

# Prevalence and Risk Factors of Peripheral Arterial Disease Among Thai Dialysis Patients

Atiporn Ingsathit MD, PhD\*\*\*, Voravech Nissaisorakarn MD\*\*,  
Pongpan Thanak MD\*\*, Chagriya Kittiyakara MD\*\*,  
Vasant Sumethkul MD\*\*, Surasak Kantachavesiri MD, PhD\*\*, Piyanut Pootracool MD\*\*\*

\* Section for Clinical Epidemiology and Biostatistics, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Thailand

\*\* Division of Nephrology, Department of Medicine, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Thailand

\*\*\* Division of Vascular Surgery, Department of Surgery, Faculty of Medicine Ramathibodi Hospital, Mahidol University, Thailand

---

**Objective:** To identify the prevalence and risk factors of peripheral arterial disease (PAD) in dialysis patients covering both hemodialysis and peritoneal dialysis.

**Material and Method:** All consecutive cases of stable dialysis patients in Ramathibodi hospital from September 2013 to December 2013 were surveyed. Patients were classified as having PAD if they had ankle-brachial blood pressure index (ABI) values of  $\leq 0.9$  or  $> 1.4$ . We also measured toe-brachial blood pressure index (TBI) and TBI  $\leq 0.6$  was classified as abnormal TBI. Data were analyzed to identify the prevalence and risk factors of PAD.

**Results:** Among these 269 stable dialysis patients, the mean age was  $48.8 \pm 15.1$  years and 56.9% were male. The mean dialysis vintage was  $52.6 \pm 41.8$  months. The prevalence of PAD was 11.5% and the prevalence of abnormal TBI was 29.7%. Multivariate regression analysis found that increased body mass index (BMI), history of coronary artery disease (CAD), and increased pulse pressure were associated with PAD.

**Conclusion:** The prevalence of PAD among long-term stable dialysis patients in Thailand was around one-tenth. The prevalence of abnormal TBI was higher than those of abnormal ABI criteria. Factors associated with PAD were increased BMI, history of CAD, and increased pulse pressure.

**Keywords:** ABI, dialysis, Peripheral arterial disease, prevalence, TBI

*J Med Assoc Thai* 2017; 100 (2): 133-141

Full text. e-Journal : <http://www.jmatonline.com>

---

Cardiovascular disease is the major cause of death for end-stage renal disease (ESRD) patients<sup>(1)</sup>. The risk of the cardiovascular disease could be increased by 30 fold in dialysis populations, compared with normal populations in the same age group<sup>(2-4)</sup>. Atherosclerotic cardiovascular disease is a systemic process involving coronary, cerebrovascular, visceral, and extremity vasculature. Peripheral arterial disease (PAD) is an atherosclerotic cardiovascular disease associated with an increased risk of cardiovascular

and cerebrovascular events, including myocardial infarction, stroke, and death. With the prevalence of approximately 27 million people in North America and Europe, PAD is a critical public health issue<sup>(5)</sup>. Approximately 30% of patients with cardiovascular disease may have PAD as the only clinical manifestation<sup>(6)</sup>. Therefore, it is important for early detection of PAD not only as a marker of generalized cardiovascular disease but also a predictor of higher morbidity and mortality<sup>(7-10)</sup>.

Many non-invasive procedures have been developed in order to detect PAD such as ankle-brachial blood pressure index (ABI), toe-brachial blood pressure index (TBI), cardio-ankle vascular index (CAVI), pulse wave velocity (PWV), and skin perfusion pressure (SPP). Among these non-invasive procedures, ABI

---

**Correspondence to:**

Ingsathit A, Section for Clinical Epidemiology and Biostatistics, Faculty of Medicine Ramathibodi Hospital, 270 Rama VI Road, Ratchathevi, Bangkok 10400, Thailand.

Phone: +66-2-2011284

E-mail: [atiporn.ing@mahidol.ac.th](mailto:atiporn.ing@mahidol.ac.th)

and TBI are recommended for screening and diagnosis of PAD<sup>(11, 12)</sup>.

ABI is a non-invasive, simple, inexpensive, and reliable procedure to access the patency of lower extremity arterial circulation<sup>(13)</sup>. The sensitivity of ABI ( $\leq 0.9$ ) for PAD detection is 95% with almost 100% specificity<sup>(7,14-16)</sup>. ABI is commonly used as a diagnostic test for PAD<sup>(9)</sup>. Many studies have reported the prevalence of PAD using ABI in normal and renal insufficient population<sup>(7,9,14,17)</sup>, but studies still remain limited for the dialysis population.

