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Circadian blood pressure variability and associated factors among chronic kidney disease patients at Nekemte Town public Hospitals, West Oromia, Ethiopia: a comparative cross-sectional study

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ABSTRACT

Objective This study aimed to assess the pattern of circadian blood pressure variability (CBPV) and associated factors among chronic kidney disease (CKD) patients admitted to Nekemte Town public Hospitals.

Design A hospital-based comparative cross-sectional study was conducted among 130 CKD patients from 01 October to 02 December 2022. Comparisons were performed between the groups using an independent t-test for CBPV (24-hour blood pressure (BP), daytime BP and night-time BP). The dipping pattern was compared by the χ^2 test. Multiple logistic regression was used to determine the factors associated with non-dipping patterns in patients with hypertensive CKD (HCKD).

Setting Two public hospitals in the Nekemte town, Western Ethiopia.

Participants The participants were two independent groups. Group I (HCKD=65) and group II (normotensive CKD (NCKD)=65).

Results The mean 24-hour SD of systolic blood pressure (SBP) was significantly different between HCKD and NCKD patients, 10.17±6.12 mm Hg versus 0.5.4±2.7 mm Hg, respectively (95% CI 0.02 to 1.77, p=0.043). The prevalence of SBP non-dippers was greater among HCKD than NCKD patients (83% vs 63%). Mean 24-hour SBP (95% CI 1.50 (1.15 to 1.96), p=0.003) and estimated glomerular filtration rate (eGFR) (95% CI 2.92 (1.21 to 47.06), p=0.038) were independently associated with non-dipping SBP in HCKD patients.

Conclusion Compared with NCKD patients, HCKD patients had significantly greater CBPV. Compared with dippers, non-dippers had a lower mean eGFR.

STRENGTHS AND LIMITATIONS OF THIS STUDY

⇒ This study compared the circadian blood pressure fluctuation, which is an additional variable of kidney disease progression.
⇒ The frequency of blood pressure measurements was relatively lower than the current standard for ambulatory blood pressure monitoring.
⇒ Due to the cross-sectional nature of the study design, cause-and-effect linkages cannot be determined.
⇒ Since the participants were hospital-admitted patients, the effect of physical activity was not determined.

BACKGROUND

Circadian rhythm refers to any biological activity in the body that repeats itself over 24 hours and maintains this regularity in the absence of external stimuli.[1] Blood pressure (BP) is a physiological variable that is controlled by the body's internal circadian clock, which is located in the suprachiasmatic nucleus.[2] BP fluctuates throughout the day, with a dramatic morning surge around 10:00 and a low at 15:00.[3]

Hypertension (HTN) is the second most common cause of chronic kidney disease (CKD). Approximately 43.2% of CKD cases were caused by elevated BP.[4] However, there is a vicious cycle between HTN and CKD. Kidney function deteriorates due to HTN, and BP regulation may also worsen due to the gradual loss of kidney function.[5 6]

In addition to high BP, variability in circadian BP (CBP) is a predictor of target organ damage.[7] Non-dipping is linked to the progression of CKD and the riser was 2.5 times higher in CKD and five times higher in end-stage kidney disease.[8 9]

Globally, the incidence of CKD is increasing; 13.4% of the general population has CKD at every stage.[10] The prevalence of CKD across the world's population increased by 29.3% between 1990 and 2017, while the mortality rate rose by 41.5%, according to the Global Burden of Diseases (GBD) study.[4] CKD is

anticipated to become the fifth most prevalent cause of death worldwide by 2040.[11]

The overall incidence of CKD in Africa was 10%, while that in HTN patients was 34.5%.[12] According to a systematic review and meta-analysis, the pooled estimate of CKD in Ethiopia was 21.71%, with stage 2 CKD accounting for 30.76% of the total CKD incidence.[13]

The GBD assessment revealed that 46% of the deaths among women and 45% of the deaths among men among CKD patients were attributable to HTN.[14] The adjusted risk of reduced estimated glomerular filtration rate (eGFR) is 76% greater in hypertensive individuals than in normotensive individuals. Similarly, the risk of developing a reduced eGFR is 25% greater in people with pre-HTN.[15]

The typical CBP variability (CBPV) in HCKD patients, including inhibited nocturnal BP decrease (non-dipping) and increased overnight BP (reverse dipping), is closely linked to kidney damage (lower eGFR or albuminuria).[16] In patients with HCKD, non-dipping BP was prevalent in 80% of the patients in Morocco and 92% of the patients in Nigeria.[17 18]

Although there is well-established evidence that indicates the linkage between CBPV and CKD in hypertensive patients. Few studies showed the association of CBPV with CKD in normotensive patients.[19–22]

Therefore, to address this gap, this study assessed the CBPV among HCKD and normotensive CKD (NCKD) patients

Notably, in Africa, in Ethiopia, studies on CBPV and its association with CKD have been limited. Therefore, the purpose of this study was to evaluate the pattern of CBPV among CKD patients with HTN and normotension.

