Associated Risk Factors for Abnormal Ankle-brachial Index in Hemodialysis Patients in a Hospital

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Ankle-brachial index (ABI) is a marker for peripheral artery disease and can predict mortality in hemodialysis patients. However, it is seldom studied in southern Taiwan, an area with high prevalence of end-stage renal disease (ESRD). The aim of this study was to investigate the prevalence and associated risk factors for peripheral artery disease in the ESRD population in a hospital. All routine hemodialysis patients in one regional hospital were included except for six patients who refused ABI examinations and four patients with atrial fibrillation. Finally, 225 patients formed our study group. ABI was measured using an ABI-form device (Colin VP1000). The prevalence of ABI < 0.9 and ≥ 1.3 was 15.6% and 5.8%, respectively. ABI < 0.9 was independently associated with advanced age ($p = 0.027$), increased pulse pressure ($p = 0.005$), increased hematocrit ($p = 0.008$) and decreased serum albumin level ($p = 0.009$). In addition, ABI ≥ 1.3 was significantly associated with diabetes mellitus ($p = 0.019$). This study demonstrated the associated risk factors of peripheral artery disease in patients with hemodialysis in a hospital. ESRD patients of advanced age and with increased pulse pressure, increased hematocrit and decreased serum albumin level had a relatively high risk for ABI < 0.9 and patients with diabetes had a relatively high risk for ABI ≥ 1.3.

Key Words: ankle-brachial index, hemodialysis, peripheral artery disease
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Cardiovascular disease is the leading cause of morbidity and mortality in patients with hemodialysis, presumably due to accumulation of risk factors for atherosclerosis [1–3]. Identification of patients at high risk for cardiovascular disease and requiring aggressive preventive and interventional strategies is an initial and essential step in managing patients with hemodialysis.

The ankle-brachial index (ABI) was reported to be a good marker for atherosclerosis and useful in the diagnosis of peripheral artery disease. An ABI < 0.9 has been used to identify peripheral artery occlusive disease (PAOD) in clinical practice and epidemiologic studies [4–6]. As previously reported, high prevalence of PAOD was found using ABI in hemodialysis patients [7–9]. In addition, ABI ≥ 1.3 is considered to indicate medial artery calcification [10]. Patients with abnormally high ABI also had poor prognosis for all-cause and cardiovascular mortality in hemodialysis

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patients [9]. However, little is known about the prevalence and associated risk factors of peripheral artery disease in an end-stage renal disease (ESRD) population in southern Taiwan [11]. The aim of this study was to evaluate the prevalence and associated risk factors for peripheral artery disease in ESRD patients undergoing hemodialysis in southern Taiwan.

Methods

Study patients and design

The study was conducted in one regional hospital in southern Taiwan. All routine hemodialysis patients in this hospital were included except for six patients who refused ABI examinations and four patients with atrial fibrillation. Finally, 225 patients (98 males, 127 females) formed our hemodialysis group. The protocol was approved by our Institutional Review Board and all enrolled patients gave written informed consent.

Hemodialysis

All hemodialysis patients underwent their routine hemodialysis three times a week using a Toray 321 machine (Toray Medical Company, Tokyo, Japan). Each session of hemodialysis lasted for 3–4 hours and was performed using a dialyzer with a blood flow rate of 250–300 mL/minute and dialysate flow rate of 500 mL/minute.

ABI measurement

Measurements of ABI were made using an ABI-form device (VP1000; Colin Co. Ltd., Komaki, Japan), which automatically and simultaneously measures blood pressure (BP) in both arms and ankles using an oscillometric method [12–14]. An ABI < 0.9 is 95% sensitive and 100% specific for angiographically documented PAOD in identifying healthy individuals [15]. ABI measurements were made 10–30 minutes before hemodialysis. Occlusion and monitoring cuffs were placed tightly around the upper arm without blood access and on both sides of the lower extremities in the supine position. ABI was calculated as the ratio of ankle systolic BP divided by arm systolic BP, with only the lower value of the ankle systolic BP used for calculation. ABI measurements were done once in each patient. An ABI ≥ 0.9 and < 1.3 was considered normal, and patients were diagnosed as having peripheral artery disease if ABI was < 0.9 or ≥ 1.3.