It is known that the prevalence of PAD in a renal insufficient population is higher than a normal population<sup>(17,18)</sup> and even higher in a dialysis population<sup>(19)</sup>. Furthermore, among dialysis patients, PAD is more common in hemodialysis (HD) patients than peritoneal dialysis (PD) patients<sup>(20)</sup>. Most studies of dialysis population were taken in HD patients with a small number taken in PD patients and even less number of studies taken in both HD and PD patients.

In addition to low ABI, high ABI (which represents vascular calcification or non-compressible arteries) is also a good tool for detection and prediction of morbidity and mortality of cardiovascular disease<sup>(21-23)</sup>. Vascular calcification is very common among dialysis populations and might contribute to the development of severe peripheral arterial disease<sup>(24)</sup>.

We performed a cross-sectional study to determine the prevalence of PAD in dialysis patients covering both HD and PD. Our secondary objective was to identify the risk factors associated with PAD among this population.

## Material and Method

### Study population

The study population was recruited from the stable dialysis patients in Ramathibodi Hospital, Bangkok, Thailand. Inclusion criteria were regularly dialysis patients who had follow up at Ramathibodi Hospital for at least 3 months and agreed to sign the informed-consent form. The exclusion criteria were previous evidence of PAD, and bilateral lower limb amputation.

### Data collection and measurements

Baseline characteristics including, age, smoking history, dialysis vintage, residual renal function (de-

finied by urine production  $>100$  ml/day), underlying diseases, and medical prescriptions were collected by direct interview and through medical records.

Clinical symptoms including leg pain, numbness, cramp, and vascular access problems were evaluated by direct personal interview. Physical examinations including chronic ulcer, blood pressure, and pulse were examined by clinicians.

Laboratory findings including: complete blood count, serum calcium, serum phosphate, fasting blood glucose (FBS), serum albumin, blood urea nitrogen, serum creatinine, lipid profile, uric acid level, and parathyroid hormone level were collected from the most recent laboratory reports at the screening time.

### Assessment of ABI & TBI

ABI and TBI were measured in all participants by a VaSera VS1500 (Fukuda Denshi, Tokyo, Japan). The measurements were performed by a well-trained technician after the patients had rested in supine position for at least 10 minutes. Cuffs were applied to both arms (if no vascular access), both legs (if possible), and both big toes (or the next toe, if amputated). The measurements were automatically calculated and reported by the machine. ABI was calculated by dividing the lower value of the ankle systolic blood pressure (SBP) by the higher value of the brachial SBP<sup>(6,23,25-28)</sup>.

Abnormal ABI was defined by  $ABI \leq 0.9$ <sup>(11, 29)</sup> or  $ABI > 1.4$ <sup>(12)</sup> and abnormal TBI was defined by  $TBI \leq 0.6$ <sup>(12,29)</sup>. If there was any abnormal ABI of each patient, the patient would be counted in the abnormal ABI group. The lower ABI and TBI values were used in the analysis.

### Statistical analysis

Sample size estimating was calculated by using the prevalence of PAD from Lee's study<sup>(20)</sup> which was 18.2% and estimated the confidence interval width of 5%, and the type one error was set of 5%. The estimated sample size was at least 229 subjects. The categorical data were expressed as frequencies and continuous data were expressed as mean values and standard deviations (SD). Fisher exact test and Chi-square test were used to compare between categorical data, while Mann-Whitney test and student's t-test were used to compare between continuous data. Association of abnormal ABI and associated factors was analyzed by logistic regression in univariate analysis. After con-