MATERIALS AND METHODS

Study area and period

The study was conducted between 01 October and 02 December of 2022 among CKD patients admitted to two public hospitals in Nekemte town. These hospitals are Nekemte Specialised Hospital (NSH) and Wollega University Referral Hospital (WURH). These hospitals are found in Nekemte Town which is situated in Western Oromia and 331 km from the capital city Addis Ababa.

Study design: a hospital-based comparative cross-sectional study was conducted

Source population

All CKD patients admitted at public hospitals in Nekemte town during the study period.

Study population

Group I (HCKD): all HCKD patients admitted to medical wards during the study period, who met the inclusion criteria.

Group II (NCKD): all NCKD patients admitted to medical wards during the study period, who met the inclusion criteria.

Inclusion and exclusion criteria

- Inclusion criteria: CKD patients ≥18 years old; those patients who remained in the hospital for the next 24 hours and patients who were willing to participate.
- Exclusion criteria: patients with mental problems; CKD patient on renal replacement therapy (RRT) (i.e., dialysis or renal transplant); diabetic mellitus; obstructive sleep apnea and cardiovascular disease.

Sample size and sampling technique

Sample size determination

The plan was to determine CBPV among HCKD and NCKD patients, which were two independent groups. Therefore, to estimate the mean difference between two independent groups, the formula for determining the sample sizes needed in each comparison group was given below by considering an unequal variance in the two groups and an equal sample size for each group.[23]

$$n = \frac{(Z_{\alpha/2}+Z_{\beta})^2(\sigma_1^2+\sigma_1^2)}{(\mu_1-\mu_2)^2}$$

Whereas:

- $Z_{\alpha/2}$ is the critical value of the normal distribution at $\alpha/2$ (95% CI=0.05 and the critical value is 1.96).
- Zβ is the power of the study (for a power of 80%, the critical value is 0.84).
- μ_1 and σ_1 are the mean and SD of group I, respectively, from the prior study.
- μ_2 and σ_2 are the mean and SD of group II, respectively, from the prior study.

The SD of daytime mean arterial pressure (101.5±21.4 vs 92.7±10.8) from a previous comparative study performed in Nigeria on CKD patients with HTN and CKD-free HTN patients was used.[18]

$$n = \frac{(1.96+0.84)^2(21.4^2+10.8^2)}{(101.5-92.7)^2}$$

n=58.172~59

Therefore, with a 10% contingency for a non-response rate equal to 6, the sample size was 59+6=65 in each group. The total sample size was equal to 130.

Sampling techniques

The study was conducted in two public hospitals in Nekemte town (WURH and NSH). The calculated sample size was proportionally allocated to both hospitals based on the number of patients admitted during the previous quarter.

As both hospitals reported in the previous quarter, the admitted number of CKD patients was 148 in NSH and 134 in WURH. So, the proportional allocation of the sample was 68 (34 HCKD and 34 NCKD) to NSH and 62 (31 HCKD and 31 NCKD) to WURH. Furthermore, in both hospitals, CKD patients were proportionally allocated based on BP (HCKD and NCKD). In each hospital, data were collected randomly from hospitalised CKD patients who met the eligibility criteria for each group.

Data collection instruments and procedures

Data collection instruments

Questionnaire

The questionnaire was developed after considering several reviewed academic papers and WHO non-communicable disease assessment tools.[24] The questionnaire included sociodemographic variables such as age, sex, educational level and residence.

CBP measuring equipment

According to the kidney disease improving global outcomes (KDIGO) 2021 guidelines, the digital oscillometric method is preferable to the auscultator method for manual BP devices.[25] A fully automatic sphygmomanometer BP device (Boso Medicus X, Test Winner Stiftung Warentest 9/2020, German) was used. Boso Medicus X is clinically validated according to the European Society of Hypertension,[26] and a survey was conducted on the Ethiopian population using this device.[27]

Serum creatinine analysis instrument

Test tubes, centrifuges and refrigerators were used for sample preparation, and a Mindray BS-200 Clinical Analyzer (Med Test Dx, USA) was used.

Data collection procedure

Interviews and CBP measurements were conducted by five trained BSc nurses. A laboratory serum creatinine test and estimation of the glomerular filtration rate (GFR) were carried out by a medical laboratory technology professional in the clinical chemistry department. All the data collectors received 1 day of training on the objectives of the study, the purpose of the investigation and the method of measurement. The training also included the current COVID-19 protocol, which includes the use of face masks and the disinfection of equipment.

