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Clinical and Demographic Characteristics of Dipper and Non-Dipper Hypertensive Patients at Moderate Altitude

Orta Rakımda Yaşayan Dipper ve Dipper Olmayan Hipertansif Hastaların Klinik ve Demografik Özellikleri

ABSTRACT

Objective: Hypertension (HT) is a complex clinical syndrome influenced by lifestyle, genetics, mental stress, and environmental factors. High-altitude (H-ALT) is one environmental factor that affects the development of HT. This study aimed to analyze the clinical and demographic features of patients residing at moderate altitude (M-ALT).

Method: A total of 515 patients with hypertension, confirmed through 24-hour ambulatory blood pressure monitoring (ABPM), were screened. After exclusions, the final study population consisted of 452 patients. Participants were divided into two groups: dippers and non-dippers.

Results: Patients in the non-dipper group were older (standard deviation [SD]: 61 ± 13 vs. 54 ± 12 years, P < 0.001) and had a significantly higher waist circumference (SD: 104 ± 13 vs. 107 ± 12 cm, P = 0.022). However, patients in the dipper group had a significantly higher daytime mean systolic blood pressure (SBP) (SD: 144 ± 16 vs. 141 ± 17 mmHg, P = 0.027) and daytime mean diastolic blood pressure (DBP) (SD: 89 ± 12 vs. 87 ± 13 mmHg, P = 0.037). Patients in the non-dipper group also had a significantly higher proportion of abnormal left ventricular global longitudinal strain (LVGLS) (n = 218 (65.6%) vs. n = 53 (46.7%), P = 0.009).

Conclusion: In our study, patients with a non-dipper pattern of hypertension, which is associated with poor multisystemic outcomes, were found to have a higher waist circumference and subclinical left ventricular dysfunction (abnormal LVGLS). Furthermore, patients with a non-dipper pattern of hypertension comprised 76% of the study population.

Keywords: Altitude, demography, dipper hypertension, hypertension, non-dipper hypertension

ÖZET

Amaç: Hipertansiyon (HT), yaşam tarzı, genetik, zihinsel stres ve çevresel faktörlerle ilişkili karmaşık bir klinik sendromdur. Yüksek rakım (H-ALT), HT gelişimini etkileyen çevresel faktörlerden biridir. Bu çalışma, orta rakımda (M-ALT) yaşayan hastaların klinik ve demografik özelliklerini analiz etmeyi amaçlamaktadır.

Yöntem: 24 saatlik ambulatuvar kan basıncı ölçümü (ABPM) ile hipertansiyonu doğrulanan toplam 515 hasta taranmıştır. Çalışma dışı bırakılanların ardından, nihai çalışma popülasyonu 452 hastadan oluşmuştur. Hastalar, dipper hipertansiyon ve non-dipper hipertansiyon olmak üzere iki gruba ayrıldı.

Bulgular: Non-dipper grubundaki hastalar daha yaşlıydı (SD: 61 ± 13 - 54 ± 12 yıl, P < 0,001) ve anlamlı derecede daha yüksek bel çevresine sahipti (SD: 104 ± 13 - 107 ± 12 cm, P = 0,022). Ancak dipper grubundaki hastaların gündüz ortalama sistolik kan basıncı (SKB) (SD: 144 ± 16 - 141 ± 17 mm Hg, P = 0,027) ve diyastolik kan basıncı (DKB) (SD: 89 ± 12 - 87 ± 13 mm Hg, P = 0,037) anlamlı derecede daha yüksekti. Non-dipper grubundaki hastalarda anormal sol ventrikül global longitudinal strain oranı anlamlı olarak daha yüksekti (n = 218 (%65,6) - n = 53 (%46,7), P = 0,009).

Sonuç: Çalışmamızda, multisistemik sonuçların kötü olduğu non-dipper hipertansiyon paternine sahip hastalarda, daha yüksek bel çevresi ve subklinik sol ventrikül disfonksiyonu (anormal LVGLS) olduğu tespit edildi. Ayrıca, non-dipper hipertansiyon paternine sahip hastalar çalışma popülasyonunun %76'sını oluşturuyordu.

