Original Article

The relationship between morning blood pressure surge and left ventricular hypertrophy in newly diagnosed hypertensive patients

Beyza Algul Durak[1], Emre Tutal[2], Musa Ilker Durak[3]

[1]Department of Nephrology, Ankara City Hospital, Ankara, Turkey
[2]Department of Nephrology, Medicana International Hospital, Ankara,Turkey
[3]Department of Cardiology, Golbasi State Hospital, Ankara, Turkey

Abstract

Aim: The main aim of this study is to reveal the relationship between the blood pressure surge pattern in newly diagnosed hypertensive patients and left ventricular hypertrophy (LVH).
Materials and Methods: 92 patients (60 female, 32 male) were included in the study. Patients who have not been diagnosed with hypertension or even if they have been diagnosed with it, have not received any medication since the last 3 weeks, and who were newly diagnosed with hypertension were included in the study. Morning Blood Pressure Surge (MBPS) was calculated by subtracting the lowest systolic blood pressure recorded during sleep from the systolic blood pressure value of the 2nd hour after awakening.
Results: It was observed that 69 patients were nondippers and 29 patients had LVH. As a result of the comparison of LVH groups, it was determined that patients with LVH had higher night time systolic blood pressure values and morning blood pressure surges. In the consequence of the correlation analysis, it was found out that there was a positive correlation between left ventricular mass index and night and 24-hour systolic blood pressure and morning blood pressure surge.
Conclusion: The morning blood pressure surge is a marker and independent predictor of hypertension in terms of cardiovascular incidents and target organ damage. In our study, we found the frequency of LVH to be significantly higher in patients with morning blood pressure surges.

Keywords: Hypertension; left ventricular hypertrophy; morning blood pressure surge

INTRODUCTION

Hypertension (HT) is a disease with a high global prevalence. HT is the most frequent and chronic prescribed disease in hospital admission reasons and the general frequency for population ranges between 25-35% (1-3).

Hypertension is a systemic disease, which manifests itself by continuous high blood pressure, and it is a major health problem since it can lead to problems such as stroke, myocardial infarction, heart failure, kidney failure and is common in the society.

Chronic or intermittent blood pressure elevation increases systemic pressure and causes volume load, and then the left ventricle (LV) workload increases and left ventricular hypertrophy (LVH) develops consecutively(4). LVH is a well-known risk factor for heart failure, myocardial infarction, arrhythmia, sudden cardiac death, and stroke (5-7).

Ambulatory blood pressure monitoring (ABPM) can be used in the diagnosis of HT. ABPM is more credible than the office blood pressure measurement with regard to target organ damage and reducing the risk of incident cardiovascular disease (CVD) (8).

Normal population studies have revealed that blood pressure shows a nocturnal decrease in adults (9). Circadian blood pressure changes cause cardiovascular structural and functional changes, increasing the risk of CVD. (10,11). Major cardiovascular system complications such as, myocardial infarction, arrhythmia, and sudden cardiac death and stroke usually occur early in the morning (12). It is known that increase in the blood pressure during night sleep and early morning also increases these complications (13). The rate of decline in night-time blood pressure can vary between the individuals, but in the great majority of the population it ranges between 10-20%. If the night-time decline is between these values, then it is

classified as dippers, while it is classified HT when the decline is below 10%.

Morning blood pressure surge (MBPS) is the physiological component of circadian or diurnal blood pressure variability. There are two most commonly accepted methods of calculation. The first calculation method is to subtract the lowest value of systolic blood pressure recorded during sleep from the systolic blood pressure value at the 2nd hour after awakening. The second method is to subtract the blood pressure value of after awakening from the average of last 4 systolic blood pressure values before awakening (14). We used the first of the above-mentioned calculation methods in our study.

The objective of our study is to reveal the relationship between LVH and MBPS in newly diagnosed hypertensive patients.

MATERIALS and METHODS

A total of 92 patients (60 females, 32 males) who applied to the Division of Internal Medicine Polyclinic of Ankara Kecioren Education and Research Hospital between July 2009 and February 2010 were included in our study. The criteria for inclusion in the study were over 18 years of age, agreeing to participate in the study, had not received medication for the last 3 weeks, even if he had not been diagnosed or diagnosed with HT before, having MPBS> 140 mmHg and / or dBP > 90 mmHg during the last two outpatient clinic examinations, and ABP assessment and left ventricular mass performed by appropriate method.