BP measurement

By using the previously validated procedure in an inpatient context (hospitalised patients), CBP was measured six times at 4-hour intervals during the day and night (06:00, 10:00, 14:00, 18:00, 22:00 and 2:00). Tan Xu and colleagues studied patients with essential HTN in an inpatient environment and compared 24-hour ABPM (ambulatory blood pressure monitoring) with sphygmomanometer measurements (three records during the daytime and three records during the night-time). The average 24-hour BP, daytime BP and nocturnal BP did not differ substantially from the 24-hour ambulatory systolic BP (SBP) (95% CI (1.26 to 0.22)), and sphygmomanometer recording and ABPM strongly agreed in identifying non-dippers (diagnostic agreement=82.58%, κ=0.608, p<0.001).[28]

Using a broad fixed-clock time technique, day and night periods were determined. The BP readings taken at 6:00, 10:00 and 14:00 constituted daytime readings, and those taken at 18:00, 22:00 and 2:00 were termed night-time measurements. The mean SBP and diastolic BP (DBP) during 24 hours, the mean SBP and DBP during the day and night and the SD of the mean 24-hour, mean daytime and mean night-time SBP and DBP were calculated for each patient.

The formula below was used to determine the degree of BP dipping.[29]

$$\left(\text{Dipping status} = \frac{(\text{mean daytime BP} - \text{mean nocturnal BP})}{\text{mean daytime BP}} \times 100\right)$$

The following method was used to measure the CBP as the International Society of Hypertension recommendations.[30] Before the measurement, patients were given a 5-min rest period; The cuff was at heart level; triplicate BP measurements were taken at each measurement, with at least 1 min between measurements, and the average result was recorded; To identify any potential disparities, the BP in both arms was measured during the initial measurement. An arm with a higher reading was used as the standard for all subsequent measurements; the patient was advised not to talk, not to cross his or her legs but rather to sit with his or her back supported and the patient was asked if he/she drank coffee or tea or smoked within 30 min. If yes, measurements were delayed for 30 min.

Serum creatinine level investigation and estimation of the GFR

Sample collection procedure

2 mL of venous blood was collected using a disposable syringe; a grey-top tube with potassium oxalate (an anticoagulant) and sodium fluoride (a preservative) was used to transfer the blood sample; the serum was separated within 45 min–2 hours after the blood sample was centrifuged at 3000 rpm for 10 min. Next, the serum was put into a necked tube and kept at 4–8 ° C for 24 hours until a lab technician could analyse the creatinine concentration.

Sample processing

A Mindray BS-200 automatic serum analyzer was used in the clinical chemistry laboratory to measure the serum creatinine concentration. Using a readily available kit, the Serum creatinine concentration was calculated using the alkaline picrate method (Bonsnes and Taussky). The following formula was used to determine the eGFR using the CKD Epidemiology Collaboration (CKD-EPI) equation for men and women.[31] For females with an SCr concentration ≤0.7 mg/dL, the eGFR was $166\times(SCr/0.7)^{-0.329}\times(0.993)^{age}$. For females with an SCr>0.7 mg/dL, the eGFR was $166\times(SCr/0.7)^{-1.209}\times(0.993)^{age}$. For males with an SCr level ≤0.9 mg/dL, the eGFR was calculated as follows: $eGFR=163\times(SCr/0.9)-0.411\times(0.993)^{age}$. For males with an SCr>0.9 mg/dL, the eGFR was $163\times(SCr/0.9)-1.209\times(0.993)^{age}$.

Five stages of CKD were identified based on the eGFR: stage 1 (eGFR>90 mL/min/1.73 m^2), stage 2 (eGFR 60–89 mL/min/1.73 m^2), stage 3a (eGFR 45–59 mL/min/1.73 m^2), stage 3b (eGFR 30–44 m^2/min/1.73 m^2), stage 4 (eGFR of 15–29 mL/min/1.73 m^2) and stage 5 (eGFR<15 mL/min/1.73 m^2).

Study variables

Dependent variable

SD of CBP pattern.

Independent variables

Sociodemographic: age, sex, education level and residence status.

Clinical variables

Reduced eGFR and BP.

Operational definitions

Chronic kidney disease

Abnormalities of kidney structure or function, present for >3 months, with implications for health as manifested by eGFR >60 mL/min per 1.73 m^2 or markers of kidney damage (elevated proteinuria, albumin-to-creatinine ratio (ACR) and ultrasound kidney damage), or both[25 32]
HCKD: patients who had an office SBP ≥130 mm Hg and a DBP ≥80 mm Hg, or an average SBP ≥125 mm Hg and a DBP ≥75 mm Hg or were currently on medication for HTN.[33]

Circadian blood pressure variability

Variability of average 24-hour BP, daytime BP and overnight BP for both SBP and DBP, explained by the SD of the CBPV index.[34]

Nocturnal BP dipping status

Nocturnal BP dipping is the percentage difference between daytime and night-time mean BP. $\left(\text{Dipping status} = \frac{(\text{mean daytime BP} - \text{mean nocturnal BP})}{\text{mean daytime BP}} \times 100\right)$.[35]

Dippers

BP drops ≥10% of daytime values during the night-time.

Extreme dippers

Dippers in which night-time BP declines >20% of the daytime values.

Non-dippers

When the nocturnal BP decrease was <10% of the daytime values.

