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Prevalence of Peripheral Artery Disease and Its Associated Factors in Patients with Predialysis Chronic Kidney Disease in Ouagadougou

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Abstract

Background: Peripheral artery disease (PAD) is associated with increased cardiovascular and overall morbidity and mortality in patients with predialysis chronic kidney disease (CKD). In sub-Saharan Africa, data on PAD in this population are scarce. **Objectives:** To evaluate the prevalence of PAD and identify associated factors in a predialysis CKD patient population. **Patients and Methods:** This cross-sectional study was conducted from June 1 to August 31, 2019, in Ouagadougou, Burkina Faso. Patients aged at least 18 years, who had been followed-up for at least three months for CKD, and who were not undergoing hemodialysis were included. PAD was defined as an ankle-brachial index (ABI) value of <0.9. **Results:** A total of 138 patients with a mean age of 56.76 years ± 15.13 years were included in the study. The sex-ratio was 1.06. The average glomerular filtration rate (GFR) was 37.50 mL/min/1.73m^2 ± 29.45 mL/min/1.73m^2, and 81.16% of patients had stage 5 CKD. PAD was present in 77 patients (55.8%). Its prevalence increased overall with the decline of renal function. In the univariate analysis, the following variables were found to be statistically

correlated with PAD: history of dyslipidemia (OR = 1.89; 95% CI: [1.6 - 2.2]), diabetic nephropathy (OR = 1.50; 95% CI: [1.11 - 2.03]), autosomal dominant polycystic kidney disease (OR = 0.24; 95% CI: [0.04 - 1.04]), pulse pressure (PP) > 60 mmHg (OR = 2.08; 95% CI: [1.05 - 4.18]), hyperuricemia (OR = 2.97; 95% CI: [1.41 - 6.36]), high triglyceride levels (OR = 7.36; 95% CI: [1.02 - 92.6]), and severe anemia (OR = 2.16; 95% CI: [1.7 - 2.6]). PP > 60 mmHg and hyperuricemia were independently associated with PAD, with odds ratios of 2.11 (95% CI: [1.05 - 4.24]) and 2.8 (95% CI: [1.38 - 5.66]), respectively, after adjusting for sex, age, and CKD duration. **Conclusion:** The prevalence of PAD is high in patients with predialysis CKD and generally increases with declining renal function.

Keywords

Peripheral Artery Disease, Ankle-Brachial Index, Chronic Kidney Disease, Prevalence, Associated Factors

1. Introduction

Cardiovascular complications are the leading cause of death in patients with CKD [1]. Since 1990, cardiovascular mortality related to chronic renal failure (CRF) has increased by 33.8% [1]. PAD is an atherosclerotic disease of the arteries of the lower limbs. In 2019, approximately 1.52% of the world's population had PAD, 42.61% of whom lived in developing countries [2].

The prevalence of PAD is higher in patients with CKD than in the general population (3): CKD is a significant independent risk factor of PAD and is associated with increased prevalence of both traditional and non-traditional cardiovascular risk factors (CVRF) [3]-[5]. PAD is associated with an increase in cardiovascular and overall morbidity and mortality in both the general population and in patients with CKD [6] [7]. Half of the patients with PAD have concomitant coronary artery disease, and 20% have cerebrovascular disease [6]. More than half of patients with PAD are asymptomatic [8]. However, the risk of death and the occurrence of cardiovascular events increased in both symptomatic and asymptomatic patients [6] [9]. The ABI is a non-invasive and reliable screening test for subclinical PAD. In adults, the normal ABI range is 1 to 1.4 [3]. An ABI < 0.9 indicates PAD [3], while an ABI > 1.4 reflects arterial stiffness due to arterial medial calcification [3] [9].

In a previous study conducted in Senegal [10], the prevalence of PAD was 47.16% among chronic hemodialysis patients. The elevated prevalence of PAD observed in patients with stage 5 CKD undergoing hemodialysis suggests that it may be present from the early stages of CKD and progresses concomitantly with the decline of renal function. Few studies on PAD have been conducted in sub-Saharan Africa in the population of patients with predialysis CKD. The objective of this study was to assess the prevalence of PAD in a population of patients with stages 1 to 5 CKD who are not on dialysis and to identify the associated factors.