Collection of demographic, medical, and laboratory data

Demographic and medical data including age, gender, smoking history and comorbid conditions were obtained from medical records and interviews with patients. Body mass index was calculated as the ratio of weight in kilograms divided by the square of height in meters. Comorbid conditions were defined as follows. Study subjects were defined as having diabetes mellitus (DM) if the claimed data had ICD-9 code 250.00 to 250.90, or the fasting blood glucose level was greater than 126 mg/dL, or hypoglycemic agents were used to control blood glucose levels. A similar definition was applied to hypertension with an ICD-9 code of 401.9, diagnosed by a physician, a systolic BP ≥ 140 mmHg or diastolic BP ≥ 90 mmHg, or 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, old myocardial infarction, or having undergone coronary bypass surgery or angioplasty.

Laboratory data were obtained from fasting blood samples using an autoanalyzer (COBAS Integra 400; Roche Diagnostics GmbH, Mannheim, Germany). High-sensitivity C-reactive protein was measured by commercially available kits (Dade Behring Marburg GmbH, Marburg, Germany). Serum intact parathyroid hormone (PTH) concentration was evaluated using a commercially available two-sided immunoradiometric assay (CIS Bio International, Gif Sur Yvette, France). Blood samples were obtained within 1 month of enrolment. Kt/V was evaluated monthly as a marker of dialysis efficiency, and was determined according to the procedure of Gotch [16].

Statistical analysis

Statistical analysis was performed using SPSS version 12.0 (SPSS Inc., Chicago, IL, USA) for Windows. Data are expressed as percentage or mean ± standard deviation. Differences between groups were determined by the χ^2 test for categorical variables or the independent *t* test for continuous variables. Logistic regression was used to identify the risk factors associated with ABI < 0.9 or ≥ 1.3. Significant variables in the univariate analysis were further analyzed by multivariate analysis. A significant difference was considered when the *p* value was less than 0.05.

RESULTS

The mean age of the 225 patients was 58.7 ± 13.0 years. The prevalence of ABI < 0.9 and ≥ 1.3 was 15.6% and 5.8%, respectively. The differences between patients with normal and pathologic ABI are shown in Table 1. Compared with patients with normal ABI, patients with ABI < 0.9 were found to be of an older age ($p < 0.001$), have higher prevalence of DM ($p < 0.001$), coronary artery disease ($p = 0.008$) and cerebrovascular disease ($p = 0.008$), lower diastolic BP ($p = 0.049$), higher pulse pressure ($p < 0.001$), lower serum albumin ($p = 0.001$), higher fasting glucose ($p = 0.043$), lower high-density lipoprotein cholesterol ($p < 0.001$), and higher hematocrit levels ($p = 0.026$). In addition, patients with ABI ≥ 1.3 had a higher prevalence of DM ($p = 0.014$) than those with normal ABI.

Table 2 shows the determinants of ABI < 0.9 among the study patients. In the univariate regression analysis, ABI < 0.9 was found to be significantly associated with advanced age ($p < 0.001$), the presence of DM ($p = 0.001$), a history of coronary artery disease ($p = 0.007$) and cerebrovascular disease ($p = 0.012$), increased pulse pressure ($p = 0.030$), decreased serum albumin level ($p = 0.002$), increased fasting glucose ($p = 0.030$), decreased high-density lipoprotein cholesterol ($p = 0.011$), and increased hematocrit level ($p = 0.019$). After multiple logistic regression analysis, ABI < 0.9 was positively associated with age ($p = 0.027$), pulse pressure ($p = 0.005$), and serum hematocrit level ($p = 0.008$), but negatively associated with serum albumin level ($p = 0.009$).

We also analyzed the determinants of ABI ≥ 1.3 in study patients, and found that DM (hazard ratio, 4.733;

Table 1. Comparison of patients with a normal ABI (between 0.9 and 1.3) and a pathologic one