**Table 1.** Clinical and demographic characteristics of population

Characteristics	Overall	Characteristics	Overall
Demographics:		Co-morbidities:	
Age (years), mean±SD	48.8±15.1	Diabetes mellitus, n (%)	57 (21.3)
Male, n (%)	153 (56.9)	Hypertension, n (%)	223 (82.9)
BMI (kg/m <sup>2</sup> ), mean±SD	162.3±8.3	Dyslipidemia, n (%)	98 (36.6)
Smoking, n (%)		Coronary artery disease, n (%)	38 (14.2)
- Non	177 (67.1)	Cerebrovascular disease, n (%)	21 (7.8)
- Ex-smoker	79 (29.9)		
- Smoker	8 (3.0)	Laboratories:	
Dialysis type, n (%)		Hct (%), mean±SD	33.8±5.4
- Hemodialysis	212 (78.8)	Hb (g/dL), mean±SD	11.0±1.8
- Peritoneal dialysis	57 (21.2)	Alb (g/L), mean±SD	36.0±5.0
Hemodialysis Frequency, n (%)		BUN (mg/dL), mean±SD	53.8±19.4
- 2 times/week	79 (37.3)	Cr (mg/dL), mean±SD	10.4±3.7
- 3 times/week	133 (62.7)	Chol (mg/dL), mean±SD	174.5±45.3
Dialysis vintage (Month), mean±SD	52.6±41.8	LDL (mg/dL), mean±SD	88.3±36.6
Urine >100ml/day, n (%)	124 (46.1)	TG (mg/dL), mean±SD	131.1±121.5
SBP (mmHg), mean±SD	155.8±23.7	FBS (mg/dL), mean±SD	104.8±38.8
DBP (mmHg), mean±SD	93.7±14.8	Uric acid (mg/dL), mean±SD	6.6±2.0
Pulse pressure, mean±SD	62.1±18.2	Ca (mg/dL), mean±SD	9.4±1.0
Vitamin D usage, n (%)	110 (40.9)	Phosphate (mg/dL), mean±SD	5.0±1.7
Antiplatelet usage, n (%)	57 (21.2)	PTH (pg/dL), mean±SD	422.7±478.5
Statin usage, n (%)	126 (46.8)		

**Table 2.** Prevalence of abnormal vascular parameters according to ABI and TBI for dialysis patients

Methods	Overall (n = 269)	Peritoneal dialysis (n = 57)	Hemodialysis (n = 212)
ABI≤0.9 or ABI > 1.4	31 (11.5%)	5 (8.8%)	26 (12.3%)
TBI≤0.6	80 (29.7%)	14 (24.6%)	66 (31.1%)

founding factors were controlled, multivariate logistic regression analysis was performed to identify the final model. The results were presented as odd ratios (OR), 95% confidence intervals (95% CI), and *p*-values (P). Results were considered statistically significant if *P* < 0.05 for both univariate and multivariate analysis. All analyzes were performed using STATA version 13 (StataCorp. College Station, TX, USA).

## Results

### Baseline characteristics

From a total 281 participants, 12 patients were excluded due to history of existed PAD. Finally, 269 patients were included for statistical analysis.

Baseline characteristics of the present study population at the enrolled time were showed in Table 1. More than half of the patients in the present study

were men (56.9%) with mean age of 48.8±15.1 years. Their average BMI was 22.4±4.3 kg/m<sup>2</sup>. Most of the patients were non-smokers (67.1%) with less ex-smokers (29.9%) and minimal smokers (3.0%). The most common mode of dialysis was hemodialysis (78.8%). The mean dialysis vintage was 52.6±41.8 months with the range of 3 to 252 months. Many patients in the study had concomitant diseases including hypertension (HTN) (82.9%), whereas some patients had dyslipidemia (DLP) (36.6%), diabetes mellitus (DM) (21.3%), coronary artery disease (CAD) (14.2%), and cerebrovascular attack (CVA) (7.8%). Symptoms of PAD including claudication, chronic wound, numbness, cramp, and vascular access problem presented in 54 (20.8%), 10 (3.9%), 69 (26.5%), 79 (30.4%), and 26 (10.0%) patients, respectively.

Other baseline characteristics and laboratory findings have been shown in Table 1.

### Prevalence of abnormal vascular parameters

ABI and TBI were measured in all participants. The mean ABI of the population was 1.1±0.1. Abnormal ABI was found in 31 patients. Thus, the estimated prevalence of abnormal ABI as described previously for the present population was 11.5%. In addition, 5 (8.8%) PD patients had abnormal ABI, whereas, 26 (12.3%) HD patients had abnormal ABI. The mean TBI was 0.8±0.2 and abnormal TBI was found in 80 (29.7%) patients. In which, 14 (24.6%) PD patients had abnormal TBI, whereas, 66 (31.1%) HD patients had abnormal TBI, as shown in Table 2.

Using ABI of each patient in the study, we divided all patients into two groups. They were "PAD" group (ABI≤0.9 or ABI>1.4) and "Non-PAD" group (0.9<ABI≤1.4).