2. Patients and Methods

2.1. Study Design and Participants

A cross-sectional study was conducted from June 1 to August 31, 2019, in the nephrology and hemodialysis department of the Yalgado Ouedraogo University Hospital in Ouagadougou, Burkina Faso. Patients aged 18 years or older who had been followed for at least three months in outpatient nephrology consultations for CKD were included in the study. Patients who had started hemodialysis were not included. All subjects had given their free and informed consent prior to their involvement in the study. The study protocol had been reviewed and approved by an Institutional Review Board of the Yalgado Ouedraogo Hospital, and the study was conducted in accordance with the ethical standards of the Declaration of Helsinki.

2.2. Clinical Data

The data were obtained from an interview with the patients, a physical examination, and a review of their clinical records. The information collected included sociodemographic parameters (age, sex), medical history (diabetes mellitus, hypertension, dyslipidemia, gout, stroke/transient ischemic attack, angina, myocardial infarction), lifestyle (alcohol consumption, smoking), primary kidney disease, and intermittent claudication. Smoking was defined as the consumption of at least 100 cigarettes in a lifetime [4]. Regular alcohol consumption was defined as the consumption of at least one drink containing alcohol every week for a period of at least one year [4]. Intermittent claudication was defined as pain, cramps, or unilateral or bilateral muscular discomfort in the lower limbs that occurs during physical activity, is reproducible, and is relieved by rest [5]. Angina pectoris, myocardial infarction, and/or stroke/transient ischemic attack were grouped as cardiovascular events.

Physical examination, blood pressure measurement, anthropometric data (weight, height), palpation of the distal pulses in the lower limbs (femoral, popliteal, posterior tibialis, and dorsalis pedis arteries), and calculation of the ABI were realized. The body mass index (BMI) was calculated using the following formula: BMI = Weight (kg)/Height^2 (m) [11]. Obesity was defined as a BMI ≥ 30 kg/m^2 [12].

Blood pressure was measured on both arms after a 10-minute rest in a calm room, using a Holtex® aneroid sphygmomanometer with an appropriately sized cuff (adult or obese) and a Littmann® Classic stethoscope. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were determined by Korotkoff phase I and V, respectively. The mean brachial SBP and DBP measurements were utilized in the present study. Hypertension was defined as SBP ≥ 140 mmHg and/or DBP ≥ 90 mmHg and/or the use of antihypertensive medication [13]. Pulse pressure (PP) was calculated using the following formula: PP = SBP − DBP. PP > 60 mmHg was considered high.

2.3. Measurement of the ABI and Diagnosis of PAD

The measurements were performed by a single trained operator, with patients in the supine position, at rest for at least 10 minutes prior to the assessment. Brachial SBP was obtained using the aforementioned procedure.

The ankle SBP was obtained using a Holtex® aneroid sphygmomanometer and a Bidop Hadeco® bidirectional Doppler device (ES-100V3, Kawasaki, Japan) with an 8 MHz transducer.

The ankle SBP was measured in the posterior tibial and dorsalis pedis arteries. After the palpation of the artery, Doppler gel was applied to the skin, and the Doppler probe was held at an angle of 45˚ to 60˚ relative to the vessel to optimize signal quality. The inflatable cuff, placed 2 cm above the tibial malleolus, was progressively inflated to 20 mmHg above the pressure at which the Doppler signal became undetectable, then slowly deflated at a rate of 2 mmHg per second. The pressure at which the Doppler signal reappeared was the SBP of the examined artery. The mean ankle SBP for each leg was determined as the average of the posterior tibial and dorsalis pedis arteries' SBP. The ABI for each leg was calculated by dividing the mean of ankle SBP measurements by the mean of brachial SBP measurements [14]. The average of the right and left ABI was used for the analysis [15], and PAD was defined by a low ABI < 0.9 [4] [15].

2.4. Laboratory Analyses

Blood parameters (creatinine, uric acid, C-reactive protein (CRP), total cholesterol, HDL-cholesterol, LDL-cholesterol, triglycerides, calcium, phosphate, hemoglobin) and 24-hour proteinuria were studied. The standards of our laboratory were considered. The following abnormalities were examined: hypocalcemia (calcemia < 2.1 mmol/l), hyperphosphatemia (phosphatemia > 1.3 mmol/l), hyperuricemia (uricemia > 357 µmol/l in women and > 428 µmol/l in men), and high CRP when it was ≥ 6 mg/dl. Diabetes mellitus was defined by a fasting blood glucose level of ≥ 1.26 g/l on two separate occasions, or a random blood glucose level of ≥2 g/l in conjunction with suggestive symptoms, or an oral glucose tolerance test result of ≥ 2 g/l, or the use of antidiabetic treatment [16]. Dyslipidemia was defined as total hypercholesterolemia > 2.4 g/l, hypo-HDL-cholesterolemia < 0.4 g/l, hyperLDL-cholesterolemia ≥ 1.6 g/l, hypertriglyceridemia ≥ 2 g/l and/or the use of lipid-lowering treatment. Anemia was defined as a hemoglobin level below 13 g/dl in men and 12 g/dl in women; it was severe when the hemoglobin level was ≤ 7 g/dl. Patients with urine protein levels of ≥0.5 g per 24 hours were designated as having proteinuria.