Anahtar Kelimeler: Rakım, demografi, dipper hipertansiyon, hipertansiyon, non-dipper hipertansiyon

ORIGINAL ARTICLE
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Hypertension (HT) is the most common chronic non-communicable disease and a major contributor to global illness and death.[1] Recent data show that about 1.4 billion people worldwide have HT.[2] Blood pressure follows a consistent circadian rhythm, with endogenous neuroendocrine oscillations and other factors leading to a 10%–20% decrease at night compared to daytime.[3] In hypertensive patients, systolic and diastolic blood pressure (BP) typically decrease by more than 10% during sleep compared to daytime. These changes often follow a pattern known as dipper HT. In contrast, non-dipper HT patients do not exhibit this circadian variation, with BP reductions falling below 10%.[4] Non-dipper hypertension has been associated with an increased risk of heart disease and damage to target organs compared to dipper hypertension.[5,6]

Hypertension is a multifaceted clinical condition influenced by lifestyle, genetics, and mental stress.[7] As understanding of hypertension risk factors advances, environmental factors have gained increased attention in recent research, one of which is residential altitude.[2] High-altitude (H-ALT) is an ecological, racial, geographic, and genetic factor that influences the development of systemic HT. The threshold for H-ALT is generally defined as above 2,500 meters above sea level.[8] Studies conducted in H-ALT regions show that the prevalence and risk of hypertension increase with altitude.[9-11] However, there are very few studies on hypertension at moderate altitudes (M-ALT) (1,500-2,500 m). This study aimed to examine the clinical and demographic characteristics of patients residing at M-ALT.

This study received approval from the Research Ethics Committee of Kafkas University (Approval Number: 80576354-050-99/669, Date: 28.05.2025). As it was a single-center retrospective study, informed consent was waived by the Ethics Committee. The study was conducted in accordance with the ethical principles outlined in the Declaration of Helsinki.

Materials and Methods

Study Participants

A total of 515 patients with hypertension, confirmed by 24-hour ambulatory blood pressure monitoring (ABPM), were screened. The study included participants who were at least 18 years old; had no prior diagnosis of hypertension; had their blood pressure measured with ABPM; were diagnosed with hypertension based on guideline threshold values using ABPM (a 24-hour average ABPM of ≥ 130 and/or ≥ 80 mmHg); and had resided at a moderate altitude for at least two years. Participants were excluded from the study if they had a left ventricular ejection fraction (LVEF) of less than 35%; primary severe heart valve disease requiring intervention or surgery; a history of stroke, myocardial infarction, or coronary artery bypass graft surgery within the previous 30 days; congenital heart disease; were on dialysis; or were pregnant. After applying these criteria, 452 patients remained in the final sample (Figure 1).

The patients' baseline demographic and clinical characteristics, along with their medical history—including comorbidities and education level—were retrieved from the hospital's electronic database and patient charts. Education level was categorized as illiterate/primary/secondary school (combined as one group) and high school or above (as another group).

ABBREVIATIONS

2CH	Two-chamber
2DS	Two-dimensional strain
4CH	Four-chamber
ABPM	Ambulatory blood pressure monitoring
ASE	American Society of Echocardiography
BNP	B-type natriuretic peptide
BP	Blood pressure
BSA	Body surface area
CAD	Coronary artery disease
DBP	Diastolic blood pressure
DM	Diabetes mellitus
ECG	Electrocardiogram
eGFR	Estimated glomerular filtration rate
GLS	Global longitudinal strain
H-ALT	High-altitude
HbA1C	Hemoglobin A1C
HBPM	Home blood pressure monitoring
HT	Hypertension
LVEF	Left ventricular ejection fraction
LVGLS	Left ventricular global longitudinal strain
LVM	Left ventricular mass
LVMI	Left ventricular mass index
M-ALT	Moderate altitude
OBP	Office blood pressure
SBP	Systolic blood pressure
TDI	Tissue Doppler imaging

24-Hour ABPM Recordings

Using an ABPM device (SunTech Medical Inc., Morrisville), hypertension was diagnosed according to current guidelines.[12] Twenty-four-hour ABPM recordings were taken every 15 minutes during the day (6:00 a.m. to 10:00 p.m.) and every 30 minutes at night (10:00 p.m. to 6:00 a.m.). From the collected data, patients' awake and sleep periods were identified. The nocturnal blood pressure dip was calculated using the formula: 100 × [1 – (sleep systolic BP / awake systolic BP)]. Circadian blood pressure patterns were classified as dipper (0.8 < ratio ≤ 0.9) or non-dipper (ratio > 0.9) based on nocturnal blood pressure decline.[13] Consequently, if mean systolic and diastolic blood pressure levels decreased by more than 10%, the patient was considered a dipper with hypertension; if they decreased by less than 10%, the patient was classified as a non-dipper; and if they showed an increase, the patient was considered a reverse-dipper.[14] Patients were then divided into two categories for evaluation: dipper and non-dipper (including non-dippers and reverse dippers).