In our study, exclusion criteria were determined as; a history of secondary or malignant HT, symptoms and indications of heart failure, coronary artery disease, cerebrovascular disease sequel, atrial fibrillation, congenital valve disease, determined heart valve disease, malignancy, kidney failure, obstructive sleep apnea.

Office blood pressure measurement, kidney function tests, ABPM and left ventricular mass were computed for all patients.

Office blood pressure measurement of the patients was measured with mercury sphygmomanometer. Systolic and diastolic blood pressures were taken by the same person on two separate days with reference to the JNC-7 criteria.

24-hour ABPM was performed through portable, compact digital recorder (Travker NIBP2, Delmar Reynolds Ltd, Hertford, UK). Measurements were carried out during the daytime and sleep with the intervals of 15 minutes and 30 minutes respectively. In the data, 24-hour blood pressure, heart rate, mean arterial blood pressure, day and night-time periods were included in the statistics individually. In office scans, the patients with; MPBS> 140, dBP> 90, ABPM mean 24-hour MPBS> 130, dBP> 80, intraday mean MPBS> 135, dBP> 85, night-time average MPBS> 125, dBP> 75 HT, were considered as HT and included in the study. Night-time declines were classified as dippers HT,

if the decline is higher than %10 when compared with the day-time declines, while were classified as non-dippers if the decline is below 10%.

Morning blood pressure surge was computed with subtracting the lowest systolic blood pressure recorded during sleep from the systolic blood pressure value of the 2nd hour after awakening.

Echocardiography measurements were examined through using standard two-dimensional M Mode echocardiography device (Philips, 1.3-4.2 Mhz electronic probe, Los Angeles, CA, USA).by the same cardiologist in reference to the criteria of the American Echocardiography Association.

The LV mass (LVM) value was determined to be 1.04 x ((LVEDD) + İVST + PWT)3- (LVEDD)3)- 13.6 according to the Devereux formula. LV hypertrophy (LVH) development was defined as LVMI ≥ 134g/m^2 in male and LVMI≥ 110 g/m^2 in female.

Body mass index (BMI) = Body weight (kg.) / the square of the height (m.).

Ethics Committee
Our study was ethically approved by the Turkish Ministry of Health Ankara Kecioren Training and Research Hospital Local and Central Ethics committees on 30.06.2009 in accordance with the decision number of 2009/06/79.

Statistical Analysis
Obtained findings were *analyzed with statistically appropriate methods by using program of SPSS for Microsoft Windows 11.3 (SPSS Inc., Chicago, IL, USA. Kolmogorov-Smirnov* test was used for determining whether the data conformed to a normal distribution. Normally distributed results of data were expressed as mean ± standard deviation, nd non-normally distributed data were expressed as "median - interquartile range (IR)". The patients were divided into two groups as with and without LVH and were analyzed. Correlation analysis was conducted to all patients without dividing into gender groups. With regard to normal distribution state, either Pearson or Spearman correlation analyzes was preferred. Results for p<0.05 were considered statistically significant.

RESULTS

A total of 92 patients (60 female, 32 male) were included in the study. The mean age of the patients was 49.1 ± 13.7 (Mean ± SD). The mean BMI of the patients included in the study was 33.8 ± 9 and was within the limits considered as obesity. Upon examining the accompanying chronic diseases, DM was the most common with 15.2%. Demographic (age, gender, body mass index, additional diseases) and laboratory findings of all patients participating in the study are shown in Table 1. Upon examining the ABPM characteristics of the study group, 69 patients (75%) were found to be nondipper and the patients' office systolic blood pressure (OSBP) and office diastolic blood pressure (ODBP) was significantly higher

than the 24-hour mean systolic blood pressure (24SBP), and 24-hour mean diastolic blood pressure (24DBP). When the MBPS was computed, it was found to be 26.5 ± 16.9 and LVH was also observed for 29 patients (31.5%). These data are presented in Table 2.

