Medical and Research Publications
International Open Access

Research Article **Journal of MAR Cardiology (Volume 4 Issue 6)**

Effect of Dipping Pattern on Cardiovascular Morbidity Assessed by LVH in Different Races with Assessment of Efficacy of Antihypertensive Medications on Dipping

Mohammed Abdelmoneim M Othman MBCCH Msc. PhD, MBA, Ahmed Mounir Gabr MBCCH Msc

Corresponding Author: Mohammed Abdelmoneim M Othman, Al Dhannah Hospital UAE.

Received Date: August 26, 2022

Published Date: September 01, 2022
DOI: 10.1027/marcy.2022.0190

Abstract

***Keywords**: Hypertension, dipping, left ventricular hypertrophy,*

***Background:** Hypertension is a serious medical condition that significantly increases the risks of heart, brain, kidney and other diseases, An estimated 1.28 billion adults aged 30-79 years worldwide have hypertension, ABPM provides the average of BP readings over a defined period, Nocturnal BP normally drop between 10-20 % of diurnal BP , loss of dipping cause serious complications*

***Aim of the Study:** Study relation between dipping status and end organ damage of the cardiac muscle assessed by presence of LVH in ECG & Echocardiography, the relation between antihypertensive type & dipping status & relation between dipping status & race*

***Methods and Results:** Study was designed as observational cross sectional Nested case control study. After assessing prevalence of dipping among studied cases ; cases were divided into groups based on dipping status then association was assessed with finding in ECG , echo ,ambulatory blood pressure monitoring and sociodemographic characteristics.*

Study was carried out in Al Dhannah Hospital Abu Dhabi UAE

***Conclusion:** presence of LVH criteria in ECG in reverse dipper & non dipper was statistically significant than normal dipper with P value < 0.001 ,presence of LVH in Echo was statistically significant in non-dipping & reverse dipping groups than normal dipper with P value < 0.001 ,the best dipping was achieved by ARBIs then combination therapy then equally CCBs & ACEIs & the worst was BB that why BB was not preferred to be used as a solo antihypertensive drugs as it is not decreasing also the central aortic pressure , the worst dipping was in patients from Philippine , the best dipping was achieved in Arab then Indian then African but the number of African was low.*

: Systolic Blood Pressure , DBP: Diastolic Blood Pressure , LV: Left Ventricle , IVSD : Interventricular Septum Diastolic , CKD: Chronic Kidney Disease , CV: Cardiovascular , ECG Electrocardiogram , ABI: Ankle Brachial Index

Abbreviations:

HTN : hypertension , ACEI : Angiotensin convertase Enzyme Inhibitors , ARBI: Angiotensin Receptor Blockers , CCBs: Calcium Channel Blockers , BB: Beta Blockers , HBPM : Home Blood Pressure Monitoring , ABPM : Ambulatory Blood Pressure Monitoring , BP: Blood Pressure , LVH: Left Ventricular Hypertrophy , SBP: Systolic Blood Pressure , DBP: Diastolic Blood Pressure , LV: Left Ventricle IVSD : Interventricular Septum Diastolic , CKD: Chronic Kidney Disease , CV: Cardiovascular , ECG Electrocardiogram , ABI: Ankle Brachial Index

Introduction

Hypertension − or elevated blood pressure − is a serious medical condition that significantly increases the risks of heart, brain, kidney and other diseases ,An estimated 1.28 billion adults aged 30-79 years worldwide have hypertension, most (two-thirds) living in low- and middle-income countries ,An estimated 46% of adults with hypertension are unaware that they have the condition. , Less than half of adults (42%) with hypertension are diagnosed and treated , Approximately 1 in 5 adults (21%) with hypertension have it under control.

Hypertension is a major cause of premature death worldwide, One of the global targets for noncommunicable diseases is to reduce the prevalence of hypertension by 33% between 2010 and 2030. **[**1**]**

Hypertension is defined as office SBP values >_140 mmHg and/or diastolic BP (DBP) values >_90 mmHg. This is based on evidence from multiple RCTs that treatment of patients with these BP values is beneficial

Hypertension was Classified (office blood pressure & hypertension grade) as follow

Category	Systolic (mmHg)		Diastolic (mmHg)
Optimal	<120	and	<80
Normal	120–129	and/or	80–84
High normal	130–139	and/or	85–89
Grade 1 hypertension	140–159	and/or	90–99
Grade 2 hypertension	160–179	and/or	100–109
Grade 3 hypertension	≥180	and/or	≥110
Isolated systolic hypertension[b]	≥140	and	<90

Blood pressure is measured by the following method:

❖ Conventional office blood pressure measurement

Auscultatory or oscillometer semiautomatic or automatic sphygmomanometers are the preferred method for measuring BP in the doctor's office. These devices should be validated according to standardized conditions and protocols. BP should initially be measured in both upper arms, using an appropriate cuff size for the arm circumference **[**2**]**.

❖ Unattended office blood pressure measurement

Automated multiple BP readings in the doctor's office improve the reproducibility of BP measurement, and if the patient is seated alone and unobserved, the 'white-coat effect' can be substantially reduced or eliminated. Moreover, the BP values are lower than those obtained by conventional office BP measurement and are similar to, or even less than, those provided by daytime ambulatory blood pressure monitoring (ABPM) or home blood pressure monitoring (HBPM) **[**3**]**.