2.5. Estimation of the Glomerular Filtration Rate (GFR) and Definition of CKD

The estimated glomerular filtration rate (eGFR) was calculated from serum creatinine using the simplified Modification in Diet in Renal Disease (MDRD) formula

[17]: in men = 186 × (creatinine (µmol/l) × 0.0113)$^{-1.154}$ × age$^{-0.203}$ × 1.21 for subjects of African origin × 0.742 for women.

The eGFR was used to classify CKD into 5 stages [18]: stage 1: kidney damage with a normal or increased eGFR ≥ 90 ml/min/1.73m^2; stage 2: kidney damage with mild decrease in eGFR (60 ≤ eGFR < 90 ml/min/1.73m^2); stage 3: moderate kidney disease (30 ≤ eGFR < 60 ml/min/1.73m^2); stage 4: severe kidney disease (15 ≤ eGFR < 30 ml/min/1.73m^2) and stage 5: kidney failure (eGFR < 15 ml/min/1.73m^2). CKD was defined as abnormalities of kidney structure or function or decreased GFR < 60 ml/min/1.73m^2 present for 3 months or more [19].

2.6. Statistical Analysis

The data were analyzed using Microsoft Excel (version 2016) and R-Studio (version 1.2.5001, 2009-2019). Quantitative variables were expressed as means ± standard deviations, and qualitative variables, as numbers and percentages. For the comparison of qualitative variables, the Chi-square test was used for sample sizes ≥ 5 and Fisher's exact test for sample sizes < 5. The Student's t-test was used to compare quantitative variables. Multivariate analysis was performed by logistic regression. The model was adjusted for sex, age, and duration of CKD. Values of $p < 0.05$ were considered significant.

3. Results

During the study period, 269 patients were followed up in the nephrology outpatient consultations, of whom 138 (51.31%) met the inclusion criteria (**Appendix 1**).

3.1. General Characteristics of the Study Population

The mean age of the study population was 56.76 ± 15.13 years (range 23 - 84 years). The sex-ratio was 1.06. The most frequent CVRFs were hypertension and diabetes mellitus, which were observed in 106 (76.8%) and 32 (23.2%) patients, respectively. The mean follow-up duration for the CKD was (47.78 ± 32.92) months (range: 7 and 187 months). The etiology of CKD was predominantly benign nephroangiosclerosis (NAS) (46.38%), chronic glomerulonephritis (GN) (16.67%), and diabetic nephropathy (13.04%). Six patients (4.35%) had intermittent claudication. Peripheral pulses were weakly perceived in 18 patients (13.04%). The average eGFR was (37.50 ± 29.34) ml/min/1.73m^2 (range 2.68 - 157.3 ml/min/1.73m^2). One hundred and twelve patients (81.16%) had an eGFR < 60 ml/min/1.73m^2, with approximately one-third (32.6%) of patients having an eGFR between 30 and 60 ml/min/1.73m^2 (**Table 1**).

3.2. Distribution of the ABI according to the eGFR

Patients' average ABI was 0.89 ± 0.19 (range 0.43 - 1.69). The maximum, median, and minimum ABI values decreased overall from CKD stage 1 to 5, with the exception of stage 3 (**Figure 1**).

Table 1. General characteristics of the study population.