Risers

Non-dippers in which night BP is greater than daytime values (night-time fall in BP <0%).

Data quality management and control

Before data collection, five BSc nurses who collected the data were trained for a full day on the COVID-19 protocols, the study's significance, how to measure CBP and other pertinent topics to ensure the quality of the data. The data collection tool was created in English, translated into Amharic and Afaan Oromoo and then returned to English for consistency of the data so that the respondents could comprehend it and provide a correct response. Before beginning the real data collection, 5% of the sample of CKD patients at Arjo Hospital was pretested with the questionnaire and the tools were corrected. The clinical chemistry laboratories' standard operating procedures have been closely adhered to ensure the quality of the laboratory analysis. Physical measurements were taken by the international and national standards. Furthermore, well-trained and experienced laboratory technologists and nurses carried out the laboratory analysis and physical measurements, respectively.

Patient and public involvement

Patients and/or the public were not involved in the design, conduct, reporting or dissemination plans of this research.

Data processing and analysis

The data were cleaned and coded before being entered into Epi Data V.4.4.1. The data were then exported and analysed using SPSS version V.26.0. Continuous variables were reported as mean and SD, while categorical data were presented as frequency and percentage. An independent sample t-test was performed to compare the mean and SD of over 24 hours, daytime and night-time BP for both SBP and DBP. The assumption of independent sample t-test was checked (Levene's test and Shapiro-Wilk test were not significant) for mean 24-hour SBP.

The dipping pattern of BP was computed using the formula (mean daytime minus mean night-time divided by mean daytime)×100 SBP/DBP and expressed as a percentage. The CKD patients were then categorised as non-dippers (BP decreases ≤10%) and dippers (BP decreases >10%). The dipping status (dippers and non-dippers) was compared by χ^2 test among HCKD and NCKD patients. Logistic regression analysis was carried out separately to identify factors associated with non-dipping BP in HCKD patients. Predictor variables with p<0.25 were used in multiple logistic regression analyses. Multiple logistic regression analysis was used to assess variables associated with non-dipping patterns in HCKD patients. Variables with a p<0.05 were declared statistically significant.

RESULTS

Sociodemographic characteristics and behavioural factors

The mean age of the participants was 53.83±13.18 years for hypertensive CKD (HCKD) patients and 55.17±18 years for NCKD patients. The mean ages of the two groups were not significantly different (mean age of HCKD and NCKD, p=0.628); thus, these groups were comparable.

Approximately 33 (50.8%) HCKD patients were female, while the majority of the 44 (67.7%) NCKD patients were male. Regarding the educational status of the participants, less than half (38.5%) of the participants were illiterate, 21 (32.3%) had secondary school education in HCKD, and 39 (60.0%) of the NCKD patients were illiterate.

Concerning the participants' residences, 48 (73.8%) of those with HCKD lived in rural areas, and 43 (66.2%) of those with NCKD also lived in rural areas (table 1).

Table 1 Frequency distributions of the sociodemographic characteristics of the participants at public hospitals in Nekemte town, West Oromia, Ethiopia, 2022

Variables		HCKD, N (%) or mean±SD	NCKD, N (%) or mean±SD	P value
Age		53.83±13.18	55.17±18	0.628*
Sex	Male	32 (49.2%)	44 (67.7%)	0.033
	Female	33 (50.8%)	21 (32.3%)	
Educational status	Illiterate	25 (38.5%)	39 (60.0%)	0.003
	Primary school	5 (7.7%)	4 (6.2%)	
	Secondary school	21 (32.3%)	11 (16.9%)	
	College/university	9 (13.8%)	8 (12.3%)	
	Postgraduates	5 (7.7%)	3 (4.6%)	
Residence	Urban	17 (26.2%)	22 (33.8%)	0.339
	Rural	48 (73.8%)	43 (66.2%)	

*age is not statistically significant
*
HCKD, hypertensive chronic kidney disease; NCKD, normotensive chronic kidney disease; SD , standard deviation.

Circadian blood pressure variability

The CBPV (daytime and night-time SBP, daytime and night-time DBP, mean 24-hour SBP and DBP and SD of 24-hour SBP and DBP) of the HCKD patients and NCKD patients were compared using an independent sample t-test.

The mean 24-hour SBP was 145.61±10.11 mm Hg in HCKD patients and 112.84±8.6 mm Hg in NCKD patients. Among the HCKD and NCKD patients, the mean daytime and night-time SBP were 146.57±10.61 mm Hg versus 115.40±11.0 mm Hg and 144.40±12.11 mm Hg versus 115.40±11.0 mm Hg, respectively.

The 24-hour SD of each patient's SBP was calculated. The mean 24-hour SD of the SBP was significantly different between the HCKD and NCKD patients (10.17±6.12 mm Hg vs 5.4±2.7 mm Hg, p=0.043). However, the mean daytime SD of the SBP was not significantly different among the groups (p=0.599), while there was a significant difference in the mean night-time SD of the SBP between HCKD and NCKD patients (5.06±3.08 and 3.26±1.63, p<0.001, respectively).