The mean age of patients in PAD group (59.3±18.6 years) was older than the Non-PAD group (47.4±14.1 years) ( $p<0.001$ ). The BMIs of the PAD group (24.2±6.1 kg/m<sup>2</sup>) were higher than the Non-PAD group (22.2±3.9 kg/m<sup>2</sup>) ( $p = 0.015$ ). Patients in the PAD group had higher rate of having concomitant diseases which include; DLP (51.6%), DM (48.4%), CVA (22.6%), and CAD (35.5%) ( $p<0.05$ ). There were nearly the same proportions of hypertensive patients in PAD and Non-PAD groups (80.7% and 83.5%, respectively), but the mean SBP of the PAD group (167.6±25.4 mmHg) was higher than the mean SBP of the Non-PAD group (154.2±23.1 mmHg) ( $p = 0.003$ ).

Nevertheless, the mean diastolic blood pressure (DBP) of the PAD group (86.9±16.2 mmHg) was lower than the mean DBP of the Non-PAD group (94.6±14.4 mmHg) ( $p = 0.006$ ), significantly. The mean pulse pressure of the PAD group (80.7±21.2 mmHg) was higher than the Non-PAD group (59.6±16.3 mmHg) ( $p<0.001$ ).

The proportion of anti-platelet agents usage was higher in the PAD group (41.9%) compared to the Non-PAD group (18.5%) ( $p = 0.003$ ) whereas the usage of vitamin D and statin were not significantly different.

Moreover, patients in the PAD group had less mean serum albumin and mean PTH levels than the Non-PAD group, whereas the mean FBS level was higher in the PAD group than the Non-PAD. Other baseline characteristics and laboratory findings of PAD and Non-PAD groups were showed in Table 3.

Univariate logistic regression analysis between abnormal ABI and associated factors was showed in Table 4. Univariate logistic regression showed that abnormal ABI was significantly correlated with age, BMI, history of DM, CAD, CVA, pulse pressure, serum albumin, FBS, and anti-platelet usage ( $p<0.05$ ).

From multivariate logistic regression analysis, only BMI, history of CAD, and pulse pressure were statistically significant associate with abnormal ABI (Table 4). This suggested that the risk of PAD was 1.13 times higher (adjusted OR 1.13, 95% CI: 1.03-1.23) among ESRD patients for each unit increase in BMI (kg/m<sup>2</sup>). Patients with history of CAD had approximately 3 times higher risk of PAD than those without CAD history. In addition, patients with 1 mmHg higher in pulse pressure had higher risk of PAD about 6% (adjusted OR 1.06, 95% CI: 1.04-1.09).

### Discussion

In the present study, we found that the prevalence of PAD (defined as ABI≤0.9 or ABI>1.4) among the participants was 11.5%. The prevalence of PAD of the HD group was higher than the PD group (12.3% and 8.8%, respectively). By using TBI<0.6 as a cut point, the prevalence of PAD among all participants, HD group, and PD group were increased to 29.7%, 31.1%, and 24.6%, respectively. Consistent with ABI, the prevalence of PAD of the HD group was still higher than the PD group. We found three factors that were significantly associated with PAD including increased BMI, history of CAD, and increased pulse pressure. We