❖ Home blood pressure monitoring

Home BP is the average of all BP readings performed with a semiautomatic, validated BP monitor, for at least 3 days and preferably for 6–7 consecutive days before each clinic visit, with readings in the morning and the evening, taken in a quiet room after 5 min of rest, with the patient seated with their back and arm supported. Two measurements should be taken at each measurement session, performed 1–2 min apart. Compared with office BP, HBPM values are usually lower, and the diagnostic threshold for hypertension is >_135/85 mmHg (equivalent to office BP >_140/90 mmHg) when considering the average of 3–6 days of home BP values. Compared with office BP, HBPM provides more reproducible BP data and is more closely related to HMOD, particularly LVH **[**4**]**.

Recent meta-analyses of the few available prospective studies have further indicated that HBPM better predicts cardiovascular morbidity and mortality than office BP. There is also evidence that patient self-monitoring may have a beneficial effect on medication adherence and BP control **[**5**]**.

Ambulatory blood pressure monitoring:

ABPM provides the average of BP readings over a defined period, usually 24 h. The device is typically programmed to record BP at 15 - 30 min intervals, and average BP values are usually provided for daytime, night-time, and 24 h. A diary of the patient's activities and sleep time can also be recorded. A minimum of 70% usable BP recordings are required for a valid ABPM measurement session. ABPM values are, on average, lower than office BP values, and the diagnostic threshold for hypertension is >_130/80 mmHg over 24 h, >_135/85 mmHg for the daytime average, and >_120/70 for the night time average (all equivalent to office BP >_140/90 mmHg), ABPM is a better predictor of HMOD than office BP. Furthermore, 24 h ambulatory BP mean has been consistently shown to have a closer relationship with morbid or fatal events, and is a more sensitive risk predictor than office BP of CV outcomes such as coronary morbid or fatal events and stroke **[**6**]**.

Definitions of hypertension according to office, ambulatory, and home blood pressure levels

Category	SBP (mmHg)		DBP (mmHg)
Office BP[a]	≥140	and/or	≥90
Ambulatory BP			
Daytime (or awake) mean	≥135	and/or	≥85
Night-time (or asleep) mean	≥120	and/or	≥70
24 h mean	≥130	and/or	≥80
Home BP mean	≥135	and/or	≥85

Dipping of blood pressure

BP normally decreases during sleep. Although the degree of night time BP dipping has a normal distribution in a population setting, an arbitrary cut-off has been proposed to define patients as 'dippers' if their nocturnal BP falls by >10% of the daytime average BP value **[**7**]**.

Recognised reasons for an absence of nocturnal BP dipping are sleep disturbance, obstructive sleep apnoea, obesity, high salt intake in salt-sensitive subjects, orthostatic hypotension, autonomic dysfunction, CKD, diabetic neuropathy, and old age. Studies that accounted for daytime and night-time BP in the same statistical model found that night-time BP is a stronger predictor of outcomes than daytime BP. The night-to-day ratio is also a significant predictor of outcome, and patients with a reduced night time dip in BP (i.e. <10% of the daytime average BP or a night-to-day ratio >0.9) have an increased cardiovascular risk 8. Moreover, in those in whom there is no night-time dip in BP or a

higher night-time than daytime average BP, there is a substantially increase in risk. Paradoxically, there is also some evidence of increased risk in patients who have extreme dipping of their night-time BP, although the limited prevalence and reproducibility of this phenomenon makes interpretation of data difficult **[**9**]**.

Classification of dipping pattern in hypertensive patients

- Extreme dippers: decline in nocturnal blood pressure 20% or more of daytime BP
- Dippers: decline from 10 to 20%
- Non-dippers: decline from 0 to 10%
- Inverted dippers: no BP decline

Inverted dippers had the highest mortality risk, followed by non-dippers; no difference in mortality was found between extreme dippers and dippers.

White-coat hypertension

Although the prevalence varies between studies, white-coat hypertension can account for up to 30 - 40% of people (and >50% in the very old) with an elevated office BP. It is more common with increasing age, in women, and in non-smokers. Its prevalence is lower in patients with HMOD, when office BP is based on repeated measurements, or when a doctor is not involved in the BP measurement. A significant white-coat effect can be seen at all grades of hypertension (including resistant hypertension), but the prevalence of white-coat hypertension is greatest in grade 1 hypertension

Assessment of hypertension-mediated organ damage

Basic screening tests for HMOD	Indication and interpretation
12-lead ECG	Screen for LVH and other possible cardiac abnormalities, and to document heart rate and cardiac rhythm
Urine albumin:creatinine ratio	To detect elevations in albumin excretion indicative of possible renal disease
Blood creatinine and eGFR	To detect possible renal disease
Fundoscopy	To detect hypertensive retinopathy, especially in patients with grade 2 or 3 hypertension
More detailed screening for HMOD	
Echocardiography	To evaluate cardiac structure and function, when this information will influence treatment decisions
Carotid ultrasound	To determine the presence of carotid plaque or stenosis, particularly in patients with cerebrovascular disease or vascular disease elsewhere
Abdominal ultrasound and Doppler studies	• To evaluate renal size and structure (e.g. scarring) and exclude renal tract obstruction as possible underlying causes of CKD and hypertension • Evaluate abdominal aorta for evidence of aneurysmal dilatation and vascular disease • Examine adrenal glands for evidence of adenoma or phaeochromocytoma (CT or MRI preferred for detailed examination); see section 8.2 regarding screening for secondary hypertension • Renal artery Doppler studies to screen for the presence of renovascular disease, especially in the presence of asymmetric renal size
PWV	An index of aortic stiffness and underlying arteriosclerosis
ABI	Screen for evidence of LEAD
Cognitive function testing	To evaluate cognition in patients with symptoms suggestive of cognitive impairment
Brain imaging	To evaluate the presence of ischaemic or haemorrhagic brain injury, especially in patients with a history of cerebrovascular disease or cognitive decline

