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Report

Nocturnal high blood pressure and left ventricular hypertrophy in patients with chronic kidney disease

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ABSTRACT

Background: Patients diagnosed with chronic kidney disease (CKD) have a heightened risk of developing masked uncontrolled hypertension (MUCH), leading to hypertension-induced organ damage. This study aimed to estimate the prevalence and characteristics of MUCH and to investigate risk factors of left ventricular hypertrophy (LVH) in those with CKD. **Methods**: A retrospective study was conducted on data from 178 patients diagnosed with CKD and having controlled office blood pressure at Nhan Dan Gia Dinh Hospital between October 2018 and June 2019. These participants underwent 24-hour ambulatory blood pressure monitoring (ABPM) using the SunTech Oscar 2 device. Subsequently, echocardiography was performed to assess for the presence of LVH. **Results**: The prevalence of MUCH was 48.9%. Notably, all patients with MUCH demonstrated elevated nighttime blood pressure. LVH was more prevalent in the MUCH group when compared to those with controlled hypertension (55.2% and 38.5%, respectively). MUCH and CKD staging 4-5 were independent risk factors of LVH with ORs 1.97 (95% CI, 1.03-3.85) and 2.58 (95% CI, 1.16-5.94), respectively. **Conclusions**: We recommend routinely using ABPM to detect MUCH in CKD patients even with controlled office hypertension. Screening for LVH is necessary in those with MUCH.

Key words: Masked uncontrolled hypertension, chronic kidney disease, ambulatory blood pressure monitoring, left ventricular hypertrophy

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INTRODUCTION

Ambulatory blood pressure monitoring (ABPM) provides valuable data on mean 24-hour, daytime, and nocturnal blood pressure (BP). Patients with masked uncontrolled hypertension (MUCH) exhibit controlled office BP but elevated out-of-office BP. Similar to sustained hypertension, MUCH increases the risk of cardiovascular events and mortality compared to controlled BP[1,2]. Notably, MUCH in CKD patients contributes to the progression of left ventricular hypertrophy (LVH) and end-stage renal disease (ESRD), but there are few related studies on the Vietnamese CKD population[3–5]. Therefore, this study aimed to estimate the prevalence and characteristics of MUCH and to examine the risk factors associated with left ventricular hypertrophy (LVH) in individuals with chronic kidney disease (CKD).

METHOD

Study design and participants

We revisited data from 196 patients with CKD and controlled office BP undergoing 24-hour ABPM at Nhan Dan Gia Dinh Hospital from October 2018 to June 2019 with the following study criteria as follows:

- Inclusion criteria were (1) age $\geq$ 18 years and consented, (2) an estimated glomerular filtration rate (eGFR) by the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI, 2009) equation < 60 mL/min per 1.73 m^2, measured on two occasions within 3 months[6], and (3) controlled office blood pressure with systolic BP 120-139 mmHg and diastolic BP 80-89 mmHg[7].
- Exclusion criteria included patients with acute illness, pregnancy, acute glomerulonephritis, systemic lupus erythematosus, as well as those with ESRD receiving renal replacement therapy.

We excluded 4 patients since they stopped engaging in the study and 14 patients who had inadequate ABPM recordings, thus yielding 178 subjects completing the investigation. The recruitment flow is illustrated in **Figure 1**.

Data collection

Office BP readings were taken from the Yamasu sphygmomanometer, Japan, by trained nurses at clinics. Three sitting readings were recorded in a row, and office BP was calculated as the average of the last two measurements. Ambulatory blood pressure in 24