**Table 3.** Clinical and demographic characteristics of PAD and Non-PAD patients

Characteristics	PAD n = 31	Non-PAD n = 238	<i>p</i> -value*
Demographics:			
Age (years), mean±SD	59.3±18.6	47.4±14.1	<0.001
Male, n (%)	20 (64.5)	133 (55.9)	0.361
BMI (kg/m <sup>2</sup> ), mean±SD	24.2±6.1	22.2±3.9	0.015
Smoking, n (%)			
- Non	19 (61.3)	158 (67.8)	0.342
- Ex-smoker	12 (38.7)	67 (28.8)	
- Smoker	0 (0)	8 (3.4)	
Dialysis type, n (%)			
- Hemodialysis	26 (83.9)	186 (78.2)	0.464
- Peritoneal dialysis	5 (16.1)	52 (21.9)	
Hemodialysis Frequency, n (%)			
- 2 times/week	10 (38.5)	69 (37.1)	0.893
- 3 times/week	16 (61.5)	117 (62.9)	
Dialysis vintage (Month), mean±SD	59.8±48.9	51.8±40.8	0.419
Urine >100ml/day, n (%)	15 (48.4)	109 (45.8)	0.866
SBP (mmHg), mean±SD	167.6±25.4	154.2±23.1	0.003
DBP (mmHg), mean±SD	86.9±16.2	94.6±14.4	0.006
Pulse pressure, mean±SD	80.7±21.2	59.6±16.3	<0.001
Vitamin D usage, n (%)	17 (54.8)	93 (39.1)	0.093
Antiplatelet usage, n (%)	13 (41.9)	44 (18.5)	0.003
Statin usage, n (%)	15 (48.4)	111 (46.6)	0.826
Co-morbidities:			
Diabetes mellitus, n (%)	15 (48.4)	42 (17.7)	<0.001
Hypertension, n (%)	25 (80.7)	198 (83.5)	0.685
Dyslipidemia, n (%)	16 (51.6)	82 (34.6)	0.064
Coronary artery disease, n (%)	11 (35.5)	27 (11.4)	0.001
Cerebrovascular disease, n (%)	7 (22.6)	14 (5.9)	0.005
Laboratories:			
Hct (%), mean±SD	35.1±6.1	33.6±5.3	0.152
Hb (g/dL), mean±SD	11.5±2.0	11.0±1.8	0.122
Alb (g/L), mean±SD	33.7±6.6	36.3±4.7	0.007
BUN (mg/dL), mean±SD	50.3±18.7	54.2±19.5	0.301
Cr (mg/dL), mean±SD	9.2±4.0	10.6±3.7	0.055
Chol (mg/dL), mean±SD	169.4±34.5	175.2±46.6	0.515
LDL (mg/dL), mean±SD	90.2±46.3	88.0±34.9	0.826
TG (mg/dL), mean±SD	125.9±86.9	131.8±125.6	0.747
FBS (mg/dL), mean±SD	119.2±54.8	102.9±36.0	0.031
Uric acid (mg/dL), mean±SD	6.3±1.8	6.7±2.0	0.358
Ca (mg/dL), mean±SD	9.4±1.1	9.4±1.0	0.727
Phosphate (mg/dL), mean±SD	4.5±1.5	5.1±1.7	0.071
PTH (pg/dL), mean±SD	305.8±386.4	438.8±488.4	0.033

\* *p*-value for difference between PAD and Non-PAD groups

**Table 4.** Univariate and multivariate logistic regression analysis of factors associated with PAD

Factors	Univariate Odd ratio (95% CI)	p-value	Multivariate Odd ratio (95% CI)	p-value
Age (years), mean±SD	1.05 (1.03-1.08)*	<0.001*		
BMI (kg/m <sup>2</sup> ), mean±SD	1.10 (1.02-1.19)*	0.018*	1.13 (1.03-1.23)	0.007*
Diabetes mellitus, n (%)	4.35 (2.00-9.49)*	<0.001*		
Coronary artery disease, n (%)	4.28 (1.85-9.89)*	0.001*	2.96 (1.13-7.71)	0.027*
Cerebrovascular disease, n (%)	4.65 (1.71-12.63)*	0.003*		
Pulse pressure, mean±SD	1.06 (1.04-1.08)*	<0.001*	1.06 (1.04-1.09)	<0.001*
Antiplatelet usage, n (%)	3.18 (1.45-6.98)*	0.004*		
Alb (g/L), mean±SD	0.90 (0.84-0.97)*	0.008*		
FBS (mg/dL), mean±SD	1.01 (1.00-1.02)*	0.041*		
PTH (pg/dL), mean±SD	1.00 (1.00-1.00)	0.173		

\* Statistically significant ( $p < 0.05$ )

also found a trend of association of PAD with increasing age and DM, but there was no statistical significance. The prevalence of PAD in the present study was lower than that obtained from the previous study of Lee CC et al (18.2%)<sup>(20)</sup>, taken in Taiwan. This may be due to the exclusion criteria to exclude the patients with known PAD before the analysis was performed. Another reason is that the average age of participants in the present study was lower compared with the previous study (48.8±15.1 vs. 57.9±13.64, respectively). This may be explained by the lipid-rich plaque producing focal stenosis in aging population. However, unlike normal population, PAD in renal insufficient groups is caused by intense medial arterial calcification causing stenosis of the artery, which occurs at an earlier age and with greater severity<sup>(30-32)</sup>. Most PAD patients in this population were hemodialysis patients (83.9%), which is compatible with the previous study of Lee CC et al (94.3%)<sup>(20)</sup>. Also among the HD group 12.3% had PAD, while among the PD group 8.8% had PAD, which was similar to the previous study of Lee CC et al (29.6% and 4.8%, respectively)<sup>(20)</sup>.

A number of previous studies showed several factors associated with PAD including advanced age, DM, CAD, CVA, smoking, low DBP, low serum albumin, and increased pulse pressure<sup>(33,34)</sup>. However, in the present study, we only confirmed some of these associated factors with PAD. The reason that we cannot show all of these associations may be due to the sample size was too small or there were stronger associated factors. In the present study, we only confirmed that history of CAD and increased pulse pressure are asso-

ciated with PAD but not the others mentioned above. In addition, we found that increased BMI was also associated with PAD, which was similar to the study with 5,419 participants in the US<sup>(35)</sup>.