Table 1. Demographic characteristics and biochemical data of the patients in the study group

	(n:92)
Gender (F/M)	60/32
Age (years)	49.1 ± 13.7
Body mass index (kg/m²)	33.8 ± 9.1
Diabetes mellitus	14 (15.2%)
Glucose (mg/dL)	103.4 ± 29.3
AST (U/L)	22.5 ± 9.5
ALT (U/L)	23.5 ± 16..6
GGT (U/L)	34.5 ± 20.5
ALP (U/L)	209 ± 68.1
Albumine (g/dl)	4.1 ± 0.32
Total Cholesterol (mg / dL)	193.1 ± 38.8
LDL (mg/dl)	121.6 ± 29.6
HDL, mg/dl	46.8 ± 13.5
Triglyceride, mg/dl	152.5 ± 76.7
Creatinine (mg/dl)	1.4 ± 0.8
Thyroid stimulating hormone (mIU / l)	2.0 ± 1.5
Hematocrit (%)	40.0 ± 5.5
Potassium (mmol / L)	4.3 ± 0.3

AST: Aspartate Aminotransferase ALT: Alanine Aminotransferase GGT: Gamma Glutamyltransferase ALP: Alkaline Phosphatase LDL: Low Density Lipoprotein HDL: High Density Lipoprotein

Table 2. ABPM characteristics of the study group

	(n:92)
NON DIPPER	69 (75%)
OFFICE MPBS	154.9 ± 12.8
OFFICE dBP	100.8 ± 10.2
ABPM 24 hours MPBS average (mmHg)	136.6 ± 14.2
ABPM 24 hours dBP average (mmHg)	83.4 ± 9.9
ABPM daytime MPBS average (mmHg)	138.6 ± 14.1
ABPM day-time dBP average (mmHg)	84.9 ± 9.7
ABPM night-time MPBS average (mmHg)	131.7 ± 16.3
ABPM night-time dBP average (mmHg)	78.9 ± 11.7
Morning Blood pressure surge (mmHg)	26.5 ± 16.9
Left ventricular hypertrophy	29 (31.5%)
Left ventricular mass	106.6 ± 24.7

ABPM: Ambulatory blood pressure monitoring, SBP: Systolic blood pressure, dBP: Diastolic blood pressure

The patients were divided into two groups with regard to LVH. Group 1 was identified as patients with LVH (n: 29) and group 2 was not without LVH (n: 63). While the mean left ventricular mass was 132.4 ± 20.9 in group 1, the mean left ventricular mass was calculated as 94.7 ± 15.6 in group 2 and was statistically higher in group 1 than group 2. (p <0.0001). It was observed that group 1 had higher systolic blood pressure values than group 2.

(p <0.03). Moreover, the mean MBPS was 32.1 ± 15.6 in group 1, while it was computed as 23.9 ± 16.9 in group 2 and was significantly higher in group 1. (p <0.03). These data are presented in Table 3. There was no difference between the groups in terms of biochemical and demographic data. In the consequence of the correlation analysis, it was found out that there was a positive correlation between left ventricular mass index and 24-hour systolic blood pressure (Figure 1) (r:.240, p < 0.02), left ventricular mass index and night-time systolic blood pressure (Figure 2) (r:.312, p < 0.002), left ventricular mass index and MBPS (Figure 3) (r:.212, p < 0.04).

Table 3. Comparison of patients with and without LVH in terms of ABPM features

	Patients with left ventricular hypertrophy n:29	Patients without left ventricular hypertrophy n:63	p value
Office MPBS	158.1 ± 14.4	153.4 ± 11.9	NS*
Office dBP	101.3 ± 10.2	100.5 ± 10.2	NS
ABPM 24 hours MPBS average (mmHg)	139.7 ± 15.4	135.2 ± 13.5	NS
ABPM 24 hours dBP average (mmHg)	82.8 ± 7.8	83.7 ± 10.8	NS
ABPM day-time SBP average (mmHg)	140.9 ± 15.3	137.6 ± 13.4	NS
ABPM day-time Dbp average (mmHg)	84.1 ± 8.3	85.4 ± 10.3	NS
ABPM night-time SBP average (mmHg)	137.6 ± 17.7	129.1 ± 14.9	0.03
ABPM night-time dBP average (mmHg)	79.0 ± 10.3	78.9 ± 12.4	NS
Left ventricular mass	132.4 ± 20.9	94.7 ± 15.6	0.0001
Morning blood pressure surge (mmHg)	32.1 ± 15.6	23.9 ± 16.9	0.03