The diastolic CBP among HCKD and NCKD patients were mean 24-hour DBP (93.0±8.05 vs 73.56±5.73), mean daytime DBP (96.21±8.28 vs 74.66±6.68) and mean night-time DBP (92.09±9.60 vs 72.61±8.06), respectively.

The mean night-time SD of DBP (5.04±2.0 vs 4.05±3.64) was significantly different (p=0.044) among HCKD and NCKD patients, while the mean daytime SD of DBP (5.03±3.09 vs 4.80±3.66) and mean 24-hour SD of DBP (6.75±2.5 vs 6.18±3.67) were not significantly different among the groups (p>0.05) (table 2).

Renal function test results and CKD stage

The mean serum creatinine levels of the HCKD and NCKD patients were 2.144±0.99 mg/dL and 1.33±0.68 mg/dL, respectively ($p=2.9\times10^{-7}$). The mean eGFRs of the HCKD and NCKD patients were 41.25±21.45 mL/min/1.73 m^2 and 69.65±30.94 mL/min/1.73 m^2, respectively. This showed a significant difference in renal function between HCKD and NCKD patients.

The current study compared the decline in kidney function between dippers and non-dippers in terms of SBP in both groups. In the HCKD patients, the mean eGFR (38.39±21.13 mL/min/1.73 m^2) in the non-dippers was significantly lower than that in the dippers (55.27±17.85 mL/min/1.73m^2), (p=0.014). In NCKD patients, the difference in the mean eGFR between non-dippers (67.27±36.65 mL/min/1.73m^2) and dippers (73.71±17.35 mL/min/1.73m^2) was not statistically significant (p=0.345).

Current findings indicate that HCKD patients with a lower eGFR (<60 mL/min/1.73 m^2) are three times more likely to be non-dippers than are those with a higher eGFR (>60 mL/min/1.73 m^2) (adjusted odd ratio (AOR) =2.92, 95% CI (1.21 to 47.06), p=0.038).

The majority of HCKD patients were diagnosed with CKD stage 4 ([25]38.5%), followed by CKD stage 2 ([18]27.7%) and only two (3.1%) were diagnosed with CKD stage 5. However, no HCKD patients were diagnosed with stage 1 CKD. Less than half (41.5%) of the NCKD patients were diagnosed with CKD stage 2, followed by CKD stage 3a ([16]24.6%), CKD stage 1 ([10]15.4%), CKD stage 4 ([8]12.3%) and CKD stage 3b ([4]6.2%), and no NCKD patients were diagnosed with CKD stage 5 (table 3).

Prevalence of dipping patterns among the participants

The present study revealed a significantly greater incidence of non-dippers in SBP among HCKD patients than among NCKD patients (83% (73.5%, 91%) versus 63% (50.7%, 74.6%, p=0.010)). Dippers were found in 11 (17% (8%, 26.5%)) HCKD patients and 24 (37% (25.4%, 49.3%)) NCKD patients.

When the SBP dipping pattern was further subdivided into dipping patterns in HCKD patients, 48% (37%,

Table 2 Comparisons of circadian blood pressure variability among HCKD patients and NCKD patients in public hospitals in Nekemte town, West Oromia, Ethiopia, 2022

Variables	HCKD mean±SD	NCKD mean±SD	t value	95% CI	P value
Mean 24-hour SBP (mm Hg)	145.61±10.11	112.84±8.6	19.910	(29.51 to 36.03)	<0.01*
Mean daytime SBP	146.57±10.61	115.40±11.0	16.49	(27.46 to 34.95)	<0.01*
Mean night-time SBP	144.40±12.11	109.28±9.0	18.732	(31.37 to 38.79)	<0.01*
Mean daytime SD of SBP	4.01±2.27	3.802±2.30	0.527	(–0.58 to 1.004)	0.599
Mean night-time SD of SBP	5.06±3.08	3.26±1.63	4.158	(0.94 to 2.66)	<0.01*
Mean 24-hour SD of SBP	10.17±6.12	5.4±2.7	2.020	(0.02 to 1.77)	0.043*
Mean 24-hour DBP	93.0±8.05	73.56±5.73	15.865	(17.01 to 21.87)	<0.01*
Mean daytime DBP	96.21±8.28	74.66±6.68	16.324	(18.94 to 24.16)	<0.01*
Mean night-time DBP	92.09±9.60	72.61±8.06	12.536	(16.41 to 22.56)	<0.01*
Mean daytime SD of DBP	5.03±3.09	4.80±3.66	0.411	(–0.93 to 1.42)	0.682
Mean night-time SD of DBP	5.04±1.94	4.05±3.64	1.920	(0.03 to 2.01)	0.044*
Mean 24-hour SD of DBP	6.75±2.5	6.18±3.67	1.027	(–0.53 to 1.66)	0.306

*Statistically significant at 95% CI with p value <0.05.
CKD, chronic kidney disease; HCKD, hypertensive chronic kidney disease; NCKD, normotensive chronic kidney disease; SBP, systolic blood pressure.