Parameters	(N = 138) (%)
Gender	
Male	71 (51.45)
Female	67 (48.55)
History	
Hypertension	106 (76.8)
Diabetes mellitus	32 (23.2)
Alcohol consumption	27 (19.6)
Smoking	16 (11.6)
Gout	8 (5.8)
Dyslipidemia	7 (5.1)
*Cardiovascular event	9 (6.52)
Primary disease	
NAS	64 (46.38)
GN	23 (16.67)
Diabetic nephropathy	18 (13.04)
ADPKD	12 (8.7)
CTIN	8 (5.8)
Unknown nephropathy	11 (7.97)
Unilateral renal agenesis	2 (1.45)
Intermittent claudication	6 (4.35)
Decrease/Abolition of peripheral pulses	18 (13.04)
eGFR (mL/min/1.73m^2)	
≥90	9 (6.52)
]90 - 60]	17 (12.32)
]60 - 30]	45 (32.6)
]30 - 15]	36 (26.1)
<15	31 (22.46)

ADPKD: Autosomal Dominant Polycystic Kidney Disease; CTIN: Chronic Tubulointerstitial Nephritis; GN: Glomerulonephritis; NAS: Nephroangiosclerosis. *Cardiovascular event: stroke: 7, myocardial infarction: 1, angina pectoris: 1.

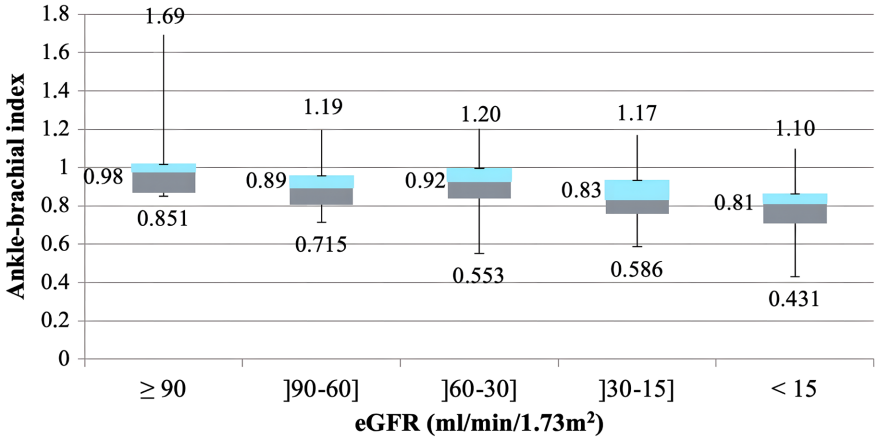

Figure 1. Distribution of ABI according to estimated glomerular filtration rate.

3.3. Prevalence of PAD

The ABI was less than 0.9 in 76 patients, with a prevalence of PAD of 55.07%. Two patients (1.45%) had an ABI > 1.4. The mean ABI of patients with PAD was significantly lower than that of patients without PAD (0.79 ± 0.09 vs. 1.03 ± 0.19, p = 10^{-6}). A significant proportion of patients with stage 4 (69.44%) and 5 (64.52%) CKD were found to have PAD. The prevalence of PAD increased overall with the decline in renal function, without a statistically significant correlation (p = 0.064) (Figure 2).

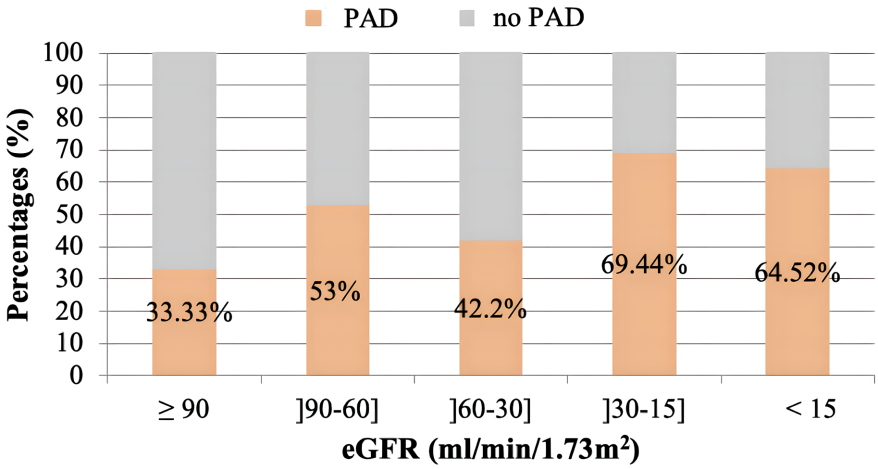

Figure 2. Percentage of patients with PAD according to the stage of CKD.