Electrocardiographic (ECG) Analysis

All patients' standard 12-lead electrocardiograms (ECGs) were obtained from the hospital's records. All ECG strips were scanned, uploaded to a computer, and analyzed using ImageJ digital image processing software (available at imagej.nih.gov/ij/).

Echocardiographic Evaluation

All patients underwent transthoracic echocardiography using a Philips HD 11 XE ultrasound machine (Andover, MA). Measurements adhered to the American Society of Echocardiography (ASE)[15] guidelines. All echocardiographic

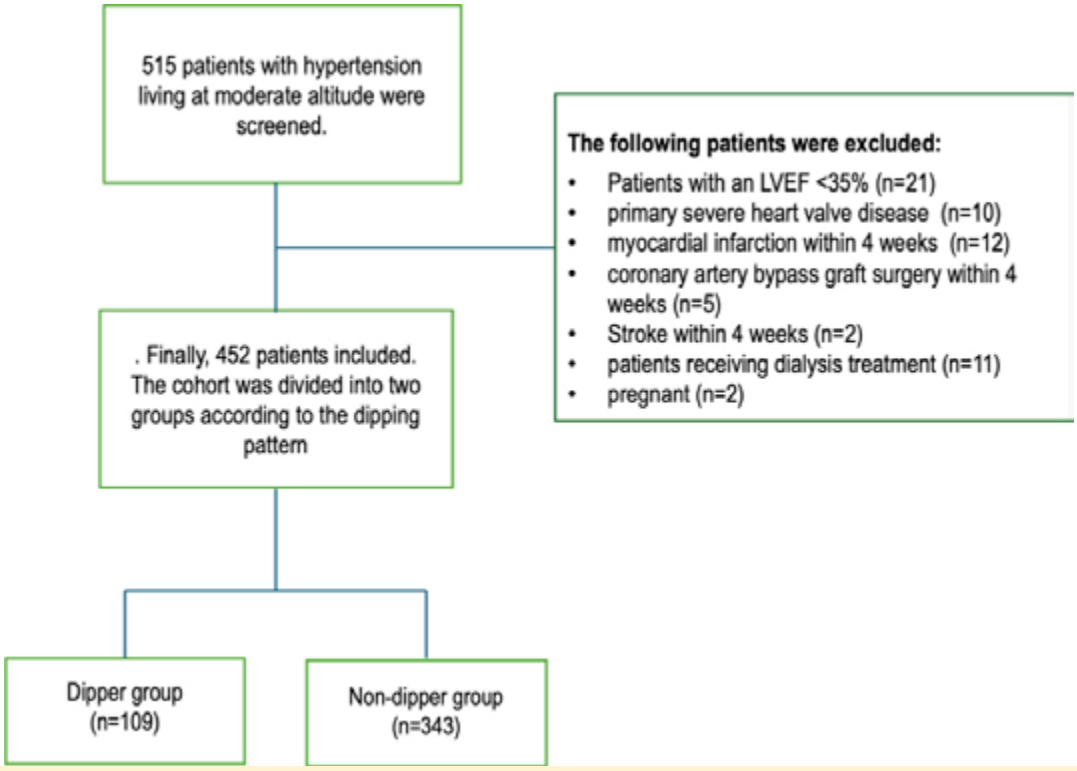

Figure 1. A flow chart illustrating the study design.

measurements were performed transthoracically while the patient was in the left lateral decubitus position. Standard 2D, M-mode, pulsed-wave Doppler, and tissue Doppler imaging (TDI) procedures were carried out according to ASE guidelines.[15] The LVEF was determined using the modified Simpson method. Transmitral inflow velocities (E and A waves) were measured using pulsed-wave Doppler in the apical four-chamber view, with the sample volume positioned at the mitral leaflet tips. TDI was performed at both the septal and lateral mitral annuli to record early diastolic velocity (Em) and late diastolic velocity (Am). Left ventricular global longitudinal strain (LVGLS) was analyzed in three apical left ventricular-focused (LV-focused) views (the apical long-axis, four-chamber (4CH), and two-chamber (2CH) views) using the two-dimensional strain speckle-tracking application (2DS).