The most used simple criteria and recognized cut-off points for definitions of electrocardiogram left ventricular hypertrophy

ECG voltage criteria	Criteria for LVH
$S_{V1}+R_{V5}$ (Sokolow–Lyon criterion)	>35 mm
R wave in aVL	≥11 mm
$S_{V3}+R_{aVL}$ (Cornell voltage)[a]	>28 mm (men)
Cornell duration product[b]	>20 mm (women)
	>2440 mm.ms

Echocardiographic

Left ventricular dimensions including left ventricular internal diameter in diastole (mm), interventricular septal thickness in diastole (IVSd, mm), and posterior wall thickness in diastole (PWTd, mm) & measurement of left ventricular mass is highly sensitive , specific & accurate to assess the presence of left ventricular hypertrophy according to ASE guidelines

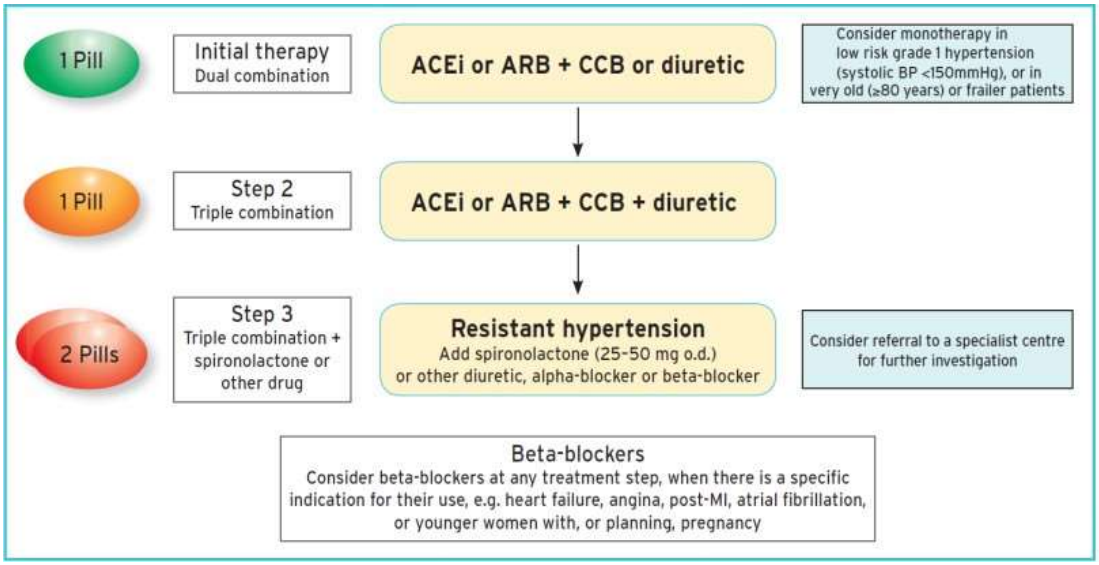
	Male				Female			
	Normal range	Mildly abnormal	Moderately abnormal	Severely abnormal	Normal range	Mildly abnormal	Moderately abnormal	Severely abnormal
LV mass by linear method								
Septal wall thickness (cm)	0.6–1.0	1.1–1.3	1.4–1.6	>1.6	0.6–0.9	1.0–1.2	1.3–1.5	>1.5
Posterior wall thickness (cm)	0.6–1.0	1.1–1.3	1.4–1.6	>1.6	0.6–0.9	1.0–1.2	1.3–1.5	>1.5
LV mass (g)	88–224	225–258	259–292	>292	67–162	163–186	187–210	>210
LV mass/BSA (g/m^2)	49–115	116–131	132–148	>148	43–95	96–108	109–121	>121

Hypertension treatment according to ESC & ACC guidelines should follow these steps

❖ When to initiate antihypertensive treatment

All guidelines agree that patients with grade 2 or 3 hypertension should receive antihypertensive drug treatment alongside lifestyle interventions. Guidelines are also consistent in recommending that patients with grade 1 hypertension and high CV risk or HMOD should be treated with BP-lowering drugs **[**10**]**.

- Lifestyle changes, Dietary sodium restriction , moderation of alcohol consumption , wight reduction , regular physical activity & smoking cessation
- pharmacological; drug therapy
- Angiotensin convertase enzyme & Angiotensin receptor inhibitors in the study ACEI used are lisinopril, ramipril & perindopril , ARBI used were candesartan , valsartan , Olmesartan & telmisartan
- Calcium channel blockers were amlodipine & lercadipine
- Thiazide like diuretics
- Beta blockers was bisoprolol
- Other drugs; centrally acting & alpha blockers



Relation between non dipping & left ventricular hypertrophy

Ambulatory blood pressure profile has a better relationship to the LVM in hypertensive subjects with LVH than OBP measurements, Also the regression of LVH associated with improved CV prognosis is more closely correlated to the reductions in ABP than OBP **[**11**]**.