3.4. Factors Associated with PAD

PAD was statistically correlated with a history of dyslipidemia (OR = 1.89; 95% CI: [1.6 - 2.2]), diabetic nephropathy (OR = 1.50; 95% CI: [1.11 - 2.03]), autosomal dominant polycystic kidney disease (ADPKD) (OR = 0.24; 95% CI: [0.05 - 0.91]) and PP > 60 mmHg (OR = 2.08; 95% CI: [1.05 - 4.18]). A comparison of the two groups (with PAD vs. without PAD) revealed that patients with PAD were more likely to be older (51.32% vs. 45.16%; p = 0.471), diabetic (28.94% vs. 16.13%; p =

0.075), and to have a history of cardiovascular events (7.9% vs. 4.84%; p = 0.469). However, these differences did not reach statistical significance. There was no association between PAD and intermittent claudication (p = 0.317), nor poor perception of peripheral pulses on palpation (p = 0.290) (Table 2).

Table 2. Association between PAD and sociodemographic and clinical parameters.

Parameters	All (N = 138)	PAD		p-value	OR [95% CI]
		Yes (n = 76)	No (n = 62)		
Age ≥ 60 years	67 (48.55%)	39 (51.32%)	28 (45.16%)	0.471	-
Gender					
Male	71 (51.45%)	39 (51.32%)	32 (51.6%)	0.972	-
Female	67 (48.55%)	37 (48.68%)	30 (48.38%)		
CKD duration (months) (mean ± SD)	47.78 ± 32.92	51.21 ± 35.28	43.58 ± 29.51	0.177	-
History					
Hypertension	106 (76.8%)	59 (77.63%)	47 (75.8%)	0.800	-
Diabetes mellitus	32 (23.2%)	22 (28.94%)	10 (16.13%)	0.075	-
Dyslipidemia	7 (5.1%)	7 (9.21%)	0	0.014	1.89 [1.6 - 2.2]
Smoking	16 (11.6%)	7 (9.21%)	9 (14.52%)	0.332	-
Alcohol consumption	27 (19.6%)	11 (14.47%)	16 (25.8%)	0.095	-
Gout	8 (5.8%)	4 (5.26%)	4 (6.45%)	0.766	-
Cardiovascular event	9 (6.52%)	6 (7.9%)	3 (4.84%)	0.469	-
Primary disease					
NAS	64 (46.38%)	40 (52.63%)	24 (38.71%)	0.102	-
GN	23 (16.67%)	11 (14.47%)	12 (19.35%)	0.444	-
Diabetic nephropathy	18 (13.04%)	14 (18.42%)	4 (6.45%)	0.037	1.50 [1.11 - 2.03]
ADPKD	12 (8.7%)	3 (3.95%)	9 (14.52%)	0.035	0.24 [0.04 - 1.04]
CTIN	8 (5.8%)	3 (3.95%)	5 (8.06%)	0.466	-
SBP (mmHg)					
≥140	67 (48.55%)	40 (52.63%)	27 (43.55%)	0.288	-
<140	71 (51.45%)	36 (47.37%)	35 (56.45%)		

Continued

DBP (mmHg)					
≥90	29 (21%)	17 (22.37%)	12 (19.35%)	0.665	-
<90	109 (79%)	59 (77.63%)	50 (80.65%)		
PP (mmHg)					
>60	74 (53.62%)	47 (61.84%)	27 (43.55%)	0.032	2.08 [1.05 - 4.18]
≤60	64 (46.38%)	29 (38.16%)	35 (56.45%)		
Obesity				0.525	-
Yes	16 (11.6%)	10 (13.16%)	6 (9.68%)		
No	122 (88.4%)	66 (86.84%)	56 (90.32%)		
Claudication					
Yes	6 (4.35%)	5 (6.58%)	1 (1.61%)	0.317	-
No	132 (95.65%)	71 (93.42%)	61 (98.39%)		
Lower extremities pulses					
Attenuated/Absent	18 (13.04%)	12 (15.8%)	6 (9.68%)	0.290	-
Present	120 (86.96%)	64 (84.2%)	56 (90.32%)		

ADPKD: Autosomal Dominant Polycystic Kidney Disease; CTIN: Chronic Tubulo-Interstitial Nephritis; DBP: Diastolic Blood Pressure; GN: Glomerulonephritis; NAS: Nephroangiosclerosis; PP: Pulse Pressure; SBP: Systolic Blood Pressure; SD: Standard Deviation.

Hyperuricemia (OR = 2.97; [95% CI: 1.41 - 6.36]), hypertriglyceridemia (OR = 7.36; 95% CI: [1.02 - 92.6]), and severe anemia (OR = 2.16; 95% CI: [1.7 - 2.6]) were statistically correlated with PAD. The proportion of patients with proteinuria was higher in the PAD group (61.11% vs. 42.6%; p = 0.054) (Table 3).