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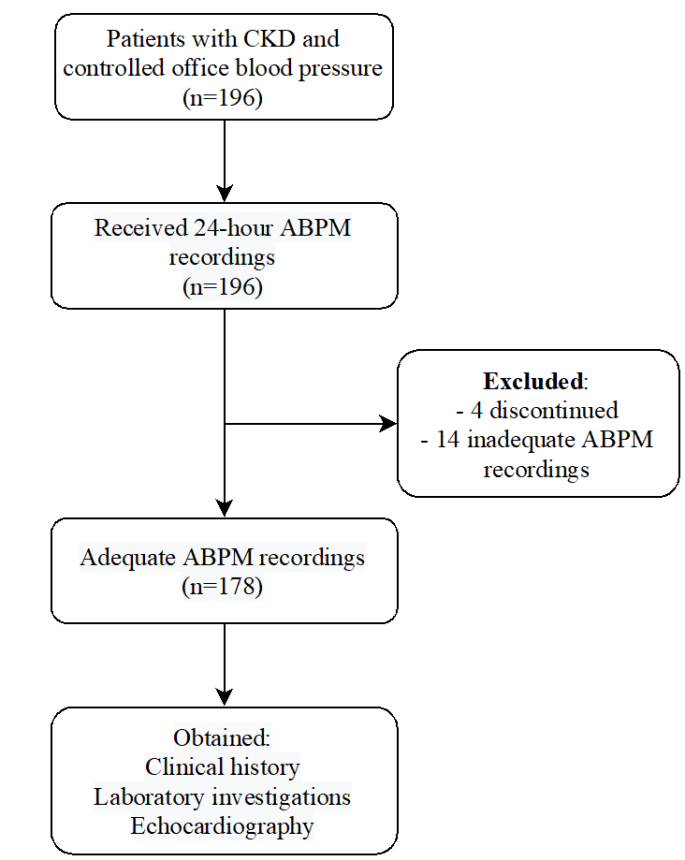

Figure 1: **Recruitment flow**. **Abbreviations**: **CKD**: chronic kidney disease, **ABPM**: ambulatory blood pressure monitoring

hours was measured by the American SunTech Oscar 2 device. The size of the cuff was selected according to the patient's arm circumference. AccuWinPro version 3.0 software was used to process the results. The daytime and nighttime measuring intervals were set to be every 30 minutes and 60 minutes, respectively. BP readings were not displayed on the meter screen. Patients with acceptable BP measurements greater than 70% of total measurements were considered to have adequate ABPM assessments. Controlled office BP was defined as having systolic BP < 140 mmHg and diastolic BP < 90 mmHg at clinics. We classified patients into 2 groups based on MUCH and controlled hypertension (CH). MUCH was defined as having controlled office BP and 24-hour ambulatory BP $\geq$ 130/80 mmHg and/or daytime BP $\geq$ 135/85 mmHg and/or nighttime BP $\geq$ 120/70 mmHg. Otherwise, patients were in the CH group[7]. Left ventricular mass (LVM) was assessed by echocardiography. The procedure was performed by a certified cardiologist using an American Philips Affiniti 50G ultrasound machine. LVM (g) was calculated by the equation 0.832 x [(IVSd + LVEDd + PWTd)^3 – LVEDd^3] + 0.6 (g) in which LVEDd: LV end-diastolic dimension (mm), IVSd: interventricular septal thickness at end-diastole (mm), PWTd: posterior wall thickness at end-diastole (mm). LVM index was expressed as LVMI = LVM/Body surface area (g/m^2) in which body surface area = 0.007184 x W°.^{25} x H°.^{25} (m^2) (W = Weight, H = Height). LVH was defined as LVMI $\geq$ 115 g/m^2 in men and LVMI $\geq$ 95 g/m^2 in women[8]. Serum creatinine and other laboratory tests were measured at Nhan Dan Gia Dinh Hospital. Quality control for laboratory testing complied with the regulations of the Vietnamese Ministry of Health. Grading of CKD was based on eGFR[6].

Statistical analyses

Continuous variables with normal distribution were displayed as the means ± standard deviation (SD). Continuous variables with skewed distributions were presented as the medians (M25–M75). Shapiro–Wilk

test of normality was used to determine the normal distribution of data. Categorical variables were presented by percentage (%) and compared using the Chi-squared test or Fisher's exact test when appropriate. For continuous variables, we compared the average values of the two groups by Student's t-test. In the case of a non-normal distribution, a Mann–Whitney non-parametric test was utilized. Univariate and multivariate logistic regression analyses were conducted to ascertain risk factors of LVH. Multivariate logistic regression analysis was done using confounders that were significant in the univariate model. Univariate and multivariate logistic regression analyses examined 1-SD changes in continuous variables. The presence of dichotomous variables was coded as 1 and the absence as 0. All probabilities were expressed as 2-tailed, with statistical significance inferred at $P < 0.05$. All confidence intervals were computed at the 95% level. Statistical analysis was done using STATA 20.0 (StataCorp, College Station, TX, USA).

RESULTS

A total of 178 patients with CKD and controlled office BP completed the study. After assessing 24-hour ABPM, 87 patients had MUCH (48.9%) and 91 patients were in the CH group.