*: not significant
ABPM: Ambulatory Blood Pressure Monitoring, SBP: Systolic Blood pressure, dBP: Diastolic Blood Pressure
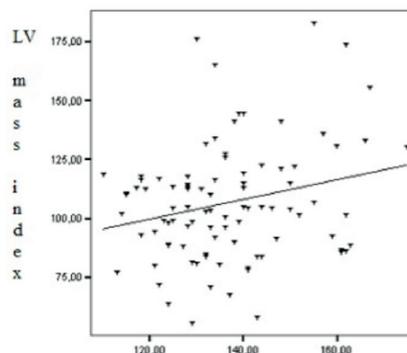
Figure 1. 24 hour Systolic Blood Pressure (mmhg)
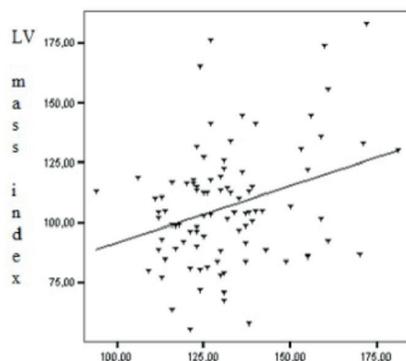
Figure 2. Night-time Systolic Blood Pressure (mmhg)
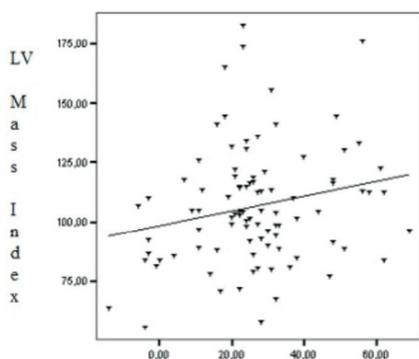
Figure 3. Morning Blood Pressure Surge (mmhg)

DISCUSSION

Hypertension is considered an important public health problem, regarding its frequency in the community and its negative effects on CVD formation. The impacts of HT on LV structural and functional remodeling are well known (15,16). The relationship between the risk of developing CVD and blood pressure is independent of other risk factors, and as the higher the blood pressure, the higher the risk of CVD (17). The prevalence of LVH is largely associated with the prevalence, chronicity and severity of HT.

There are an increasing number of studies revealing that cardiovascular incidents, particularly myocardial infarction, ischemic and hemorrhagic strokes develop more frequently in the morning hours (18). When considered from this point of view, ABPM may play a role in hindering target organ damage and cardiovascular incidents through putting forward the diurnal blood pressure pattern.

CONCLUSION

Morning blood pressure surge is a marker and independent predictors of HT in terms of cardiovascular incidents and target organ damage. In most studies, which involves morning blood pressure surge, a high correlation was found to be between MPBS and cardiovascular incidents (14).

In our study, it was intended to examine the potential relationships between ABPM and left ventricular mass measurements in newly diagnosed hypertensive patients. In order to realize this objective, data of 92 patients were evaluated. Those with LVH had higher night time systolic blood pressure and morning blood pressure surges than those without LVH. There was a significant relationship between 24-hour systolic blood pressure elevation and LVH. In our study, LVH was detected in 31.5% of newly diagnosed patients. Similarly, in a prospective study of 1033 patients with essential HT over 50 years old, LVH was detected in 29% of patients (19). In our study, the mean left ventricular mass of patients with LVH was 132 ± 20.9, while the left ventricular mass index was 125gr / m^2. Based on this, strict blood pressure control should be ensured to reduce the risk of LVH-related CVD. In another study, which revealed LVH decline in 387 patients, it was observed that the benefits for CVD were directly associated with the degree of decrease in left ventricular mass index (20). It plays a vital role in the progression of LVH by increasing blood pressure surge, cardiac afterload and arterial stiffness. In hypertensive patients, the increase in MBPS is correlated with an increase in the left ventricular mass index (21-22). In the Ohasama study conducted in 1430 patients (mean age: 61 years, 64% women), it was put revealed that MBPS was predictive in patients who developed cerebrovascular hemorrhage during the follow-up, which lasted 10.4 years (22).

Studies have put forward that this difference is more than 25 mmhg and every 10 mmhg rise gradually increases incidents CVD. In our study, MBPS was 32.1 ± 15.6 in patients with LVH, which is known to be the risk factor for CVD developing due to HT, whereas in patients without LVH, MPBS was found to be 23.9 ± 16.9 and the difference was statistically significant (23) .

In updated guidelines, the approach to HT is based on cardiovascular risk assessment, blood pressure level and target organ damage. In studies related to this, there are data indicating that blood pressure surge increases target organ damage. Particularly thanks to the developing digitalized non-invasive microsensor technologies, it will assist to get early diagnosis and appropriate treatment with HT and blood pressure variability diagnosis. More prospective studies are needed to evaluate blood pressure variability, in particular morning blood pressure surge, target organ damage and incidents cardiovascular.