	ABI ≥ 0.9 to 1.3 ($n = 177$)	ABI < 0.9 ($n = 35$)	ABI ≥ 1.3 ($n = 13$)	All patients ($n = 225$)
ABI	1.12 ± 0.10	0.72 ± 0.14*	1.38 ± 0.09*	1.08 ± 0.19
Age (yr)	57.4 ± 12.8	66.9 ± 9.7*	54.6 ± 14.6	58.7 ± 13.0
Male gender (%)	45.8	31.4	46.2	43.6
Duration of dialysis (mo)	55.6 ± 47.1	62.2 ± 51.7	51.0 ± 36.5	56.4 ± 47.2
Smoking history (%)	28.3	22.9	23.1	27.1
Diabetes mellitus (%)	33.3	68.6*	69.2†	40.9
Hypertension (%)	67.8	77.1	84.6	70.2
Coronary artery disease (%)	26.0	48.6†	38.5	30.7
Cerebrovascular disease (%)	7.9	22.9†	0	9.8
Systolic BP (mmHg)	143.3 ± 24.2	151.8 ± 34.4	139.2 ± 22.1	144.3 ± 25.9
Diastolic BP (mmHg)	79.5 ± 14.9	73.7 ± 18.3†	76.2 ± 20.1	78.5 ± 15.8
Pulse pressure (mmHg)	63.8 ± 16.3	78.1 ± 19.2*	63.0 ± 15.8	65.8 ± 17.4
Body mass index (kg/m^2)	23.9 ± 3.4	24.0 ± 4.3	22.7 ± 3.4	23.8 ± 3.5
Laboratory parameters				
Albumin (g/dL)	3.8 ± 0.3	3.7 ± 0.3†	3.8 ± 0.3	3.8 ± 0.3
Fasting glucose (mg/dL)	115.9 ± 52.6	140.2 ± 63.5†	124.9 ± 46.2	120.2 ± 54.5
Triglyceride (mg/dL)	167.3 ± 126.7	161.7 ± 91.6	185.3 ± 174.0	167.5 ± 124.6
Cholesterol (mg/dL)	184.4 ± 41.9	177.3 ± 44.4	170.5 ± 42.5	182.5 ± 42.3
HDL cholesterol (mg/dL)	47.4 ± 15.1	39.9 ± 9.3*	43.8 ± 14.1	46.1 ± 14.5
LDL cholesterol (mg/dL)	88.5 ± 27.6	89.5 ± 23.8	74.0 ± 19.7	87.8 ± 26.8
Hematocrit (%)	30.6 ± 3.1	32.0 ± 3.8†	29.0 ± 3.8	30.7 ± 3.3
Calcium (mg/dL)	9.8 ± 0.8	9.8 ± 0.8	10.0 ± 0.7	9.8 ± 0.8
Phosphate (mg/dL)	4.8 ± 1.2	4.8 ± 1.2	5.2 ± 1.0	4.8 ± 1.2
Uric acid (mg/dL)	7.6 ± 1.5	7.7 ± 1.8	8.3 ± 1.5	7.6 ± 1.5
PTH (pg/mL)	514.2 ± 470.6	450.2 ± 321.8	754.9 ± 523.3	516.4 ± 455.5
C-reactive protein (mg/L)	0.7 ± 1.3	0.9 ± 1.0	0.8 ± 1.3	0.73 ± 1.23
Kt/V	1.3 ± 0.2	1.3 ± 0.3	1.3 ± 0.3	1.3 ± 0.2

*$p < 0.001$ and †$p < 0.05$ compared with ABI ≥ 0.9–1.3. ABI = ankle-brachial index; BP = blood pressure; HDL = high-density lipoprotein; LDL = low-density lipoprotein; PTH = parathyroid hormone.