Baseline characteristics

Compared with patients with CH, those having MUCH had longer hypertension duration at baseline. There was no significant difference in CKD stages between the two groups. Notably, LVH was more prevalent in the MUCH group. All laboratory results did not have a statistically significant difference in the two groups (**Table 1**).

Characteristics of MUCH in CKD patients

The difference between office and ambulatory BP in the two groups was statistically significant, with the BP values being higher in the MUCH group (**Table 2**). The rate of MUCH on 24-hour BP, daytime BP, and nighttime BP were 21.9%, 11.8%, and 48.9%, respectively, and based on all three criteria above was 48.9% (**Table 1**).

Univariate and multivariate logistic regression analyses

As shown in univariate analysis, CKD stage 4-5 and MUCH were associated with LVH (OR = 2.64, 95% CI: 1.29 – 5.61, P = 0.009 and OR = 1.97, 95% CI: 1.09 – 3.60, P = 0.026, respectively) (Table 3). Both factors remained statistically significant when included in multivariate analysis. Patients with CKD stage 4-5 had a higher risk of LVH (OR = 2.58; 95% CI: 1.16 – 5.94, P = 0.036) while MUCH patients were at increased risk of LVH two times higher (OR = 1.97; 95% CI: 1.03 – 3.85, P = 0.026) (**Table 3**).

DISCUSSION

We used daytime and/or nighttime and/or 24-hour BP values to define MUCH and confirmed the rate was 48.9%. Other studies demonstrated that the prevalence of MUCH in CKD patients ranges from 45.0 to 70.0% depending on diagnostic criteria[3,5,9]. All patients participating in this study had elevated nocturnal BP while only 11.8% had high daytime BP. This finding suggested that the routine use of office BP measurement and even home BP monitoring had a limited role in detecting MUCH in patients with CKD. Though 24-hour ABPM does not provide day-to-day BP variations, this is a favorable approach in clinical settings for BP evaluation.

The precise mechanism by which CKD elevates nocturnal BP involves complex processes, including volume-dependent hypertension exacerbated by serum sodium dysregulation, comorbidities like diabetes mellitus, and autonomic nervous dysfunction[10–12]. The dysfunction of endothelial cells lowers nitric oxide production causing suppression of the sympathetic nervous system[13]. Nocturnal hypertension is common in individuals with CKD due to disruptions in the body's circadian rhythm as well. Fukuda et al. suggested that decreased renal function reduces sodium excretion during the day and nighttime BP then rises to increase sodium clearance[14].

Our results are similar to the AASKD report, in which participants with masked hypertension had higher LVH proportions than those with normal BP and white-coat hypertension[3]. In the CRIC study, masked hypertension was associated with increased LVMI and pulse wave velocity compared to those with truly controlled BP[4]. LVH in individuals with CKD results from increased volume and pressure loads. Factors such as activation of the RAAS, inhibition of nitric oxide synthesis, intravascular volume expansion, secondary anemia, and arteriovenous fistulas contribute to myocardial cell elongation and the development of eccentric or asymmetric LVH, potentially progressing to LV fibrosis[15]. Clinical evidence underscores the association between LVH, myocardial fibrosis, and heightened mortality risk, along with increased cardiovascular events in CKD and ESRD, as evidenced by elevated rates of sudden cardiac death[15]. Regression of left ventricular mass