59.4%) were risers, 35% (24%, 47%) were non-dippers and 17% (8%, 26.5%) were dippers. Among NCKD patients, 25% (14.88%, 35%) were risers, 38% (27.33%, 50%) were non-dippers and 37% (25.4%, 49.3%) were dippers. No extreme dippers were discovered in either group in this study.

Concerning DBP dipping status, 53 (81.5% (66.7%, 87%)) patients with HCKD were non-dippers, 12 (18.5% (13%, 33.3%)) were dippers and 39 (60% (48.4%, 72%)) were non-dippers, while 26 (40% (28%, 51.6%)) were dippers (p=0.007). Further categorisation of the DBP dipping profile revealed 25 (38.5%) risers, 28 (43%) non-dippers and 12 (18.5%) dippers in HCKD patients and 14 (21.5% (12%, 31.7%)) risers, 25 (38.5% (26.2%, 50.7%)) non-dippers and 26 (40% (28%, 52%)) dippers in NCKD patients (p=0.015) (figure 1).

Table 3 Comparisons of renal function status and CKD stages among HCKD patients and NCKD patients in Nekemte town public hospitals, West Oromia, Ethiopia, 2022

Variables		HCKD mean±SD	NCKD mean±SD	P value
Serum creatinine (mg/dl)		2.144±0.99	1.33±0.68	2.9×10^{-7}*
eGFR (mL/min/1.73 m^2)		41.25±21.45	69.65±30.94	1.6×10^{-8} *
	Non-dipper	38.39±21.13		0.014*
	Dipper	55.27±17.85		
	Non-dipper		67.27±36.65	0.345
	Dipper		73.71±17.35	
CKD stages (%)	1	-	10 (15.4%)	<0.01*
	2	18 (27.7%)	27 (41.5%)	
	3a	6 (9.2%)	16 (24.6%)	
	3b	14 (21.5%)	4 (6.2%)	
	4	25 (38.5%)	8 (12.3%)	
	5	2 (3.1%)	-	

*Statistically significant at 95% CI with p value <0.05.
CKD, chronic kidney disease; eGFR, estimated glomerular filtration rate; HCKD, hypertensive chronic kidney disease; NCKD, normotensive chronic kidney disease.

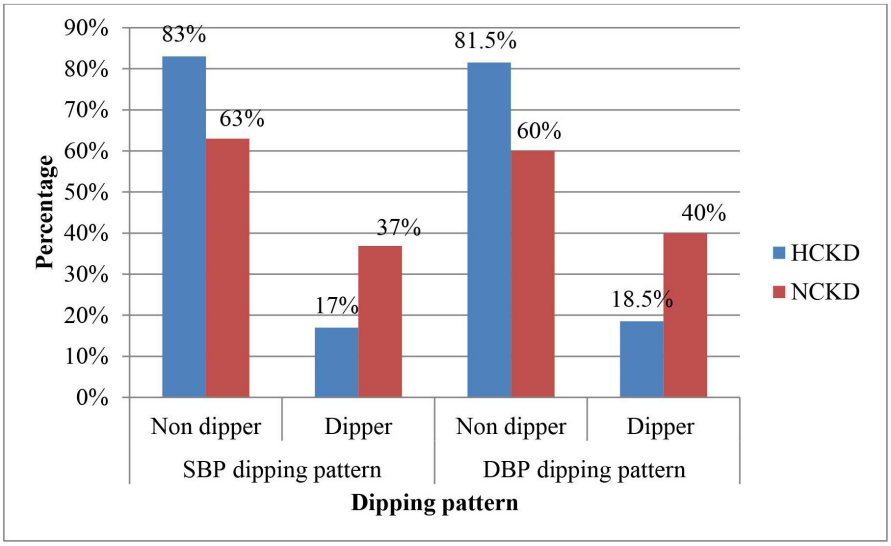

Figure 1 Circadian systolic blood pressure (SBP) dipping pattern among HCKD and NCKD patients in Nekemte town public hospitals, West Oromia, Ethiopia, 2022. HCKD, hypertensive chronic kidney disease; NCKD, normotensive chronic kidney disease.

Factors associated with the non-dipping pattern of CBP among HCKD patients

Bivariate analysis was performed separately for SBP and DBP non-dipping patterns. The variables associated with the SBP non-dipping pattern were the mean 24-hour SBP, the mean 24-hour SD of SBP and the eGFR. Only the eGFR (AOR=2.92, 95% CI (1.21 to 47.06), p=0.038) and mean 24-hour SBP (AOR=1.50, 95% CI (1.15 to 1.96), p=0.003) were variables independently associated with non-dipping SBP in HCKD patients according to multivariate analysis.