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

Parameter	Univariate		Multivariate	
	OR (95% CI)	*p*	OR (95% CI)	*p*
Age (per 1 year)	1.073 (1.036–1.112)	< 0.001	1.055 (1.006–1.106)	0.027
Male *vs.* female	0.543 (0.252–1.170)	0.119	–	–
Smoking (ever *vs.* never)	0.766 (0.327–1.792)	0.539	–	–
Diabetes mellitus	3.914 (1.807–8.478)	0.001	1.878 (0.618–5.701)	0.266
Hypertension	1.520 (0.652–3.545)	0.332	–	–
Coronary artery disease	2.574 (1.233–5.376)	0.007	1.649 (0.615–4.416)	0.320
Cerebrovascular disease	3.725 (1.428–9.713)	0.012	2.071 (0.567–7/571)	0.271
Duration of dialysis (per 1 month)	1.003 (0.996–1.010)	0.427	–	–
Systolic BP (per 1 mmHg)	1.013 (0.999–1.029)	0.078	–	–
Diastolic BP (per 1 mmHg)	0.978 (0.956–1.001)	0.064	–	–
Pulse pressure (per 1 mmHg)	1.022 (1.002–1.041)	0.030	1.039 (1.012–1.068)	0.005
Body mass index > 27 kg/m^2	1.362 (0.507–3.662)	0.540	–	–
Laboratory parameters				
Albumin (per 1 g/dL)	0.147 (0.045–0.481)	0.002	0.128 (0.028–0.598)	0.009
Fasting glucose (mg/dL)	1.006 (1.001–1.012)	0.030	0.998 (0.989–1.006)	0.576
Triglyceride (per 1 mg/dL)	1.000 (0.996–1.003)	0.763	–	–
Cholesterol (per 1 mg/dL)	0.996 (0.987–1.005)	0.428	–	–
HDL cholesterol (per 1 mg/dL)	0.957 (0.926–0.990)	0.011	0.964 (0.920–1.011)	0.129
LDL cholesterol (per 1 mg/dL)	1.003 (0.989–1.017)	0.698	–	–
Cholesterol/HDL (per 1.0)	1.139 (0.896–1.448)	0.288	–	–
LDL/HDL (per 1.0)	1.485 (0.976–2.259)	0.065	–	–
Hematocrit (per 1%)	1.132 (1.021–1.256)	0.019	1.194 (1.048–1.359)	0.008
Calcium (per 1 mg/dL)	1.016 (0.649–1.592)	0.943	–	–
Phosphate (per 1 mg/dL)	1.008 (0.749–1.357)	0.957	–	–
Calcium-phosphate product	1.003 (0.976–1.032)	0.812	–	–
Uric acid (per 1 mg/dL)	1.019 (0.806–1.288)	0.875	–	–
PTH (per 1 pg/mL)	1.000 (0.999–1.000)	0.358	–	–
C-reactive protein (per 1 mg/L)	1.116 (0.866–1.439)	0.395	–	–
Kt/V (per 1.0)	1.703 (0.386–7.515)	0.482	–	–

OR = odds ratio; CI = confidence interval; BP = blood pressure; HDL = high-density lipoprotein; LDL = low-density lipoprotein; PTH = parathyroid hormone.

$p = 0.019$) was significantly correlated with ABI ≥ 1.3 in the multivariate analysis.

Discussion

In the present study, we evaluated the prevalence and associated risk factors for peripheral artery disease in 225 hemodialysis patients in Taiwan. We found that ABI < 0.9 was positively associated with age, pulse pressure and hematocrit, but negatively associated with serum albumin level. In addition, the presence of DM was independently associated with ABI ≥ 1.3.

Recently, PAOD has been reported to be prevalent in hemodialysis patients and influences their mortality [17,18]. In our study, the prevalence of PAOD, defined as ABI < 0.9, was 15.6%, which was close to that (16.5%) obtained from a large-scale epidemiologic survey conducted by Ono et al [9]. Previous studies have identified several risk factors for PAOD in hemodialysis patients, including old age, DM, a history of cardiovascular disease and cerebrovascular disease, low albumin level and high pulse pressure [9,19–22]. In the current study, we confirmed most of these factors, including advanced age, increased pulse pressure and lower albumin level. An interesting finding was the strong correlation between ABI < 0.9 and increased

hematocrit. It is known that certain hemorheologic factors such as hematocrit, as well as blood and plasma viscosity, are associated with clinical manifestations of atherosclerosis by promoting endothelial damage and diffuse intimal thickening during the atherosclerotic process [23–25]. Another reasonable explanation is that a high hematocrit level might be a consequence of using high doses of erythropoietin. In an animal model, continuous administration of erythropoietin in mice resulted in an increased size of the atherosclerotic lesion [25]. Thus our results show an association between elevated hematocrit and ABI < 0.9.

It is well recognized that diabetes is a risk factor of peripheral artery disease [26,27]. In the present study, although DM was a strong predictor for PAOD in univariate analysis, the statistical impact was no longer significant in multivariate analysis. The impact of DM might be confounded by malnutrition, such as low levels of serum albumin. The multivariate analysis demonstrated that PAOD was independently associated with low serum albumin level in our study. A low serum albumin level has been regarded not only as indicative of a malnutrition status but also the presence of chronic inflammation [28]. Chronic inflammation is recognized as a risk factor for atherosclerosis [29,30].