Many studies had concluded that the non-dipping pattern of hypertension may the responsible for the development of the concentric type of LVH, Blood pressure profile without the physiological dipping in the night is believed to promote structural changes in the LV, The rennin angiotensin aldosterone system is most probably responsible for that **[**12, 13**]**.

The non-dipper patients are at higher risk of micro-and macrovascular complications than dipper patients, and the non-dipping status is a determinant of cardiac remodelling and LV diastolic dysfunction (LVDD) and may result in a cardiovascular risk independent of the increase in LVM, Hypertensive subjects with the non-dipper pattern have more LVM than those with the dipper pattern. Also subjects with the non-dipper pattern have higher risks of cardiac and extra-cardiac morbidity **[**14,15**]**.

Other consequences of non-dipping & reverse dipping in hypertensive patients is documented in numerous studies that have shown that hypertensive subjects with a nocturnal BP fall lower than 10% have more pronounced left ventricular hypertrophy (LVH), abnormalities in carotid structure, renal dysfunction, preclinical cerebrovascular disease and increased likelihood of cardiovascular (CV) events than their counterparts with preserved nocturnal BP fall**[**16**]**.

Patients and Methods

Sample size

Sample size calculation was based on 34 % prevalence of dipping that is retrieved from previous research (Abdalla et al., 2017).Using Daniel equation (1999), $n = \frac{Z2p(\$\%p)}{d}$

With 95% Confidence interval and acceptable margin error of 5%. The calculated sample size in the study was at least 345 where, 20% will be added to overcome dropout rate to finally be approximately 414 at least.

Study was designed as observational cross sectional Nested case control study. After assessing prevalence of dipping among studied cases ; cases were divided into groups based on dipping status then association was assessed with finding in ECG , echo, ambulatory blood pressure monitoring and sociodemographic characteristics.

Inclusion criteria:

All hypertensive patients or patients with high blood pressure readings more than 140/90 mmhg ranging from 18 to 75 years old

Exclusion criteria:

- Age below 18 or above 75 years old

Electrocardiogram

12 leads ECG was done by certified nurses using Medtronic ECG machine & interpretated by certified cardiologists.

Ambulatory blood pressure device

Participants were fitted with an ABPM device (WelchAllyn) on their dominant arm following the baseline examination. Ambulatory BP was recorded every 60 minutes. After 24 hours, the device was

removed, and data were downloaded onto a computer and processed with WelchAllyn software was used to define whether the ABPM period was complete.

Specifically, participants were considered to have a complete ABPM if they had ≥10 daytime (10 AM– 10 PM) and ≥5 night time (11PM– 7 AM) SBP and DBP measurements.

Mean daytime SBP and DBP and mean night time SBP and DBP were calculated by averaging the readings during the daytime and night time periods, respectively.

Mean 24-hour BP was defined by averaging all available BP measurements from ABPM.

The night time to daytime SBP ratio was defined as mean night time SBP divided by mean daytime SBP.

Dipping was categorized into 4 patterns based on the night time to daytime SBP ratio:

- Extreme dippers: decline in nocturnal blood pressure 20% or more of daytime BP
- Dippers: decline from 10 to 20%
- Non-dippers: decline from 0 to 10%
- Inverted dippers: no BP decline

Echocardiography

Certified experienced cardiologists performed 2-dimensional transthoracic echocardiography (Philips Medical Systems, EPIQCVX) using standardized protocols. Left ventricular dimensions including left ventricular internal diameter in Diastole (mm), interventricular septal thickness in diastole (IVSd, mm), and posterior wall thickness in diastole (PWTd, mm), were assessed according to American Society of

Echocardiography (ASE) recommendations.

Aim of the study:

1) Study relation between dipping status and end organ damage of the cardiac muscle assessed by presence of LVH in ECG & Echocardiography

2) Study the relation between antihypertensive type & dipping status 3) Study relation between dipping status & race

Statistical analysis

Data were fed to the computer and analyzed using IBM SPSS Corp. Released 2013. IBM SPSS Statistics for Windows, Version 22.0. Armonk, NY: IBM Corp. Qualitative data were described using number and percent. Quantitative data were described using mean, standard deviation for normally distributed data after testing normality using Kolmogrov-Smirnov test. Significance of the obtained results was judged at the (0.05) level. Chi-Square and Monte Carlo tests for comparison of 2 or more groups for qualitative variables. One Way ANOVA test was used to compare more than 2 independent groups with Post Hoc Tukey test to detect pair-wise comparison. Binary stepwise logistic regression analysis was used for prediction of left ventricular hypertrophy. Significant predictors in the Univariate analysis were entered into regression model using forward Wald method . Adjusted odds ratios and their 95% confidence interval were calculated.

Study was carried out in Al Dhannah hospital in UAE

Results

The present study is observational cross sectional nested case control study that is carried out on 500 to assess relationship between dipping and left ventricular hypertrophy and other associated factors. Mean age of the studied cases is 46.65 years ranging from 16 to 70 years , 94.4% are males . Antihypertensive drugs used are distributed as following ; 27.4% Angiotensin convertase enzyme inhibitors, 26.2% Calcium channel blockers, 25.1% ARABS , 18% combination therapy(Sevikar and Exforge). Among studied cases ;39% have dipping , 18.4% reversed dipping and 33.6% non dipping and 5 cases extreme dipping. Race of the studied cases distributed as following; 41.2% are Indians , 28.8% Asian including Indonesian , Philippines and Bangladish , 21% arabs , 6.4% other races including American , Australian and Romanaian as shown in table (1)

