儀器來為病人測量踝臂血壓比及臂踝脈搏傳遞速度。經過多變量分析，踝臂血壓比 **< 0.9** 與糖尿病 (***p* = 0.014**) 呈有意義正相關，而與腎絲球過濾率 (***p* = 0.049**) 呈有意義負相關。另外，臂踝脈搏傳遞速度的增加則與年紀較大 (***p* < 0.001**)，糖尿病 (***p* = 0.001**)，較高的收縮壓 (***p* < 0.001**)，脈壓降低 (***p* < 0.001**) 及低的腎絲球過濾率 (***p* = 0.030**) 呈有意義相關。本篇文章提供在南台灣慢性腎臟病病人周邊動脈阻塞疾病及動脈硬化的相關危險因子。在這群病人，除傳統的動脈粥狀硬化的危險因子以外，腎絲球過濾率的下降亦與周邊動脈阻塞疾病及動脈硬化有相關性。

關鍵詞：踝臂血壓比，動脈硬化，臂踝脈波傳遞速度，慢性腎臟病，周邊動脈阻塞疾病
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