Table 1: Baseline characteristics according to BP groups

	All (N = 178)	MUCH (N = 87)	CH (N = 91)	P-value
Age (year)	68.2 ± 8.8	67.5 ± 9.2	68.8 ± 8.4	0.558
Male	92 (51.7)	41 (47.1)	51 (56.0)	0.294
BMI (kg/m^2)	24.4 ± 3.0	24 ± 3.2	24.7 ± 2.8	0.079
Current smoker	24 (13.5)	14 (16.1)	10 (11.0)	0.627
CKD staging				
G3a G3b G4 G5	53 (29.8) 85 (47.8) 33 (18.5) 7 (3.9)	23 (26.4) 42 (48.3) 18 (20.7) 4 (4.6)	30 (33.0) 43 (47.3) 15 (16.5) 3 (3.3)	0.738
Diabetes mellitus	114 (64.0)	55 (63.2)	59 (64.8)	0.876
Dyslipidemia	162 (91.0)	78 (89.7)	84 (92.3)	0.606
Stroke	13 (7.3)	6 (6.9)	7 (7.7)	0.999
Heart failure	9 (5.1)	5 (5.7)	4 (4.4)	0.743
Ischemic heart disease	16 (9.0)	7 (7.9)	9 (4.4)	0.704
HTN duration (year)	9.5 ± 7.2	10.4 ± 7.1	8.5 ± 7.2	0.015
CKD duration (year)	2.9 ± 2.9	3.1 ± 3.2	2.7 ± 2.4	0.953
Serum creatinine (mmol/L)	138.4 (121.8-167.3)	139.9 (129.1-172.6)	134.6 (120.7-162.1)	0.168
eGFR (ml/min/1.73 m^2)	37.4 ± 11.4	36.1 ± 12.1	38.7 ± 10.6	0.113
Cholesterol (mg/dL)	170.0 ± 28.8	174.8 ± 59.3	165.3 ± 53.1	0.184
Triglyceride (mg/dL)	171.7 (127.4-243.7)	166.1 (128.7-252.2)	172.2 (127.0-230.9)	0.795
HDL-cholesterol (mg/dL)	43.2 (37.3-48.2)	43.6 (37.9-50.9)	42.4 (36.9-47.1)	0.280
LDL-cholesterol (mg/dL)	82.8 (57.2-117.6)	86.5 (59.2-116.4)	80.7 (54.7-117.0)	0.478
LVEF (%)	68.9 ± 7.3	68.4 ± 8.4	69.3 ± 6.1	0.710
LVMI (g/m^2)	106.5 ± 28.8	110.4 ± 29.3	102.6 ± 27.8	0.095
LVH	83 (46.6)	48 (55.2)	35 (38.5)	0.025
ACE inhibitors	30 (16.8)	12 (13.8)	18 (19.8)	0.286
ARBs	97 (54.5)	49 (56.3)	48 (52.8)	0.632
Thiazides	22 (12.3)	9 (10.4)	13 (14.3)	0.425
Loop diuretics	39 (21.9)	20 (23.0)	19 (20.9)	0.734
CCBs	122 (68.6)	61 (70.1)	61 (67.0)	0.658
Beta blockers	88 (49.4)	43 (49.4)	45 (49.5)	0.997

Abbreviations: **BMI**: bodymass index, **HTN**: hypertension, **CKD**: chronic kidney disease, **eGFR**: estimated glomerular filtration rate, **HDL**: high density lipoprotein, **LDL**: low density lipoprotein, **LVEF**: left ventricular ejection fraction, **LVMI**: left ventricular mass index, **LVH**: left ventricular hypertrophy, **ACE**: angiotensin-converting enzyme, **ARB**: angiotensin receptorblocker, **CCB**: calciumchannel blocker

Table 2: Blood pressure characteristics according to BP groups

Blood pressure (mmHg)	All (n = 178)	MUCH (n = 87)	CH (n = 91)	P-value
Office SBP	128.6 ± 6.2	130.4 ± 5.9	126.8 ± 6	< 0.001
Office DBP	73.9 ± 7.0	75.6 ± 6.7	72.3 ± 7.0	0.002
24-hour	118.7 ± 12.0	128.1 ± 8.1	109.6 ± 7.3	< 0.001
24-hour DBP	66.7 ± 7.9	70.6 ± 7.9	63.0 ± 5.8	< 0.001
SBP day	120.1 ± 11.5	128.3 ± 8.5	112.2 ± 8.0	< 0.001
DBP day	67.8 ± 8.0	71.1 ± 8.2	64.7 ± 6.5	< 0.001
SBP night	116.0 ± 14.7	128.0 ± 9.3	104.5 ± 8.3	< 0.001
DBP night	64.9 ± 9.0	70.3 ± 8.6	59.8 ± 5.9	< 0.001

Abbreviations: **BP**: blood pressure, **SBP**: systolic blood pressure, **DBP**: diastolic blood pressure, **CH**: controlled hypertension, **MUCH**: masked uncontrolled hypertension

Table 3: Univariate and multivariate regression analyses for factors associated with LVH in patients with CKD