The mean 24-hour SD of DBP and eGFR were variables associated with non-dipping patterns in DBP. According to the multivariate analysis for DBP non-dipping, only the eGFR (AOR 2.8 (1.6, 30.24), p=0.022) was independently associated with non-dipping DBP in HCKD patients (table 4).

DISCUSSION

In CKD patients, where HTN is common, controlled HTN is particularly pertinent. CKD patients are more likely to progress to an advanced stage of the disease as a result of abnormal CBPV. To control HTN in CKD patients, a circadian-based approach to BP measurements has become a prominent research area in recent years. The current study compared CBPV between HCKD and NCKD patients.

The mean night-time SD of SBP (5.06±3.08 mm Hg vs 3.26±1.63 mm Hg, p=0.001) and the mean night-time SD

Table 4 Factors associated with the non-dipping pattern of circadian BP (CBP) among HCKD patients in public hospitals in Nekemte town, West Oromia, Ethiopia, 2022

Variables		SBP dipping pattern		COR (95% CI)	AOR (95% CI)	P value
		Non-dipper	Dipper			
eGFR	<60 mL/min/1.73 m^2	47(87)	4 (36.4)	0.09 (0.02 to 0.37)	2.92 (1.21 to 27.06)	0.038**
	≥60 mL/min/1.73 m^2	7 (13)	7 (63.6)	1	1	
Mean 24-hour SBP	Mean±SD	145.61±6.7 mm Hg		1.34 (1.12 to 1.6)	1.50 (1.15 to 1.96)	0.003**
SD 24-hour SBP	Mean±SD	10.11±2.53 mm Hg		0.8 (0.61 to 1.04)	0.82 (0.52 to 1.29)	0.94
		DBP dipping pattern				
		Non-dipper	Dipper			
SD 24- hour DBP	Mean±SD	6.75±2.5 mm Hg		0.44 (0.28 to 0.67)	0.87 (0.704 to 1.08)	0.208
eGFR	<60 mL/min/1.73 m^2	40 (75.5)	5 (41.7)	4.31 (1.17 to 15.92)	2.8 (1.6 to 30.24)	0.022**
	≥60 mL/min/1.73 m^2	13 (24.5)	7 (58.3)		1	

*Statistically significant at 95% CI with p value <0.05; 1-reference; mean 24-hour SBP—mean 24-hour systolic blood pressure.
AOR, adjusted OR; COR, crude OR; DBP, diastolic blood pressure; HCKD, hypertensive chronic kidney disease.

of DBP (5.15±1.94 mm Hg vs 4.05±3.64 mm Hg, p=0.044) were significantly different between the HCKD and NCKD patients. However, neither the mean daytime SD of SBP (4.01±2.27 vs 3.802±2.30, p=0.599) nor the mean daytime SD of DBP (96.21±8.28 vs 74.66±6.68, p=0.682) was significantly different. This may be due to physical activity and environmental changes that affect short-term BPV, which are less frequent at night than during the day. So night-time BPV reflects endogenous influences rather than external factors. According to previous studies, short-term overnight BPV was better at predicting renal structural changes than short-term daylight BPV,[36] and a reduced magnitude of the diastolic dip was related to the deterioration of kidney functions.[37] The magnitude of BPV was lower than that reported in studies conducted in Spain and Italy, which showed that HCKD patients had higher night-time SDs of SBP and SDs of DBP (12.1±4.1 mm Hg vs 9.2±2.9 mm Hg) than did their control group.[38 39] This discrepancy may be explained by variations in study design, sample size and the clinical environment in which CBP was measured.

The mean 24-hour SD of the SBP was significantly greater in HCKD patients (10.17±6.12 mm Hg) than in NCKD patients (5.4±2.7 mm Hg) (p=0.043). This result was in line with the conclusion of an earlier study in which the 24-hour SD of SBP was 10.2±2.5 mm Hg[38] in HCKD patients. However, this was higher than that in a previous study in which the ARV(average real variability) of SBP was 9.2±0.2 mm Hg[40] and lower than that in a previous study in which the ARV of SBP was 15.9±4.63 mm Hg in HCKD patients.[19] This disparity could be attributed to differences in sample size, study methodology or the method employed to explain the CBPV index. Previous investigations used the ARV. However, in this study, the mean 24-hour SD was used.

This study revealed that HCKD patients were more likely to be non-dippers than NCKD patients (83% vs 63%, p=0.010, respectively) according to SBP. This result was consistent with the findings of a study performed in Morocco, in which 80% of patients with HCKD did not dip[17] more prevalently, 42.3% of the HCKD patients and 40.4% of the NCKD patients were non-dippers,[41] and 34% of the normotensive individuals in India were non-dippers.[42] This was lower than that reported in a study performed in Nigeria,[18] where 92% of HCKD patients were non-dippers. Further comparison of the dipping profile subsets revealed that the prevalence of increased SBP and DBP was 48% and 21.5%, respectively, in the HCKD group. This was more prevalent than in a previous study in which the prevalence was 17% in HCKD patients[9] and 35.3% in NCKD patients.[29] A reversed dipper was strongly related to severe renal damage[43] and was a predictor of mortality[44] in CKD patients compared with non-dipper patients.