Secondary hyperparathyroidism and the effect of calcium, phosphorous and the calcium-phosphorous product has been reported to contribute to endothelial dysfunction and vascular calcification in patients with chronic renal failure [31,32]. In our study, these parameters did not seem to be associated with PAOD. A reasonable explanation is that vascular calcification owing to secondary hyperparathyroidism and disordered calcium and phosphate homeostasis might result in false elevation of ABI values [33]. Thus, our result showed no correlation between serum PTH level and PAOD.

Dyslipidemia is a well-established atherogenic factor in hemodialysis patients [34,35]. Matsumae et al evaluated the determinants of PAOD and arterial stiffness in 143 nondiabetic hemodialysis patients and found that low-density lipoprotein cholesterol was negatively associated with ABI and aortic pulse wave velocity [8]. However, some studies reported the absence of an association between serum lipid levels and PAOD [21,22]. In our study, there was no association between serum lipid levels and ABI < 0.9, which was consistent with Cheung et al [21] and O'Hare et al [22].

So far, whether the dialysis process or ESRD itself promotes atherosclerosis progression remains controversial. Several studies have demonstrated that the duration of dialysis correlates well with peripheral artery disease [8,22,36]. They explained their finding by vascular calcification caused by alterations in the metabolism of calcium, phosphate and PTH, inflammatory alterations, oxidative stress or hyperhomocysteinemia [3,37]. However, some studies also showed contradicting results [21,38]. Our study showed no significant relationship between the duration of hemodialysis and peripheral artery disease. Further prospective studies are needed to determine whether the duration of hemodialysis contributes to atherosclerosis.

Falsely elevated pressure or incompressible arteries at the ankle level are common among patients with extensive vascular calcification of the lower extremities, which may occur in patients with diabetes or on hemodialysis [9,10]. The prevalence of PAOD may be underestimated when the criteria of ABI < 0.9 was used because many of our patients had DM (40.9%). In our study, 13 patients (5.8%) had ABI ≥ 1.3. The abnormally high ABI value or incompressible arteries had been interpreted as the presence of medial artery calcification. Hemodialysis patients with abnormally high ABI have poor prognosis for all-cause and cardiovascular mortality [9].

There are several limitations to our study. The study subjects were enrolled from only one regional hospital and the selection of patients was rather restricted. Therefore, generality of the results is limited. In addition, because the design of the study is observational, it is susceptible to selection bias. We minimized this bias by enrolling all hemodialysis patients in our dialysis clinics and statistically adjusting for several variables that may influence peripheral artery disease. Finally, this was a cross-sectional study and thus we cannot evaluate the predictors of peripheral artery disease. A prospective trial is needed to determine the predictors for the progression of peripheral artery disease.

In conclusion, this study demonstrated the associated risk factors of peripheral artery disease among hemodialysis patients in southern Taiwan. ESRD patients of advanced age and with increased pulse pressure, increased hematocrit and decreased serum albumin level had a relatively high risk for PAOD and patients with DM had a relatively high risk for ABI ≥ 1.3.

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探討一醫院血液透析病人不正常踝臂血壓比的相關危險因子

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踝臂血壓比可做為周邊動脈疾病的指標，且可用來預測血液透析病人的死亡率。但在高末期腎臟病盛行率的南台灣卻鮮少探討。此篇文章即探討一醫院血液透析病人周邊動脈疾病的盛行率及相關危險因子。除了 6 個病人拒絕及 4 個心房顫動的病人，我們收集了一間區域醫院所有常規血液透析病人，共 225 個病人。我們以 **ABI-form** (**Colin VP 1000**) 的儀器來為病人測量踝臂血壓比。踝臂血壓比 < 0.9 及 ≥ 1.3 的盛行率分別為 15.6% 及 5.8%。年紀較大 ($p = 0.027$)，脈壓增大 ($p = 0.005$)、血比容增加 ($p = 0.008$) 及白蛋白降低 ($p = 0.009$) 與踝臂血壓比 < 0.9 呈有意義相關。另外，糖尿病則與踝臂血壓比 ≥ 1.3 有相關 ($p = 0.019$)。本篇文章提供在一醫院血液透析病人周邊動脈疾病的相關危險因子。其年紀較大，脈壓較高，血比容較高及白蛋白降低為踝臂血壓比 < 0.9 的危險因子，而糖尿病則為踝臂血壓比 ≥ 1.3 的危險因子。

關鍵詞：踝臂血壓比，血液透析，周邊動脈疾病
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