Our study had both strengths and weaknesses. This is the first study to determine the prevalence of PAD and its associated factor in a dialysis population in Thailand. We used ABI, the reliable noninvasive and widely used method to detect PAD. However, there were several limitations to the present study. The study's subjects were enrolled from only one center with limited number of participants, which may not have had enough power to show some associations between PAD and some risks. The study design was a cross-sectional study, thus we could not evaluate temporal relationships between associated factors and PAD. A clinical prediction score model should be further developed to aid in identifying high-risk populations for screening. A prospective trial is needed to evaluate the clinical impact and prognosis of PAD in a dialysis population.

In summary, the present study showed the prevalence of 11.5% for PAD in a dialysis population. Among this dialysis population, HD patients were more likely to suffer from PAD than PD patients. The associated risk factors for PAD were increased BMI, history of CAD, and increased pulse pressure.

#### What is already known on this topic?

There are limited previous studies on the prevalence and the risk factors of PAD in dialysis patients and none of them were done in Thai patients. The

prevalence of PAD among dialysis patients of previous studies was around 18%. A number of previous studies showed several factors associated with PAD including advanced age, DM, CAD, CVA, smoking, low DBP, low serum albumin, and increased pulse pressure

### What this study adds?

This study showed that the prevalence of PAD among dialysis patients is 11.5% and 29.7% using the ABI and TBI method, respectively. The prevalence of PAD among the HD group was higher than the PD group. We found three factors that were significantly associated with PAD namely increased BMI, history of CAD, and increased pulse pressure. Two factors were found to be associated with PAD including increasing age and DM, but there was no statistical significance.

### Acknowledgement

The present study was partly granted by Astellas pharma (Thailand) co. Ltd. The protocol was initiated by A.I. and there was no sponsor role in the design, data collection, data analysis and manuscript writing.

### Potential conflicts of interest

None.

### References

1. Bloembergen WE, Port FK, Mauger EA, Wolfe RA. Causes of death in dialysis patients: racial and gender differences. *J Am Soc Nephrol* 1994; 5: 1231-42.
2. Churchill DN, Taylor DW, Cook RJ, LaPlante P, Barre P, Cartier P, et al. Canadian hemodialysis morbidity study. *Am J Kidney Dis* 1992; 19: 214-34.
3. Lindner A, Charra B, Sherrard DJ, Scribner BH. Accelerated atherosclerosis in prolonged maintenance hemodialysis. *N Engl J Med* 1974; 290: 697-701.
4. Sarnak MJ, Levey AS, Schoolwerth AC, Coresh J, Culleton B, Hamm LL, et al. Kidney disease as a risk factor for development of cardiovascular disease: a statement from the American Heart Association Councils on Kidney in Cardiovascular Disease, High Blood Pressure Research, Clinical Cardiology, and Epidemiology and Prevention. *Circulation* 2003; 108: 2154-69.
5. Weitz JI, Byrne J, Clagett GP, Farkouh ME, Porter JM, Sackett DL, et al. Diagnosis and treatment of chronic arterial insufficiency of the lower extremities: a critical review. *Circulation* 1996; 94: 3026-49.
6. Hirsch AT, Criqui MH, Treat-Jacobson D, Regensteiner JG, Creager MA, Olin JW, et al. Peripheral arterial disease detection, awareness, and treatment in primary care. *JAMA* 2001; 286: 1317-24.
7. Cimminiello C. PAD. *Epidemiology and pathophysiology. Thromb Res* 2002; 106: V295-301.
8. Criqui MH, Langer RD, Fronek A, Feigelson HS, Klauber MR, McCann TJ, et al. Mortality over a period of 10 years in patients with peripheral arterial disease. *N Engl J Med* 1992; 326: 381-6.
9. Levy PJ. Epidemiology and pathophysiology of peripheral arterial disease. *Clin Cornerstone* 2002; 4: 1-15.
10. McDermott MM. Ankle brachial index as a predictor of outcomes in peripheral arterial disease. *J Lab Clin Med* 1999; 133: 33-40.
11. Rooke TW, Hirsch AT, Misra S, Sidawy AN, Beckman JA, Findeiss LK, et al. 2011 ACCF/AHA Focused update of the guideline for the management of patients with peripheral artery disease (updating the 2005 guideline): a report of the American College of Cardiology Foundation/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2011; 58: 2020-45.
12. Tendera M, Aboyans V, Bartelink ML, Baumgartner I, Clement D, Collet JP, et al. ESC Guidelines on the diagnosis and treatment of peripheral artery diseases: Document covering atherosclerotic disease of extracranial carotid and vertebral, mesenteric, renal, upper and lower extremity arteries: the Task Force on the Diagnosis and Treatment of Peripheral Artery Diseases of the European Society of Cardiology (ESC). *Eur Heart J* 2011; 32: 2851-906.
13. Papamichael CM, Lekakis JP, Stamatiopoulos KS, Papaioannou TG, Alevizaki MK, Cimponeriu AT, et al. Ankle-brachial index as a predictor of the extent of coronary atherosclerosis and cardiovascular events in patients with coronary artery disease. *Am J Cardiol* 2000; 86: 615-8.
14. Belch JJ, Topol EJ, Agnelli G, Bertrand M, Califf RM, Clement DL, et al. Critical issues in peripheral arterial disease detection and management: a call to action. *Arch Intern Med* 2003; 163: 884-92.