	N	%
Age/years mean±SD (MIN-MAX)	46.65±9.61 (16-70)	
Sex		
Male	472	94.4
Female	28	5.6
Antihypertensive Drugs	n=439	
ARBS	110	25.1
Calcium channel blockers	115	26.2
Angiotensin convertase enzyme inhibitors	119	27.1
Beta blockers	16	3.6
Combination	79	18.0
ECG		
normal	315	63.0
LVH	185	37.0
Echo		
Normal	317	63.4
Mild LVH	153	30.6
Moderate LVH	27	5.4
Severe LVH	3	0.8
Dipping		
Non dipping	168	33.6
Reversed	97	18.4
Dipping	235	39.0
Extreme dipping	5	1.0
Race		
Indian	206	41.2
ARAB	105	21.0
African	13	2.6
Asian	144	28.8
others#	32	6.4
Systolic blood pressure	mean±SD (MIN-MAX)	
Total	123.90±12.99(91-167)	
Day	126.16±14.07(91-188)	
Night	121.42±15.79(85-183)	
Diastolic blood pressure	mean±SD (MIN-MAX)	
Total	81.79±9.07(53-105)	
Day	83.74±10.31(53-133)	
Night	79.83±11.31(50-122)	

#romanian, USA, Australia

Table (1) Characteristics of the studied patients

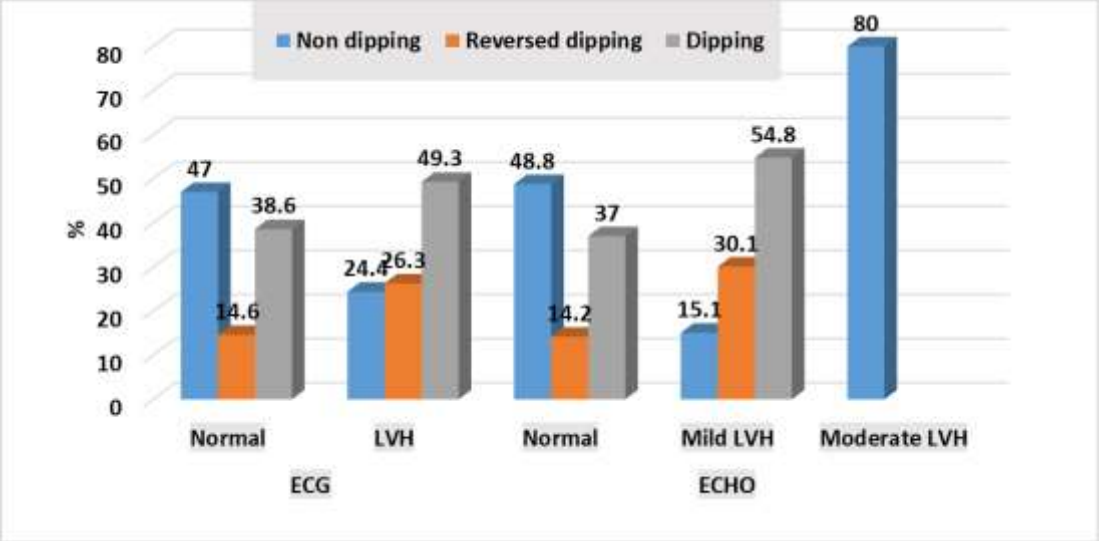
A Statistically significant relation is detected between dipping and the following factors; age , sex , race, ECG , Echo , antihypertensive drugs , systolic & diastolic blood pressure ($p<0.05$).Higher mean age is detected among cases with non-dipping than dipping ($p=0.009$).Dipping and reverse dipping are higher among females than males(66.7% versus 42.2%) and (33.3% versus 19.3%), respectively. For studied races ; 61.5% of African , 45.9% of Indian , 45.4% of arab and 35.6% of Asian race have dipping and 38.5% of African, 21.6% Indian & arab and 15.2% of Asian races have reverse dipping. Among cases with left ventricular hypertrophy by ECG ; 49.3% have dipping and 26.3% have reversed dipping and for cases with mild left ventricular hypertrophy ; 54.8%dipping and 30.1% reversed dipping with statistically significant difference between cases with reverse dipping and dipping against dipping group. Dipping is detected among 53.8% of cases on ARABS ,44.8% of cases with

combination treatment , 36.9% of cases on ACEIS , 36% of cases with CCBS and 25% of cases on beta blocker treatment as show in in table (2)