The echocardiographic value of left ventricular mass (LVM) was indexed to body surface area (BSA) according to ASE guideline recommendations.[15]

LVM was calculated as follows:

$$LVM = 0.8 \times 1.04 \times [(IVS + LVEDD + PW)^3 - LVEDD^3] + 0.6\,g$$

where

- LVM = Left ventricular mass
- IVS = Interventricular septal thickness
- LVEDD = Left ventricular end-diastolic diameter
- PW = Posterior wall thickness.

Left ventricular hypertrophy (LVH) was defined as LVM/BSA > 115 g/m² in men or > 95 g/m² in women.

Statistical Analyses

Statistical analyses were conducted using SPSS 22.0 (Statistical Product and Service Solutions for Windows, Version 22.0, IBM Corp., Armonk, New York, USA, 2013). Descriptive statistics are presented as mean ± standard deviation (SD) for normally distributed continuous variables, medians with 0.25 and 0.75 quantiles for non-normally distributed variables, and percentages for categorical data. The t-test or Mann-Whitney U test was used to compare continuous variables between groups, while Fisher's exact test or the chi-square test was applied for categorical data.

Results

The study included 233 females (51.5%) and 219 males (48.5%), with an average age of 59 ± 13 years. Overall, 109 (24.1%) patients had a dipper blood pressure pattern, while 343 (75.9%) had a non-dipper pattern. Table 1 presents the baseline demographic features, clinical characteristics, and 24-hour ABPM data of the study population. Patients in the non-dipper group were older (mean age: 61 ± 13 vs. 54 ± 12 years, P < 0.001) and had a significantly higher waist circumference (104 ± 13 vs. 107 ± 12 cm, P = 0.022). They also exhibited higher night-time mean systolic blood pressure (SBP) (139 ± 18 vs. 123 ± 14 mmHg, P < 0.001) and night-time mean diastolic blood pressure (DBP) (83 ± 13 vs. 75 ± 11 mmHg, P < 0.001). Additionally, these patients had higher rates of coronary artery disease (CAD) (77 [22.4%] vs. 12 [11%], P = 0.006), diabetes mellitus (DM) (130 [37.8%] vs. 24 [22%], P = 0.002), and chronic obstructive pulmonary disease (COPD) (32 [9.3%] vs. 2 [1.8%], P = 0.010), as well as higher smoking rates (77 [22.4%] vs. 37 [33.9%], P = 0.016). However, patients in the dipper group had significantly higher daytime mean SBP (144 ± 16 vs. 141 ± 17 mmHg, P = 0.027) and daytime mean DBP (89 ± 12 vs. 87 ± 13 mmHg, P = 0.037).

Table 1. Comparison of baseline demographic, clinical, and ambulatory blood pressure characteristics