REFERENCES

1. Muntner P, Carey RM, Gidding S, et al. Potential US Population Impact of the 2017 ACC/AHA High Blood Pressure Guideline. Circulation 2018;137:109.

2. Yoon SS, Gu Q, Nwankwo T, et al. Trends in blood pressure among adults with hypertension: United States, 2003 to 2012. Hypertension 2015;65:54.

3. Cherry DK, Burt CW, Woodwell DA. Advance data from vital and health statistics. No 337. Hyattsville, MD. National Center for Health Statistics, 2003.

4. Lang RM, Badano LP, Mor-Avi V, et al. Recommendations for cardiac chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr 2015; 28:1-39.

5. Levy D, Savage D, Garrison R, et al. Echocardiographic criteria for left ventricular hypertrophy: the Framingham Heart Study. Am J Cardiol 1987; 59:956-60.

6. Beverly L, Blase C. Left ventricular hypertrophy pathogenesis, detection, and prognosis. Circulation 2000;102:470-9.

7. Frohlich ED, Apstein C, Chobanian AV, et al. The heart in hypertension. N Engl J Med 1992; 327:998-1008.

8. Eguchi K, Pickering TG, Hoshide S, et al. Ambulatory blood pressure is a better marker than clinic blood pressure in predicting cardiovascular events in patients with/without type 2 diabetes. Am J Hypertens 2008;21:443-50.

9. Staessen JA, Bieniaszewski L, O'Brien E, et al. Hypertension 1997;29:30.

10. Cuspidi C, Sala C, Tadic M, et al. Non-Dipping pattern andsubclinical cardiac damage in untreated hypertension: A systematic review and meta-analysis of echocardiographic studies. Am J Hypertens 2015;28:1392-402.

11. Hermida RC, Ayala DE, Mojón A, et al. Blunted sleep-time relative blood pressure decline increases cardiovascular risk independent of blood pressure level--the "normotensive non-dipper" paradox. Chronobiol Int 2013;30:87-98.

12. Bath P, Algert C, Chapman N, et al. Collaborative Group PROGRESS. Association of mean platelet volume with risk of stroke among 3134 individuals with history of cerebrovascular disease. Stroke 2004;35:622-6.

13. Pickering TG, Shimbo D, Haas D. Ambulatory blood-pressure monitoring. N Engl J Med 2006;354:2368-74.

14. Kario K, Pickering TG, Umeda Y, et al. Morning surge in blood pressure as predictor of silent and clinical cerebrovascular disease in elderly hypertensives: a prospective study. Circulation 2003;107:1401-6.

15. Santos M, Shah AM. Alterations in cardiac structure and function in hypertension. Curr Hypertens Rep 2014;16:428.

16. Drazner MH. The progression of hypertensive heart disease. Circulation 2011;123:327-34.

17. Chobanian AV, Bakris GL, Black HR, et al. The seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. JAMA 2003;289:2560-72.

18. Maron BJ, Kogan J, Proschan MA, et al. Circadian variability in the occurrence of sudden cardiac death in patients with hypertrophic cardiomyopathy. J Am Coll Cardiol 1994;23:1405-9.

19. Verdecchia P, Carini G, Circo A, et al. Left ventricular mass and cardiovascular morbidity in essential hypertension: the MAVI study. J Am Coll Cardiol 2001;38:1829-35.

20. Pierdomenico SD, Lapenna D, Cuccurullo F. Regression of echocardiographic left ventricular hypertrophy after 2 years of therapy reduces cardiovascular risk in patients with essential hypertension. Am J Hypertens 2008;21:464.

21. Gosse P, Lasserre R, Minifie C, et al. Blood pressure surge on rising. J Hypertens 2004;22:1113-8.

22. Metoki H, Ohkubo T, Kikuya M, et al. Prognostic significance for stroke of a morning pressor surge and a nocturnal blood pressure decline: the Ohasama Study. Hypertension 2006;47:149-54.

23. Pierdomenico SD, Pierdomenico AM, Cuccurullo F. Morning blood pressure surge, dipping, and risk of ischemic stroke in elderly pa-tients treated for hypertension. Am J Hypertens 2014;27:564-70.