	Total number	Non dipping N=168(%)	Reversed dipping N=97(%)	Dipping N=235(%)	Test of significance	Within group significance
Age/years mean±SD	500	48.01±8.63	47.28±11.56	45.44±8.92	F=3.59 P=0.028*	P1=0.550 P2=0.009* P3=0.121
Sex						P1<0.001*
male	436	168(38.5%)	84(19.3%)	184(42.2%)	^{2}=14.63	P2<0.001*
Female	24	0	8(33.3%)	16(66.7%)	p=0.001*	P3=0.841
Race						
Indian	194	63(32.5%)	42(21.6%)	89(45.9%)		P1=0.003*
ARAB	97	32(33.0%)	21(21.6%)	44(45.4%)	^{2MC}=19.59	P2=0.004*
African	13	0	5(38.5%)	8(61.5%)	p=0.012*	P3=0.948
Philippine	132	65(49.2%)	20(15.2%)	47(35.6%)		
Others	24	8(33.3%)	4(16.7%)	12(50.0%)		
ECG						P1=0.001*
Normal	279	111(39.8)	27(8.6)	179(51.6)	^{2}=58.91	P2=0.219
LVH	181	57(31.5)	70(37.6)	56(30.9)	p<0.001*	P3<0.001*
ECHO						
Normal	281	113(40.2)	24(8.5)	179(51.2)		P1<0.001*
Mild LVH	149	33(22.1)	68(42.3)	53(35.6)	^{2MC}=91.18	P2<0.001*
Moderate LVH	27	19(70.4)	5(18.5)	3(11.1)	p<0.001*	P3<0.001*
Severe LVH	3	3(100.0)	0	0		
Antihypertensive drugs						P1=0.001*
ARBI	106	33(31.1%)	16(15.1%)	57(53.8%)	^{2MC}=33.69	P2=0.027*
CCBS	111	33(29.7%)	38(34.2%)	40(36.0%)	p<0.001*	P3=0.002*
ACEIS	111	49(44.1%)	21(18.9%)	41(36.9%)		
Beta blocker	16	12(75.0%)	0	4(25.0%)		
Combination	67	28(41.8%)	9(13.4%)	30(44.8%)		
Systolic blood pressure						
Total	500	129.09±13.22	121.15±14.20	121.62±10.51	F=20.41,P<0.001*	P1<0.001* ,
Day	500	131.30±15.18	119.91±14.49	125.83±10.98	F=38.78,P<0.001*	P2<0.001* , P3=0.763
Night	500	128.42±15.50	132.58±14.81	112.51±15.72	F=22.26, P<0.001*	P1<0.001* , P2<0.001* , P3=0.08 P1<0.001* , P2<0.001*,P3<0.001*
Diastolic blood pressure						
Total	500	86.42±8.52	78.28±7.42	80.11±8.37	F=35.39,P<0.001*	P1<0.001*,
Day	500	87.94±10.65	77.57±8.0	83.98±9.14	F=96.06,P<0.001*	P2<0.001* , P3=0.08
Night	500	85.67±9.97	87.59±9.35	73.07±8.04	F=123.16, P<0.001*	P1<0.001*, P2<0.001* , P3<0.001* P1=0.104 , P2<0.001*,P3<0.001*

F:One Way ANOVA test , 2MC:Monte Carlo test , p1:difference between non-dipping & reversed dipping , p2: difference between non- dipping &dipping, p3: difference between reversed dipping and dipping groups.

Table (2): relation between studied risk factors and dipping among studied cases



Multivariate analysis was carried out to detect predictors of left ventricular hypertrophy among cases and illustrates that cases with dipping have risk 3.21 more times of developing hypertension than non-dipping cases and cases with reverse dipping have risk of 3.18 times to develop LVH than cases with non-dipping (Adjusted odds ratio ;95% CI =3.21 ;1.77-5.82 & 3.18 ;1.96-5.19 , respectively).Arabs race have risk 1.99 more time to develop hypertension than Indian race (Adjusted odds ratio ;95% CI =1.99 ;1.117-3.575) and cases on CCBs treatment have risk of 2.45 to develop LVH than cases on ARABS (Adjusted odds ratio ;95% CI =2.45 ;1359-4.435).Overall % predicted of left ventricular hypertrophy is 65.2% by the combination of the previous significant factors as shown in table (3).

Model steps	Predictors	β	P value	odds ratio	95.0% C.I.for OR	
					Lower	Upper
Step 1[a]	Non dipping(r)		<.001*			
	Dipping	1.248	<.001*	3.485	1.999	6.073
	Reversed dipping	1.026	<.001*	2.789	1.767	4.405
	Constant	-.863	<.001*	.422		
Step 2[b]	Non dipping(r)		<.001*			
	Dipping	1.178	<.001*	3.247	1.822	5.785
	Reversed dipping	1.122	<.001*	3.071	1.908	4.943
	Anti-hypertensive drugs					
	ARABS (r)		.014*			
	CCBS	.913	.002*	2.492	1.405	4.421
	ACEIS	.307	.289	1.359	.771	2.395
	Beta blocker	1.011	.074	2.749	.905	8.349
	Combination	.125	.706	1.133	.592	2.171

	Constant	-1.281	<.001*	.278		
Step 3[c]	Non dipping (r)		<.001*			
	Dipping	1.167	<.001*	3.212	1.771	5.824
	Reversed dipping	1.159	<.001*	3.186	1.957	5.188
	Race					
	Indian (r)		.048*			
	ARAB	.692	.020*	1.999	1.117	3.575
	African	1.095	.198	2.990	.563	15.879
	Asian	-.001	.996	.999	.601	1.660
	Others	-.576	.251	.562	.210	1.503
	Anti-hypertensive drugs					
	ARABS (r)		.009*			
	CCBS	.898	.003*	2.455	1.359	4.435
	ACEIS	.374	.216	1.453	.804	2.627
	Beta blocker	1.134	.051	3.107	.996	9.692
	Combination	-.066	.851	.936	.471	1.861
	Constant	-1.411	<0.001*	.244		
Overall % predicted =65.2%						

Table (3): multivariate analysis for predictors of left ventricular hypertrophy as detected by ECG

Discussion

Many studies tried to find a relation between dipping pattern & LVH (left ventricular geometric changes) with a lot of discrepancies in the results, most studies compared only two forms of dipping pattern either dipping , non-dipping & reverse dipping.