Variables	Hypertension			P
	Dipper group n = 109 (24.1%)	Non-dipper group n = 343 (75.9%)	Total n = 452	
Age, years (SD)	54 ± 12	61 ± 13	59 ± 13	<0.001
Gender, female, n (%)	56 (51.4%)	177 (51.6%)	233 (51.5%)	0.96
Education				0.48
Less than high school, n (%)	63 (57.8%)	211 (61.5%)	274 (60.6%)	
High school or more, n (%)	46 (42.2%)	132 (38.5%)	178 (39.4%)	
BSA, m^2 (SD)	1.94 ± 0.22	1.94 ± 0.21	1.94 ± 0.21	0.658
BMI, (kg/m^2) (SD)	29.7 ± 5	30.2 ± 5	30.1 ± 5	0.360
Waist circumference, cm (SD)	104 ± 13	107 ± 12	106 ± 12	**0.022**
Mid-upper arm circumference, cm (SD)	32 ± 4	32 ± 6	32 ± 5	0.389
Neck circumference, cm (SD)	39 ± 5	40 ± 4	40 ± 4	0.190
Daytime mean SBP, mmHg (SD)	144 ± 16	141 ± 17	141 ± 17	**0.027**
Daytime mean DBP, mmHg (SD)	89 ± 12	87 ± 13	87 ± 13	**0.037**
Night-time mean SBP, mmHg (SD)	123 ± 14	139 ± 18	135 ± 18	**<0.001**
Night-time mean DBP, mmHg (SD)	75 ± 11	83 ± 13	81 ± 13	**<0.001**
Coronary artery disease, n (%)	12 (11%)	77 (22.4%)	89 (19.7%)	**0.006**
Diabetes mellitus, n (%)	24 (22%)	130 (37.8%)	154 (34.1%)	**0.002**
Chronic renal disease, n (%)	4 (3.7%)	16 (4.7%)	20 (4.4%)	0.660
Hyperlipidemia, n (%)	20 (18.3%)	80 (23.3%)	100 (22.1%)	0.276
COPD, n(%)	2 (1.8%)	32 (9.3%)	34 (7.5%)	**0.010**
CVA/TIA, n (%)	3 (2.8%)	14 (4.1%)	17 (3.8%)	0.522
OSA, n (%)	1 (0.9%)	12 (3.5%)	13 (2.9%)	0.154
Asthma, n (%)	13 (11.9%)	33 (9.6%)	46 (10.2%)	0.500
Smokers, n (%)	37 (33.9%)	77 (22.4%)	114 (25.2%)	**0.016**

BMI, Body mass index; BSA, Body surface area; COPD, Chronic obstructive pulmonary disease; CVA, Cerebrovascular accident; DBP, Diastolic blood pressure; OSA, Obstructive sleep apnea; SBP, Systolic blood pressure; SD, Standard deviation; TIA, Transient ischemic attack.

Table 2. Medications

Variables	Hypertension			P
	Dipper group n = 109	Non-Dipper group n = 343	Total n = 452	
Hydropyridine CCB, n (%)	25 (22.9%)	88 (25.7%)	113 (25%)	0.558
ACEI, n (%)	18 (16.5%)	69 (20.1%)	87 (19.2%)	0.399
ARB, n (%)	33 (30.3%)	103 (30%)	136 (30.1%)	0.975
Beta-blocker, n (%)	20 (18.3%)	98 (28.6%)	118 (26.1%)	**0.034**
MRA, n (%)	2 (1.8%)	5 (1.5%)	7 (1.5%)	0.784
Thiazide, n (%)	26 (23.9%)	86 (25.1%)	112 (24.8%)	0.786
Indapamide, n (%)	0	6 (1.7%)	6 (1.3%)	0.164
Loop diuretic, n (%)	0	8 (2.3%)	8 (1.8%)	0.108

ACEI, Angiotensin-converting enzyme inhibitor; ARB, Angiotensin receptor blocker; CCB, Calcium channel blocker; MRA, Mineralocorticoid receptor antagonist.

Table 2 shows the drug treatment of the groups. There was no difference between the groups except for the rate of beta-blocker use, which was significantly higher in the non-dipper group (n = 98 (28.6%) vs. n = 20 (18.3%), P = 0.034).

Table 3 presents the baseline electrocardiographic, echocardiographic, and laboratory characteristics of the patients. Patients in the non-dipper group had a significantly lower mean e' (SD: 7.52 ± 2.04 vs. 8.21 ± 2.03 cm/sec, P = 0.003) and a significantly higher rate of abnormal LVGLS (n = 218 [65.6%] vs. n = 53 [46.7%], P = 0.009). Additionally, they showed significantly elevated hemoglobin A1C (HbA1C) levels (SD: 6.2 ± 1.2 vs. 5.9 ± 1%, P < 0.001), urea levels (SD: 37 ± 14 vs. 34 ± 12 mg/dl, P =

Table 3. Comparison of electrocardiographic, echocardiographic, and laboratory characteristics in patients