	Univariate			Multivariate		
	OR	CI 95%	P-value	OR	CI 95%	P-value
Age	1.16	0.48 – 2.86	0.746	–	–	–
Male	0.58	0.32 – 1.06	0.077	–	–	–
Current smoker	1.34	0.72 – 2.51	0.359	–	–	–
Diabetes mellitus	1.32	0.72 – 2.47	0.374	–	–	–
HTN duration ≥ 10 years	1.16	0.64 – 2.11	0.622	–	–	–
RAAS inhibitors	0.70	-1.00 – 0.29	0.286	–	–	–
Other HTN drugs	1.88	-0.33 – 1.589	0.199	–	–	–
CKD staging 4-5	2.64	1.29 – 5.61	0.009	2.58	1.16 – 5.94	0.036
MUCH	1.97	1.09 – 3.60	0.026	1.97	1.03 – 3.85	0.026

Abbreviations: **LVH**: left ventricular hypertrophy, **HTN**: hypertension, **RAAS**: renin-angiotensin-aldosterone system, **CKD**: chronic kidney disease, **MUCH**: masked uncontrolled hypertention

could serve as a valuable surrogate marker for assessing the benefits of RAAS inhibition aimed at reducing mortality risk in hypertensive patients [16]. Similar results were observed in the CKD population with the roles of proper hemoglobin targets and hemodialysis regimens in addition to RAAS inhibition [17].

A remarkable insight of our study is the use of 24-hour ambulatory BP for diagnosing out-of-office hypertension and assessing the BP patterns of patients with CKD. The prevalence of MUCH in the CKD population was considerable. Moreover, our findings support that CKD patients with MUCH have a higher risk of left ventricular hypertrophy than those with CH. Further studies are needed to define this relationship clearly and whether targeting MUCH patients can reduce the adverse outcomes.

There are several limitations of our study. First, due to limited resources, we did not assess sleep quality and other essential factors, including lifestyle choices and socioeconomic status, which have been proven to affect nocturnal BP, CKD, and LVH and their relationships [18]. Second, the study did not include those with structural CKD stages 1 and 2. Third, the retrospective design might introduce selection and information biases, as data were obtained from medical records. Fourth, while a sample size of 178 patients is adequate, a larger cohort that includes regional variations in patient demographics could yield more robust data. Fifth, the accuracy measurements may be influenced by equipment limitations regarding ABPM devices and variations in operator technique during the heart ultrasound. Future research should over-

come these limitations by incorporating multicenter designs, recruiting larger and more diverse populations, and conducting longitudinal follow-ups to validate these findings.

CONCLUSIONS

We recommend routinely using ABPM, which is sustainable in developing countries like Vietnam, to detect MUCH, especially nocturnal high blood pressure in CKD patients even with controlled office hypertension. Screening for LVH is necessary in those with MUCH.

ABBREVIATIONS

ABPM: Ambulatory Blood Pressure Monitoring, **ACE**: Angiotensin-Converting Enzyme, **ARB**: Angiotensin Receptor Blocker, **BMI**: Body Mass Index, **BP**: Blood Pressure, **CCB**: Calcium Channel Blocker, **CH**: Controlled Hypertension, **CI**: Confidence Interval, **CKD**: Chronic Kidney Disease, **CKD-EPI**: Chronic Kidney Disease Epidemiology Collaboration, **CRIC**: Chronic Renal Insufficiency Cohort Study, **DBP**: Diastolic Blood Pressure, **eGFR**: Estimated Glomerular Filtration Rate, **ESRD**: End-Stage Renal Disease, **HDL**: High-Density Lipoprotein, **HTN**: Hypertension, **LDL**: Low-Density Lipoprotein, **LVH**: Left Ventricular Hypertrophy, **LVEF**: Left Ventricular Ejection Fraction, **LVM**: Left Ventricular Mass, **LVMI**: Left Ventricular Mass Index, **MUCH**: Masked Uncontrolled Hypertension, **OR**: Odds Ratio, **RAAS**: Renin-Angiotensin-Aldosterone System, **SBP**: Systolic Blood Pressure, **SD**: Standard Deviation

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AUTHOR'S CONTRIBUTIONS

All authors equally contributed to this work, read and approved the final manuscript.

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None.

AVAILABILITY OF DATA AND MATERIALS

Data and materials used and/or analyzed during the current study are available from the corresponding author on reasonable request.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was approved by the ethics committee of the University of Medicine and Pharmacy at Ho Chi Minh City (No. 373/IRD/UMP, October 25, 2018) and written informed consent was obtained from all patients prior to enrollment. The investigation conformed to the principles outlined in the 1975 Declaration of Helsinki.

CONSENT FOR PUBLICATION

Not applicable.

COMPETING INTERESTS

The authors declare that they have no competing interests.

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