15. Feigelson HS, Criqui MH, Fronck A, Langer RD, Molgaard CA. Screening for peripheral arterial disease: the sensitivity, specificity, and predictive value of noninvasive tests in a defined population. *Am J Epidemiol* 1994; 140: 526-34.
16. Fowkes FG. The measurement of atherosclerotic peripheral arterial disease in epidemiological surveys. *Int J Epidemiol* 1988; 17: 248-54.
17. O'Hare AM, Glidden DV, Fox CS, Hsu CY. High prevalence of peripheral arterial disease in persons with renal insufficiency: results from the National Health and Nutrition Examination Survey 1999-2000. *Circulation* 2004; 109: 320-3.
18. Yevzlin AS, Gimelli G. Diagnosis and treatment of peripheral arterial disease in CKD patients. *Semin Dial* 2013; 26: 240-51.
19. O'Hare A, Johansen K. Lower-extremity peripheral arterial disease among patients with end-stage renal disease. *J Am Soc Nephrol* 2001; 12: 2838-47.
20. Lee CC, Wu CJ, Chou LH, Shen SM, Chiang SF, Jen PC, et al. Peripheral artery disease in peritoneal dialysis and hemodialysis patients: single-center retrospective study in Taiwan. *BMC Nephrol* 2012; 13: 100.
21. Adragao T, Pires A, Branco P, Castro R, Oliveira A, Nogueira C, et al. Ankle-brachial index, vascular calcifications and mortality in dialysis patients. *Nephrol Dial Transplant* 2012; 27: 318-25.
22. Amini A, Gordon I, Wilson S, Williams RA. Noncompressible arteries correlate with increased cardiovascular mortality at 2 years. *Ann Vasc Surg* 2013; 27: 918-23.
23. Ono K, Tsuchida A, Kawai H, Matsuo H, Wakamatsu R, Maezawa A, et al. Ankle-brachial blood pressure index predicts all-cause and cardiovascular mortality in hemodialysis patients. *J Am Soc Nephrol* 2003; 14: 1591-8.
24. Rostand SG, Druke TB. Parathyroid hormone, vitamin D, and cardiovascular disease in chronic renal failure. *Kidney Int* 1999; 56: 383-92.
25. Giugliano G, Sannino A, Brevetti L, Perrino C, Schiattarella GG, Franzone A, et al. Ankle/brachial index to everyone. *BMC Surg* 2012; 12 (Suppl 1): S18.
26. Chen SC, Su HM, Mai HC, Chen JH, Chen CY, Chang JM, et al. Associated risk factors for abnormal ankle-brachial index in hemodialysis patients in a hospital. *Kaohsiung J Med Sci* 2008; 24: 473-80.
27. Otani Y, Otsubo S, Kimata N, Takano M, Abe T, Okajima T, et al. Effects of the ankle-brachial blood pressure index and skin perfusion pressure on mortality in hemodialysis patients. *Intern Med* 2013; 52: 2417-21.
28. Kim ES, Wattanakit K, Gornik HL. Using the ankle-brachial index to diagnose peripheral artery disease and assess cardiovascular risk. *Cleve Clin J Med* 2012; 79: 651-61.
29. Hirakata H, Nitta K, Inaba M, Shoji T, Fujii H, Kobayashi S, et al. Japanese Society for Dialysis Therapy guidelines for management of cardiovascular diseases in patients on chronic hemodialysis. *Ther Apher Dial* 2012; 16: 387-435.
30. Block GA, Klassen PS, Lazarus JM, Ofsthun N, Lowrie EG, Chertow GM. Mineral metabolism, mortality, and morbidity in maintenance hemodialysis. *J Am Soc Nephrol* 2004; 15: 2208-18.
31. Goodman WG, Goldin J, Kuizon BD, Yoon C, Gales B, Sider D, et al. Coronary-artery calcification in young adults with end-stage renal disease who are undergoing dialysis. *N Engl J Med* 2000; 342: 1478-83.
32. Guerin AP, Pannier B, Marchais SJ, London GM. Cardiovascular disease in the dialysis population: prognostic significance of arterial disorders. *Curr Opin Nephrol Hypertens* 2006; 15: 105-10.
33. Cheung AK, Sarnak MJ, Yan G, Dwyer JT, Heyka RJ, Rocco MV, et al. Atherosclerotic cardiovascular disease risks in chronic hemodialysis patients. *Kidney Int* 2000; 58: 353-62.
34. O'Hare AM, Hsu CY, Bacchetti P, Johansen KL. Peripheral vascular disease risk factors among patients undergoing hemodialysis. *J Am Soc Nephrol* 2002; 13: 497-503.
35. Ix JH, Biggs ML, Kizer JR, Mukamal KJ, Djousse L, Zieman SJ, et al. Association of body mass index with peripheral arterial disease in older adults: the cardiovascular health study. *Am J Epidemiol* 2011; 174: 1036-43.