Variables	Hypertension			P
	Dipper group n = 109	Non-dipper group n = 343	Total n = 452	
Rhythm				0.205
Sinus rhythm, n (%)	107 (98.2%)	331 (96.5%)	438 (96.9%)	
Atrial fibrillation, n (%)	0	4 (1.2%)	4 (0.9%)	
Paced rhythm, n (%)	0	1 (0.3%)	1 (0.2%)	
Heart rate, bpm	77 ± 13	76 ± 14	76 ± 14	0.386
Left BBB, n (%)	1 (0.9%)	16 (4.7%)	17 (3.8%)	0.070
Right BBB, n (%)	2 (1.8%)	15 (4.4%)	17 (3.8%)	0.216
QRS duration, msec (SD)	100 ± 14	102 ± 18	102 ± 17	0.318
LVEF, %	60 ± 7	59 ± 8	59 ± 8	0.156
E/A ratio (SD)	0.84 ± 0.23	0.83 ± 0.32	0.83 ± 30	0.079
E/e' ratio (SD)	8.59 ± 2.59	8.97 ± 3.33	8.88 ± 3.17	0.294
e' mean, cm/sec (SD)	8.21 ± 2.03	7.52 ± 2.04	7.69 ± 2.06	**0.003**
Left ventricular mass index, g/m^2	88 ± 22	89 ± 23	89 ± 22	0.963
Left ventricular hypertrophy, n (%)	18 (16.5)	76 (22.2)	94 (20.8)	0.206
Left atrial AP diameter, cm (SD)	3.5 ± 0.4	3.6 ± 0.4	3.6 ± 0.4	0.115
Tricuspid RV, m/sec (SD)	1.99 ± 0.53	2.02 ± 0.55	2.02 ± 0.54	0.835
LVGLS				**0.009**
GLS >-16%	56 (51.3%)	125 (36.4%)	181 (40%)	
GLS <-16%	53 (46.7%)	218 (65.6%)	271 (60%)	
White blood cell count, ×10^3/μL(SD)	7.5 ± 1.9	7.2 ± 1.9	7.3 ± 1.9	0.142
Hemoglobin, g/dl (SD)	15 ± 1.9	14.4 ± 1.9	14.5 ± 1.9	**0.018**
Hematocrit, %	45 ± 6	43 ± 6	44 ± 6	**0.020**
Platelet count, ×10^3/μL (SD)	235 ± 60	240 ± 80	239 ± 76	0.537
Fasting blood glucose, mg/dl (SD)	103 ± 38	108 ± 45	107 ± 43	0.564
Hemoglobin A1C, %	5.9 ± 1	6.2 ± 1.2	6.1 ± 1.1	**<0.001**
Albumin, g/L (SD)	4.4 ± 0.4	4.2 ± 0.4	4.2 ± 0.4	**<0.001**
eGFR, ml/min/1.73 m^2 (SD)	86 ± 18	81 ± 17	82 ± 17	**0.009**
Urea, mg/dl (SD)	34 ± 12	37 ± 14	36 ± 14	**0.016**
Serum sodium, mmol/l (SD)	139 ± 2	139 ± 2	139 ± 2	0.916
Serum potassium, mmol/l (SD)	4.2 ± 0.4	4.2 ± 0.4	4.2 ± 0.4	0.554
ALT, U/l (IQR)	22 (17-28)	19 (14-17)	20 (15-29)	0.074
AST, U/l (IQR)	20 (16-23)	19 (16-24)	20 (16-24)	0.683
TSH, mIU/L (IQR)	1.56 (1.13-2.31)	1.70 (0.93-2.46)	1.64 (0.99-2.43)	0.876
HDL-C, mg/dl (SD)	51 ± 12	50 ± 12	50 ± 12	0.583
LDL-C, mg/dl (SD)	126 ± 33	122 ± 40	123 ± 39	0.275
Triglycerides, mg/dl (IQR)	129 (99-210)	139 (99-203)	136 (99-204)	0.708
NT-proBNP, ng/ml (IQR)	25 (14-55)	39 (19-75)	34 (17-65)	**0.002**

ALT, Alanine transaminase; AST, Aspartate transaminase; eGFR, Estimated glomerular filtration rate; HDL-C, High-density lipoprotein cholesterol; IQR, Interquartile range; LDL-C, Low-density lipoprotein cholesterol; LVEF, Left ventricular ejection fraction; LVGLS, Left ventricular global longitudinal strain; RV, Regurgitant velocity; SD, Standard deviation; TSAT, Transferrin saturation; TSH, Thyroid-stimulating hormone.