Table 3. Association between PAD and biological parameters.

Parameters	All (N = 138)	PAD		p-value	OR [95% CI]
		Yes (n = 76)	No (n = 62)		
eGFR (ml/min/1.73m²)					
<60	112 (81.16%)	64 (84.2%)	48 (77.4%)	0.312	-
≥60	26 (18.84%)	12 (15.8%)	14 (22.6%)		
Uric acid (mmol/l) (n = 121)					
High	67 (55.37%)	44 (67.7%)	23 (41.07%)	0.003	2.97 [1.41 - 6.36]
Normal	54 (44.63%)	21 (32.3%)	33 (58.93%)		

Continued

Calcium (mmol/l) (n = 130)					
<2.1	26 (20%)	15 (22.06%)	11 (17.74%)	0.538	-
≥2.1	104 (80%)	53 (77.94%)	51 (82.26%)		
Phosphorus (mmol/l) (n = 121)					
>1.3	43 (35.54%)	21 (35%)	22 (36.06%)	0.902	-
≤1.3	78 (64.46%)	39 (65%)	39 (63.94%)		
Total cholesterol (g/l) (n = 40)					
>2.4	38 (95%)	22 (100%)	16 (88.9%)	0.392	-
≤2.4	2 (5%)	0	2 (11.1%)		
HDL-cholesterol (g/l) (n = 36)					
<0.4	1 (2.78%)	1 (5%)	0	0.999	-
≥0.4	35 (97.22%)	19 (95%)	16 (100%)		
LDL-cholesterol (g/l) (n = 34)					
≥1.6	32 (94.12%)	18 (100%)	14 (87.5%)	0.427	-
<1.6	2 (5.88%)	0	2 (12.5%)		
Triglycerides (g/l) (n = 28)					
≥2	10 (35.7%)	2 (14.3%)	8 (57.1%)	0.046	7.36 [1.02 - 92.6]
<2	18 (64.3%)	12 (85.7%)	6 (42.9%)		
CRP (mg/l) (n = 19)					
≥6	10 (52.63%)	8 (53.33%)	2 (50%)	0.452	-
<6	9 (47.37%)	7 (46.67)	2 (50%)		
Hemoglobin (g/dl) (n = 120)					
≤7	6 (5%)	6 (10.34%)	0	0.022	2.16 [1.7 - 2.6]
>7	114 (95%)	52 (89.66%)	62 (100%)		
Proteinuria (g/24 h) (n = 108)					
≥0.5	56 (51.85%)	33 (61.11%)	23 (42.6%)	0.054	-
<0.5	52 (48.15%)	21 (38.89%)	31 (57.4%)		

CRP: C-Reactiv Protein; eGFR: Estimated Glomerular Filtration Rate; HDL: High Density Lipoprotein; LDL: Low Density Lipoprotein.

In the multivariate analysis, only high PP (OR = 2.11; 95% CI: [1.05 - 4.24]) and hyperuricemia (OR = 2.8; 95% CI: [1.38 - 5.66]) held statistical significance in their association with PAD, after adjustment for age, sex, and duration of CKD (Table 4).

Table 4. Factors associated with PAD in logistic regression, after adjustment for age, sex, and CKD duration.

Parameters	p-value	OR [95% CI]
Diabetic nephropathy	0.052	-
ADPKD	0.059	-
History of dyslipidemia	0.99	-
PP (mmHg)		
>60	0.036	2.11 [1.05 - 4.24]
≤60		
Uric acid (mmol/l)		
High	0.004	2.8 [1.38 - 5.66]
Normal		
Triglycerides (g/l)		
≥2	0.054	-
<2		
Hemoglobin (g/dl)		
≤7	0.99	-
>7		

ADPKD: Autosomal Dominant Polycystic Kidney Disease; PP: Pulse Pressure.

4. Discussion

In sub-Saharan Africa, the prevalence of PAD ranges from 39% to 52% among populations with a high cardiovascular risk [20]. The elevated prevalence of PAD (55.07%) observed in this study corroborates this observation and is consistent with prior research findings [10] [14] [21]. The prevalence of PAD was 52.5% in a population of diabetic patients in Nigeria [14]; the ABI calculation method used was the same as in our study. In the study by Abu *et al.* [21], PAD, diagnosed by Doppler ultrasound of the lower limb arteries, was present in 48.4% of chronic hemodialysis patients. In France, the prevalence of PAD was 41.1% in a cohort of 2146 patients at high cardiovascular risk, 39.5% of whom had an eGFR of less than 60 ml/min/1.73m^2 [8]. However, Ovwasa *et al.* [22] reported a lower prevalence of 24% in a population of hypertensive patients with predialysis CKD.