In Cesare Cuspidi et they found increased incidence of LVH with non-dipping pattern of HTN mostly linked to sleep disturbance disorders & found that searching for nocturnal hypertension in clinical practice is crucial for improving CV risk stratification and choosing therapeutic strategies.**[**17**]**, Marwah Abdalla, MD, et al they found In this population-based study of blacks, a reverse dipping pattern was associated with increased left ventricular mass index and a higher prevalence of left ventricular hypertrophy. Identification of a reverse dipping pattern on ambulatory BP monitoring may help identify black at increased risk for cardiovascular target organ damage.

But in Ahmed H Abdel Moneim, et al There was no statistically significant difference between the different LV geometric patterns between dippers and non-dippers by ABPM. Daytime SBP and 24-h systolic BP elevations were associated with significant differences in LV geometric changes in hypertensive patients, but this study has serious limitation which is small sample size as only 150 patients were included.

In Marijana Tadic et al The present investigation showed that non-dipping and reverse dipping BP patters correlated with CV events during long-term follow-up. However, only reverse dipping was independently of nighttime SBP, LV remodelling, and other usual CV risk factors related with adverse CV outcome in initially untreated hypertensive patients.

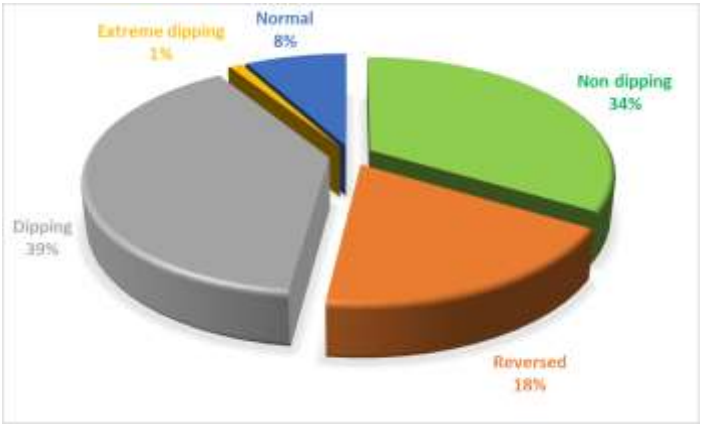
In our study the 500 patients 168 were non dipper 31 % ,111 had normal ECG , 57 (33%)had LVH criteria , 97 patients had reversed dipping 27 had normal ECG & 70 had LVH criteria (37.1) & 235 had dipping 179 had normal ECG & 56 had LVH criteria (30.9) , so presence of LVH criteria in ECG in reverse dipper & non dipper was statistically significant than normal dipper with P value < 0.001

In our study 168 patients were non-dipper 113 had normal Echo , 33 had mild LVH IVSD 12 mm 19 had moderate LVH IVSD 13 mm & 3 had sever LVH IVSD more than 14 mm , 97 patients were reverse dipper 24 had normal Echo , 68 had mild LVH IVSD 12 mm 5 had moderate LVH IVSD 13 mm & 235 patients were dipper 179 had normal Echo , 53 had mild LVH IVSD 12 mm ,3 had moderate LVH IVSD 13 mm , so presence of LVH in Echo was statistically significant in non-dipping & reverse dipping groups than normal dipper with P value < 0.001.

In our study 106 patients were using ARBI 57 (53%) were dipper , 33(31%) non dipper & 16 (15.1%) reverse dipper , 67 patients were using combination therapy 30 (44.8%) were dipper , 28(41.8%) non dipper & 9 (13.4%) reverse dipper, 111 patients were using ACEI 41 (63.9%) were dipper , 49 (44.1%) non dipper & 21 (18.9%) reverse dipper , 111 patients were using CCB 40 (36%) were dipper.

, 49 (44.1%) non dipper & 21 (18.9%) reverse dipper & 16 patients were using BB 4 (25 %) were dipper , 12 (75%) non dipper , so the best dipping was achieved by ARBIs then combination therapy then equally CCBs & ACEIs & the worst was BB.

In our study 194 patients were Indian 89 (45.9%) of them were dipper , 63 (32.5%) non dipper & 42 (21.6%) reverse dipper , 132 patients were Philippine 47 (35.6%) of them were dipper , 65 (49.2%) non dipper & 20 (15.2 %) reverse dipper , 97 patients were Arab 44 (45.4%) of them were dipper , 32 (33%) non dipper & 21 (21.6%) reverse dipper , 13 patients were African 8 (61.5%) of them were dipper & 5 (38.5%) reverse dipper , so the worst dipping was in patients from Philippine , the best dipping was achieved in Arab then Indian then African but the number of African was low.



Conclusion

Presence of LVH criteria in ECG in reverse dipper & non dipper was statistically significant than normal dipper with P value < 0.001. presence of LVH in Echo was statistically significant in non-dipping & reverse dipping groups than normal dipper with P value < 0.001.

the best dipping was achieved by ARBIs then combination therapy then equally CCBs & ACEIs & the worst was BB that why BB was not preferred to be used as a solo antihypertensive drugs as it is not decreasing also the central aortic pressure. the worst dipping was in patients from Philippine , the best dipping was achieved in Arab then Indian then African but the number of African was low.

Strengths & weakness of the study Strengths:

1. the study was conducted on good sample size 500 patients
2. multi-varieties & multi-ethnic
3. used all types of antihypertensive medications

Weakness:

1. Study measured one aspect of the side effects of lost of dipping and didn't look for cerebrovascular complications.
2. Was better to be accompanied by sleep study to measure the effect of sleep.
3. Disturbance caused by obstructive sleep apnea.
4. Number of African patients was less.