0.016), and B-type natriuretic peptide (BNP) levels (interquartile range [IQR]: 39 [19-75] vs. 25 [14-55] ng/ml, P = 0.002). In contrast, patients in the non-dipper group had significantly lower hemoglobin (SD: 14.4 ± 1.9 vs. 15 ± 1.9 g/dl, P = 0.018), hematocrit (SD: 43 ± 6 vs. 45 ± 6%, P = 0.020), platelet counts (SD: 239 ± 81 vs. 246 ± 63 ×103/μL, P = 0.025), albumin levels (SD: 4.2 ± 0.4 vs. 4.4 ± 0.4 g/dL, P < 0.001), and estimated glomerular filtration rate (eGFR) (SD: 81 ± 17 vs. 86 ± 18 ml/min/1.73 m^2, P = 0.009).

Discussion

Patients with a non-dipper pattern of hypertension made up 76% of the study population. The non-dipper group was older and had higher rates of smoking, abnormal LVGLS, CAD, DM, and COPD based on our analysis of the study population. Furthermore, waist circumference was associated with the non-dipper group, although there were no differences between the HT groups in terms of Body Mass Index (BMI), BSA, arm circumference, or neck circumference. As expected, we examined the differences between the groups and observed that the non-dipper group had higher mean nocturnal SBP and DBP but lower mean daytime SBP and DBP.

In the systematic review by Aryal et al.,[16] the prevalence of HT at H-ALT in the general population appeared to be higher in men. However, there was no gender difference between the groups in our study sample, which included individuals living at altitudes between 1,500 and 2,500 meters. This may be due to factors such as small sample size, ethnic characteristics, study design limitations, and limited geographic areas.

We found that patients in the dipper group had significantly higher mean SBP and DBP levels during the daytime compared to patients in the non-dipper group. However, the non-dipper group had significantly higher nighttime SBP and DBP. This characteristic of the non-dipper group might be related to different types of end-organ damage in this patient population. For example, some parameters reflecting left ventricular systolic and diastolic mechanics and function, such as global longitudinal strain (GLS) and e' mean, were lower. Previous studies suggest that non-dipper HT can lead to either clinical or subclinical LV dysfunction.[17,18] In our study, no significant differences were observed between the groups in echocardiographic measurements of LVEF, E/A ratio, and E/e' ratio. However, LVGLS was significantly lower in the non-dipper group. This is one of the key findings of our study. A low LVGLS, which is increasingly recognized for its role in indicating LV subclinical dysfunction,[19] can be seen as an early sign of organ dysfunction in non-dipper hypertension. Additionally, the mean e' obtained by tissue Doppler, used to assess LV diastolic dysfunction, was found to be decreased. Our findings are consistent with these earlier studies. In our study, left ventricular hypertrophy and the left ventricular mass index (LVMI) showed no differences between the groups. Reviewing the literature, we found some studies[20-22] reporting no difference between patients with LVH and different blood pressure patterns (dipper and non-dipper hypertension). Our study results were similar in this regard. The reasons for this may be that our sample size was small and/or consisted of newly diagnosed patients. An important finding is that the non-dipper pattern group had lower daytime mean BP than the dipper group. It is well known that non-dipper hypertensive patients experience more severe target organ damage—either clinical or laboratory evidence of early hypertensive damage in vascular organs—than dipper patients.[4] In their study, Tanrıverdi et al.[23] demonstrated a significant association between carotid intima-media thickness, a key indicator of atherosclerosis, and non-dipper hypertension. Patients diagnosed, followed-up, and treated based on home blood pressure monitoring (HBPM) or office blood pressure (OBP) may have a higher risk of end-organ damage that could be overlooked or diagnosed late. All patients in our study were diagnosed using ABPM, emphasizing the significance of pattern diagnosis alongside blood pressure measurement.

Previous studies have indicated that non-dippers show a stronger association with organ injury compared to patients with normal nocturnal BP decline, including renal dysfunction, DM, and cardiovascular damage.[24-26] Long-term effects of H-ALT include eGFR, reduced renal plasma and blood flow, and increased filtration fraction.[27] The development of increased erythrocytosis due to persistent hypoxia is a proposed explanation for impaired renal function at H-ALT.[28] Although these effects of high altitude have been examined in various studies, our study population was at M-ALT, yet a significantly lower eGFR was observed in the non-dipper group. This finding suggests that a combination of altitude, ethnicity, hypertension, diet, and other environmental factors may contribute to similar outcomes at different elevations.