The current study compared the decline in kidney function between dippers and non-dippers in terms of SBP in both groups. Among HCKD patients, the mean eGFR (38.39±21.13 mL/min/1.73 m^2) in non-dippers was significantly lower than in dippers (55.27±17.85 mL/min/1.73 m^2), (p=0.014). However, among the NCKD patients, the difference in the mean eGFR between non-dippers (67.27±36.65 mL/min/1.73 m^2) and dippers (73.71±17.35 mL/min/1.73 m^2) was not statistically significant (p=0.345). This finding was consistent with that of a prior study that showed that patients with nocturnal HTN had a worse prognosis than those with nocturnal normotension (p<0.05).[45] Additionally, there was no significant difference in the rate of eGFR decrease between people with and without dips in BP.[41] The incidence of renal composite outcomes was greater in hypertensive non-dipper patients than in hypertensive dippers. However, these parameters were similar between normotensive dipper and non-dipper patients.[22] However, in contrast to the findings of previous studies, CKD decline (eGFR <60 mL/min/1.73 m^2) was not significantly different between dippers and non-dippers in either the normotensive (p=0.69) or hypertensive (p=0.31) groups.[46] This disparity could be attributed to the study's design, the participants' age and their clinical status.

These findings showed that non-dippers were related to the progression of CKD. In a prior study, it was discovered that antihypertensives taken before bedtime by CKD patients reduced both the average nocturnal BP and the percentage of non-dippers.[47] Clinical evidence has shown that antihypertensive medications that restore BP and circadian rhythm lower the incidence of end-stage renal disease.[48]

According to multivariate logistic regression, the mean 24-hour SBP was independently associated with non-dipping SBP (AOR=1.50, 95% CI (1.15 to 1.96), p=0.003). In a previous study,[40] higher mean 24-hour SBP was linked to BP variability (p<0.05). This is because higher mean BP causes consequences such as arterial stiffness and increased BP variability.[16]

For both SBP and DBP, an eGFR <60 mL/min/1.73 m^2 was independently associated with non-dipping BP patterns (AOR=2.92, 95% CI (1.21 to 17.06), p=0.038 and AOR=2.81, 95% CI (1.6 to 30.24), p=0.022). This result was in line with a previous study revealing that individuals with a lower eGFR (<60 mL/min/1.73 m^2) were more likely to be non-dippers than those with a higher eGFR (>60 mL/min/1.73 m^2) (95% CI 3.10 (1.50 to 6.43), p=0.018).[49] The proposed mechanism states that increased sodium consumption and a deficiency in the kidney's ability to excrete salt lead to increased (non-dipper) BP at night. This process took place to compensate for diminished natriuresis throughout the day and increased pressure natriuresis at night.[50]

Some limitations should be considered when interpreting the results of this study. The study participants were hospitalised patients with limited physical activities and continuous treatment. Therefore, physical activity and medication restriction were not considered in this finding. Despite these limitations, this study has several strengths. For instance, BP was measured using a standard automated BP apparatus. In addition, BP was taken under

strict adherence to nationally and globally recognised protocols.

CONCLUSION AND RECOMMENDATIONS

Conclusion

Compared with NCKD patients, HCKD patients exhibited considerably greater CBPV. 83% of HCKD patients had non-dipper SBP. Furthermore, less than half of the HCKD patients were risers, which was the greatest CBP aberration. Reduced kidney function and elevated average 24-hour BP were factors associated with non-dipping BP.

Recommendations

Based on the findings, the following recommendations have been made for the respective stakeholders. For healthcare workers working in hospitals, BP should be measured both during the day and night for HCKD patients to reduce nocturnal BP complications. Federal Minister of Health and Oromia Health Bureau should develop CBP measurement guidelines for HTN diagnosis and management. For researchers, A larger-scale, longer-term investigation is needed to determine the influence of non-dipping BP on hypertensive and normotensive chronic renal disease patients.

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Ethics approval The Jimma University institutional review board approved the study(Ref. No. 77/22), which was carried out as the Declaration of Helsinki. The data collectors were trained and were familiar with the community's culture and language. Information about the purpose, procedure, duration and patient rights was explained by the data collectors to the participants by reading the Afaan Oromoo and Amharic translated information sheets. Blood samples for serum creatinine determination were drawn by trained medical laboratory professionals. Study participants were not subjected to major discomfort, and only a 2 mL blood sample was taken. To ensure data confidentiality, participants' identities, personal information and responses were not disclosed to third parties. Participants were notified that the information was confidential and that their identities would not be recorded on the questionnaire. Participants were notified of their right to join or withdraw from the event. Each participant provided oral consent.

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