The high prevalence of PAD in this population can be attributed to several factors. CKD is an independent risk factor for PAD [3] and is associated with an "accelerated" arterial aging process characterized by atherosclerosis and arteriosclerosis [23]. This phenomenon can be attributed to the synergistic and cumula-

tive effects of both traditional (e.g., advanced age, diabetes mellitus, hypertension, dyslipidemia, smoking) and non-traditional (e.g., chronic inflammation, oxidative stress, abnormal angiogenesis, albuminuria) CVRF in CKD [3] [5] [23]. The presence of uremia-related CVRF, including anemia, calcium and phosphate metabolism disorders, and uremic toxins, contributes to the increased risk of CVD in patients with CKD [3] [23].

In the general population, the ABI is slightly lower in women, individuals of short stature, those of African descent, and the elderly [9]. When considered individually, these parameters exert minimal influence on the ABI. However, their combined effect in a certain proportion of a study population may lead to an overestimation of the prevalence of PAD [9]. The high prevalence of PAD in the present study may be partially explained by ethnic factors. In the United States, the prevalence of PAD is two to three times higher in African Americans than in other ethnic groups [24].

According to the ABI calculation method, the proportion of patients with PAD varies within the same population [9]. Thus, in a study of 15,105 apparently healthy individual in Brazil, the prevalence of PAD was 2.7% when the lowest of the four systolic pressures recorded at the ankles was used for the calculation, 0.5% with the highest pressure of the four systolic pressures recorded at the ankles and 0.9% with the average of the four systolic pressures of the ankles [25]. In a review by Xu *et al.* [26], the first method demonstrated higher sensitivity than the second method (83.7% and 89% vs. 68% and 69.3%) but lower specificity (between 64.3% and 93% vs. 83.3% and 99%). The third method, which was utilized in this study, exhibited intermediate sensitivity (70.6%) and specificity (88.5%).

The ABI measurement is a diagnostic tool that has been found to be effective in the early detection of subclinical PAD. In fact, less than 15% of patients with PAD diagnosed by ABI calculation have typical claudication [5]. In the present study, only 6.58% of patients with PAD had claudication. However, the ABI may be falsely normal or elevated (>1.4) despite the presence of PAD in older subjects, patients with advanced CKD, and patients with diabetes mellitus. These individuals tend to have calcifications and rigidification of the arterial wall, leading to incompressible arteries. In such cases, measurement of the toe-brachial index or the post-exercise ABI limits the risk of underdiagnosing PAD [3] [9].

The prevalence of PAD increases with the progression of CKD [3] [22] [27]. We made the same observation overall: the proportion of patients with PAD increased as GFR decreased, with the exception of stage 3 CKD (**Figure 2**). In the series by Luo *et al.* [27], the prevalence of PAD increased from 22.3% in patients with normal renal function to 48.7% in stage 4 CKD. In the study by Chen *et al.* [4], according to eGFR between 50 and 60 ml/min/1.73m^2, 40 and 49 ml/min/1.73m^2, 30 and 39 ml/min/1.73m^2, and <30 ml/min/1.73m^2, the prevalences of PAD were 16.5%, 19.4%, 20.7% and 20.9%. The decrease in GFR and proteinuria are independent risk factors for PAD [28]. The prevalence of PAD is greater in patients with decreased GFR and microalbuminuria [29]. In our study, the prevalence of

PAD was higher in patients with a GFR < 60 ml/min/1.73m^2 (84. 2% vs. 77.4%; p = 0.312) and in patients with proteinuria (61.11% vs. 42.6%; p = 0.0541).