Living at H-ALT is known to increase hemoglobin levels.[29-31] In our study, neither group was anemic[32] based on mean hemoglobin levels. However, we found that the non-dipper group had lower hemoglobin and hematocrit levels. This may be due to the negative effects of the non-dipper pattern, as well as the small study population, nutritional factors, and ethnicity. We believe that further research into hemoglobin differences related to impaired circadian blood pressure regulation is necessary.

In their MAPEC trial (Monitorización Ambulatoria para Predicción de Eventos Cardiovasculares/Ambulatory Blood Pressure Monitoring for the Prediction of Cardiovascular Events), Hermida et al.[33] found that after a 5.6-year follow-up, abnormal nighttime blood pressure measured by ABPM was independently associated with the development of diabetes. Condoleo et al.[34] and Santilli et al.[35] also found a significant correlation between DM and HT, particularly with the non-dipper pattern.[34-36] Aryal et al.[37] provided evidence that prediabetes and living at H-ALT are linked. To compare hypertensive individuals regarding diabetes, our study did not include a normotensive group. However, diabetes was more common in the non-dipper group. We believe that this notable adverse effect of the non-dipper pattern should be further investigated in populations living at moderate-to-high altitudes, with larger sample sizes and across different geographic regions. Ambulatory mean systolic BP (particularly nighttime systolic blood pressure) has been shown in previous population studies to better predict cardiovascular events than clinic BP.[38] Inflammation contributes to both HT and CAD, as demonstrated in various studies.[39-41] The connection between chronic inflammation, hypertension, and accelerated atherosclerosis can be explained by increased endothelial dysfunction and decreased vasodilation due to persistently high cytokine levels.[42] Additionally, prior research indicates that both hypertensive and normotensive individuals with a non-dipping blood pressure pattern exhibit a more pronounced inflammatory response.[43-45] Although our study population included hypertensive individuals living at M-ALT, the non-dipper group showed a stronger association with CAD.

Approximately 78% of the risk for primary HT in men and 65% in women is attributed to excessive weight gain, which has long been recognized as a major cause of HT.[46] Studies have indicated that body fat distribution may explain the differences in obesity-related risk, since excess subcutaneous fat is less harmful than abdominal fat accumulation.[47,48] BMI remains the most important measure for estimating body fat and assessing

the cardiometabolic risk associated with obesity.[49] Despite high BMIs, there was no difference between the two groups in our study sample. However, waist circumference was significantly higher in the non-dipper group. Therefore, BMI alone cannot fully represent the cardiometabolic risk associated with obesity. Waist circumference shows a significant relationship with the absolute amount of abdominal fat compared to BMI.[50,51] The statistically significant finding of higher waist circumference in the non-dipper group in our study is important. We believe it is necessary to investigate this finding in a larger cohort of patients from different regions and to establish the potential relationship with more substantial evidence.

Limitations of the Study

The main limitations of this study include its single-center, retrospective design and relatively small sample size. Additionally, ethnic differences could not be identified because the cohort was primarily from one geographic location. Another limitation is the lack of comparison between our patient population and a control (normotensive) group, as well as with hypertensive patients living at sea level.

Conclusion

This study showed that the non-dipper pattern rate may be high in hypertensive individuals living at M-ALT. Since the non-dipper phenotype is associated with worse outcomes related to overall cardiovascular and end-organ damage, the importance of ABPM in diagnosing non-dipper hypertension is emphasized. It is also noteworthy that the mean daytime systolic and diastolic blood pressures in patients with non-dipper hypertension may be lower than in those with dipper patterns, which is an important finding that clinicians should not overlook. Waist circumference is another measure that should be considered in hypertensive patients when evaluating clinical outcomes, even though the link between high BMI and HT is well known. Clinicians should recognize that hypertensive patients living at M-ALT may have elevated mean nocturnal blood pressure levels; therefore, appropriate testing, treatment, and follow-up plans should be implemented.

Ethics Committee Approval: This study received approval from the Research Ethics Committee of Kafkas University (Approval Number: 80576354-050-99/669, Date: 28.05.2025).

Informed Consent: As it was a single-center retrospective study, informed consent was waived by the Ethics Committee.

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