Acknowledgment

I would like to thank Al Dhannah hospital & Adnoc management for all of the support they offered for research work , all of nursing team in cardiology department for their help in collecting data , prof. Hind for statistical analysis & my wife Dr Noha & my kids Habiba , Razan & Hamza as I owe them the time I am spending in research.

References

1. Cardiovascular diseases (CVDs). Key facts. 17 May 2017 **[**website**]** (https://www.who.int/news-room/fact- sheets/detail/cardiovascular-diseases-(cvds), accessed 31 March 2021

2. Stergiou GS, Alpert B, Mieke S, Asmar R, Atkins N, Eckert S, Frick G, FriedmanB, Grassl T, Ichikawa T, Ioannidis JP, Lacy P, McManus R, Murray A, Myers M, Palatini P, Parati G, Quinn D, Sarkis J, Shennan A, Usuda T, Wang J, Wu CO, O'Brien E. A universal standard for the validation of blood pressure measuring devices: Association for the Advancement of Medical Instrumentation/European Society of Hypertension/International Organization for Standardization (AAMI/ ESH/ISO) Collaboration Statement. J Hypertens 2018;36:472–478.

3. Myers MG. A short history of automated office blood pressure - 15 years to SPRINT. J Clin Hypertens (Greenwich) 2016;18:721–724.

4. Bliziotis IA, Destounis A, Stergiou GS. Home versus ambulatory and office blood pressure in predicting target organ damage in hypertension: a systematic review and meta-analysis. J Hypertens 2012;30:1289–1299.

5. McManus RJ, Mant J, Bray EP, Holder R, Jones MI, Greenfield S, Kaambwa B, Banting M, Bryan S, Little P, Williams B, Hobbs FD. Telemonitoring and self-management in the control of hypertension (TASMINH2): a randomised controlled trial. Lancet 2010;376:163–172

6. Banegas JR, Ruilope LM, de la Sierra A, Vinyoles E, Gorostidi M, de la Cruz JJ, Ruiz-Hurtado G, Segura J, Rodriguez-Artalejo F, Williams B. Relationship between clinic and ambulatory blood-pressure measurements and mortality. N Engl J Med 2018;378:1509– 1520.

7. Omboni S, Parati G, Palatini P, Vanasia A, Muiesan ML, Cuspidi C, Mancia G. Reproducibility and clinical value of nocturnal hypotension: prospective evidence from the SAMPLE study. Study on Ambulatory Monitoring of Pressure andLisinopril Evaluation. J Hypertens 1998;16:733–738

8. Parati G, Stergiou G, O'Brien E, Asmar R, Beilin L, Bilo G, Clement D, de la Sierra A, de Leeuw P, Dolan E, Fagard R, Graves J, Head GA, Imai Y, Kario K, Lurbe E, Mallion JM, Mancia G, Mengden T, Myers M, Ogedegbe G, Ohkubo T, Omboni S, Palatini P, Redon J, Ruilope LM, Shennan A, Staessen JA, vanMontfrans G, Verdecchia P, Waeber B, Wang J, Zanchetti A, Zhang Y, European Society of Hypertension Working Group on Blood Pressure Monitoring and Cardiovascular Variability. European Society of Hypertension practice guidelines for ambulatory blood pressure monitoring. J Hypertens 2014;32:1359–1366.

9. Mancia G, Verdecchia P. Clinical value of ambulatory blood pressure: evidence and limits. Circ Res 2015;116:1034–1045.

10. Kjeldsen S, Feldman RD, Lisheng L, Mourad JJ, Chiang CE, Zhang W, Wu Z, Li W, Williams B. Updated national and international hypertension guidelines: a review of current recommendations. Drugs 2014;74:2033–2051.

11. Verdecchia P, Porcellati C, Schillaci G, Borgioni C, Ciucci A, et al. (1994) Ambulatory blood pressure. An independent predictor of prognosis in essential hypertension. Hypertension. 24: 793-801.

12. Kieć B, Stolarz K, Sliwa A, Zdzienicka A (2010) Peripheral blood concentrations of TGF beta 1, IGF-1 and Bf GF and remodelling of the left ventricle and blood vessels in hypertensive patients. Kardiol Pol 68: 996-1002.

13. Soylu A, Duzenli M, Yazici M, Ozdemir K, Tokac M, et al. (2009) The effect of non-dipping blood pressure patterns on cardiac structural changes and left ventricle diastolic functions in normotensives. Echocardiography 26: 378-387.

14. Zhu H, Zhang W, Shi Y, Ma Y, Han Y, et al. (2014) Relationship between Nocturnal Blood Pressure and Left Ventricular Hypertrophy in Hypertensive Patients. J Hypertens 3: 151.

15. Ghali J, Liao Y, Cooper RS (1998) Influence of LV geometric patterns on prognosis in patients with or without coronary artery disease. J Am Coll Cardiol 31: 1635-1640.

16. Ohkubo T, Hozawa A, Yamaguchi J et al. Prognostic significance of nocturnal decline in blood pressure in subjects with and without high 24-hour blood pressure: the Ohasama study. J. Hypertens. 20, 2183–2189 (2002).

17. Palatini P, Parati G. Modulation of 24-h blood pressure profiles: a new target for treatment. J. Hypertens. 23, 1799–1801 (2005).