---

ความชุกและปัจจัยเสี่ยงต่อการเกิดโรคหลอดเลือดแดงส่วนปลายตีบในผู้ป่วยไตวายระยะสุดท้ายที่ได้รับการฟอกไต

อดิพร อิงค์สาธิต, วรเวสส นิสสัยสรการ, ผ่องพรรณ ทานาก, ชาตรีย์ กิตติยากร, วสันต์ สุเมธกุล, สุรศักดิ์ กันตชูเวสศิริ, ปิยะนุช พุตระกูล

**วัตถุประสงค์:** เพื่อวิเคราะห์ความชุกและปัจจัยเสี่ยงต่อการเกิดโรคหลอดเลือดแดงส่วนปลายตีบในผู้ป่วยไตวายระยะสุดท้ายที่ได้รับการฟอกไต

**วัสดุและวิธีการ:** วิเคราะห์อัตราส่วนความดันโลหิตระหว่างข้อเท้าและต้นแขน และระหว่างนิ้วเท้าและต้นแขน เพื่อแยกผู้ป่วยที่มีโรคหลอดเลือดแดงส่วนปลายตีบออกจากผู้ป่วยที่ไม่มีโรคหลอดเลือดแดงส่วนปลายตีบ โดยเก็บข้อมูลจากผู้ป่วยที่ได้รับการฟอกไตอย่างต่อเนื่องในโรงพยาบาลรามธิบดีในช่วงเดือนกันยายนถึงเดือนธันวาคม พ.ศ. 2556

**ผลการศึกษา:** จากผู้ป่วยที่เข้าร่วมงานวิจัยทั้งหมด 269 คน ค่าเฉลี่ยของอายุเท่ากับ  $48.8 \pm 15.1$  ปี และ 56.9% เป็นเพศชาย ความชุกของโรคหลอดเลือดแดงส่วนปลายตีบโดยพิจารณาจากค่า ABI และ TBI เท่ากับ 11.5% และ 29.7% ตามลำดับ จาก *Multivariate regression analysis* พบว่า BMI ที่สูงขึ้น, ประวัติโรคหลอดเลือดหัวใจตีบ และ *pulse pressure* ที่สูงขึ้น สัมพันธ์กับการเกิดโรคหลอดเลือดแดงส่วนปลายตีบอย่างมีนัยสำคัญ

**สรุป:** ความชุกของโรคหลอดเลือดแดงส่วนปลายตีบในผู้ป่วยไตวายระยะสุดท้ายที่ได้รับการฟอกไตโดยพิจารณาจากค่า ABI เท่ากับ 11.5% ซึ่งน้อยกว่าอัตราชุกที่ได้จาก TBI ซึ่งเท่ากับ 29.7% ปัจจัยเสี่ยงที่เกี่ยวข้องของได้แก่ BMI ที่สูงขึ้น, ประวัติโรคหลอดเลือดหัวใจตีบ และ *pulse pressure* ที่สูงขึ้น

---