Traditional CVRF, including diabetes mellitus, hypertension, dyslipidemia, and smoking, are associated with PAD [4] [6] [30]. The presence of diabetes mellitus results in a two to fourfold elevated risk of developing PAD [6]. This risk is known to increase in proportion to the duration and severity of diabetes mellitus. Diabetic nephropathy is a microvascular complication that occurs in approximately 40% of diabetic patients [31]. In diabetic patients, inadequate glycemic control, insulin resistance, hyperinsulinism, and the presence of CVRF, notably hypertension, obesity, and dyslipidemia, result in the long-term development of microvascular and macrovascular lesions through endothelial dysfunction and chronic inflammation. The present study identified a history of dyslipidemia, diabetic nephropathy, hypertriglyceridemia, and severe anemia as statistically associated with PAD. However, these associations lost significance in multivariate analysis after adjustment for sex, age, and duration of CKD. This finding may be attributable to the limited sample size and the presence of potential confounding variables. The prevalence of CVRF and the risk of PAD increase with age and the progression of CKD [23], as severe anemia is more frequently observed in advanced CKD. Similar to Pessinaba *et al.* in Senegal [32] and unlike other authors [27] [29] [33], we did not find a correlation between smoking and PAD. However, smoking increases the risk of PAD two to three times, especially when it is associated with other CVRF [6] [30]. The relatively low number of patients who are smokers in Pessinaba's [32] series and in our cohort provides a potential explanation for this result.

Elevated PP was independently associated with PAD in our study, as well as in other publications [34]-[36]. In a population of subjects aged at least 50, each 10 mmHg increase in PP was associated with a 19% rise in the risk of developing PAD [36]. In a cohort of 1795 patients with advanced CKD not undergoing dialysis, Bansal *et al.* [37] found a strong association between PP and PAD, with the risk of occurrence 2.67 times higher in patients with PP > 68 mmHg than in those with PP < 51 mmHg. An elevated PP is an indicator of arterial stiffness and premature vascular aging due to arteriosclerosis [35].

Consistent with the findings of other authors [34] [38] [39], a correlation between elevated uric acid levels and PAD has been observed in this study. Uric acid has a dual property: it is both beneficial as an antioxidant and harmful as a prooxidant [4] [40]. Hyperuricemia plays a pro-atherogenic role and is directly linked to PAD by the chronic inflammation and endothelial dysfunction it promotes [40]. The association between PAD and hyperuricemia may be indirect as elevated uric acid levels are frequently linked to various CVRF, particularly hypertension, diabetes mellitus, dyslipidemia, and CKD [40]. Two-thirds of uric acid is excreted by the kidneys [40]. After taking kidney function into consideration by using fractional excretion of uric acid (FEUA), Hu *et al.* [41] found an independent association between lower FEUA and a higher prevalence of low ABI. This finding indicates that renal function may play a mediating role in uric acid's contribution

to the development of PAD.

The etiology of CKD is an important prognostic factor because the rate of decline in renal function and the profile of associated complications differ according to the nature of the primary disease. In the present study, an initial protective association between ADPKD and PAD was observed in univariate analysis. However, this association lost its statistical significance after multivariate adjustment. In a prospective study conducted in South Korea, patients with diabetic nephropathy and ADPKD were found to be at a higher risk of cardiovascular complications (coronary artery disease, PAD, ischemic and hemorrhagic stroke, cerebral arterial aneurysm, heart failure) and death than those with chronic glomerulonephritis or nephroangiosclerosis [42]. In ADPKD, dysregulation of factors involved in angiogenesis and vascular remodeling induces structural and functional abnormalities of the blood vessels [43]. In the series by Helal *et al.* [44], PAD was the second most common cardiovascular complication in patients with ADPKD, occurring in 16.5% of cases.

Our study had some limitations. The unavailability of certain paraclinical tests for some patients may have led to selection bias. Moreover, given the cross-sectional nature of the study, it was not possible to ascertain a causal relationship between the associated factors identified and peripheral PAD. Furthermore, the method used to calculate the ABI, which is the ratio of mean ankle SBP to mean brachial SBP, may have led to an overestimation of PAD prevalence. However, this effect could be offset by defining PAD as an ABI of less than 0.9, which could lead to an underestimation of its prevalence in patients with a reduced glomerular filtration rate.

5. Conclusion

This study reveals a high prevalence of PAD (55.8%) in patients with predialysis CKD. Pulse pressure and hyperuricemia were independently associated with PAD. Early PAD screening by ABI measurement, especially in CKD patients with increased pulse pressure and hyperuricemia, is easy to implement and should be integrated into nephrological follow-up in Burkina Faso. This would allow for a reassessment of cardiovascular risk in this population and the initiation of appropriate therapeutic measures to reduce the high cardiovascular morbidity and mortality in these patients. Further multicenter cohort studies are needed to determine the incidence of PAD and its risk factors in this population.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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Appendix 1. Flow Chart