ORIGINAL RESEARCH ARTICLE

Self-Measured Blood Pressure and Target Organ Damage in Newly Detected Hypertension in South Gujarat, India

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ABSTRACT

Introduction: Blood pressure transient spikes have been considered to be noise and only a hindrance to a proper assessment of typical blood pressure, which is defined as the actual underlying average blood pressure over a long period of time. The current study aimed to see if the highest Self measured Systolic blood Pressure could be utilized to forecast the occurrence of Target organ damage and evaluate the independent association between the maximum Self measured Systolic blood Pressure and Target organ damage in individuals with untreated hypertension.

Method: We evaluated the urine albumin/creatinine ratio (UACR) and carotid intima-media thickness (IMT) using ultrasonography in 462 hypertensive individuals who had never taken treatment for their hypertension. Residential blood pressure was recorded.

Result: The maximal Self measured Systolic blood Pressure had considerably higher association coefficients with left ventricular mass index (LVMI) and carotid intima-media thickness than the mean Self measured Systolic blood Pressure. Irrespective of the mean Self measured Blood pressure level, multivariate regression studies showed that the maximal Self measured Systolic blood Pressure was independently related with left ventricular mass index and carotid intima-media thickness.

Conclusion: Transiently high blood pressure measurements recorded at Self measured shouldn't be dismissed as noise but rather taken seriously as significant warning signs of hypertensive Target organ damage in the heart and arteries.

Key words: Systolic blood pressure, Diastolic blood pressure, Left ventricular mass index, Carotid intima media thickness, Urine albumin/creatinine ratio

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INTRODUCTION

Transient spikes in blood pressure have been thought to be noise and just a barrier to accurate assessment of typical blood pressure, which is defined as the genuine underlying average blood pressure over an extended period of time. In this situation, such increases would produce a so-called "regression dilution bias," which would significantly underestimate the strength of the actual connection between normal blood pressure and cardiovascular risk.[1]

Recently, research by Rothwell et al. demonstrated that the highest recorded systolic blood pressure (SBP) in an office setting was a reliable predictor of cardiovascular events, regardless of the mean SBP during a period of 12 to 36 months.[2] Additionally, Ko et al. demonstrated that the onset of brain haemorrhagic transformation was substantially correlated with the maximal SBP during the initial 72 hours of acute ischemic stroke, regardless of the mean SBP level.[3] As a result, those with high blood pressure episodes may have a significant risk of cardiovascular disease.

The fact that reaching the highest SBP needs numerous office visits over time is a disadvantage of using it in normal clinical therapy of hypertension. Observing the highest SBP obtained through at-Self measured is one potential solution, since this allows for the collection of several BP readings in a brief amount of time under well controlled settings.

Additionally, there is a growing body of information showing that Self measured blood pressure is more closely associated to target organ damage.[4-9] indicating that office BP may not be the best predictor of cardiovascular events.[10,11] Therefore, compared to office BP measurement, Self-measured BP measurement can deliver more accurate data on maximal BP in a relatively short amount of time. The importance of maximum Self measured blood pressure in relation to TOD or cardiovascular events, however, has not been studied.

The mean Self measured SBP, which has been categorically proven to be a predictor of TOD and a driver of cardiovascular prognosis, may not accurately reflect the severity of TOD, according to our hypothesis. The objective of the current study was to evaluate the independent relationship between the maximum Self measured SBP and TOD in individuals with untreated hypertension and to see if the highest Self measured SBP may help with TOD presence prediction.

In this investigation, we assessed renal, cardiac, and vascular damage using the urine albumin/creatinine ratio (UACR), carotid intima-media thickness (IMT), and left ventricular mass index (LVMI). All three have been identified as indicators for TOD with subclinical hypertension.[12] independently derived, and predictive of future cardiovascular events.[13-15]

METHODOLOGY

Research Subjects: This study was conducted in SMIMER hospital, Surat, India. From July-2021 to Dec 2022, all the patients with untreated hypertension visiting outpatient's department of General Medicine were sequentially enrolled. Cases who fulfilled the inclusion criteria—an average SBP of at least 140 mm Hg and/or a diastolic blood pressure (DBP) of at least 90 mm Hg or both on two different occasions were included.

Furthermore, cases were excluded who had severe cardiovascular illness or arrhythmia or heart failure or stroke or history of coronary artery disease or renal insufficiency (serum creatinine >2 mg/dL) or mental problems, or secondary hypertension or chronic inflammatory disease.

Those patients who were following inclusion criteria undergone a medical check-up, anthropometric measures, blood and urine tests, and ultrasonography at the time of enrolment which was considered as the baseline measurement. Written informed permission was acquired from each participant, and this study was approved by the institutional ethics committee of the SMIMER Hospital. All instruments used for blood pressure measurement were calibrated regularly throughout study period.

Basic meaning of BP Indices: This method was used to calculate the maximum Self measured blood pressure. Average morning and evening blood pressure readings were taken in triplicate each day. The maximum Self measured Blood pressure was determined by taking the highest average out of these for each individual. The mean Self measured BP was calculated by averaging all of the values for each individual. The difference between the maximum and mean Self measured SBP is what researchers refer to as the "peak size in Self measured SBP." The daily Self measured Blood pressure variability was defined as the SD of each individual's Self measured BP (average of morning and evening bps).

Carotid ultrasound and echocardiography: A high-resolution B-mode ultrasound scanner was used for the ultrasound studies. LVM was calculated using an anatomically proven formula employing M-mode echocardiography, guided by 2-dimensional echocardiography, and end diastolic dimensions. From the LVM divided by the body surface area, the LVMI was derived. Left ventricular hypertrophy (LVH) was indicated by an LVMI of less than 125 g/m2 in men and less than 110 g/m2 in women. Carotid IMT was measured at three locations close to the bilateral carotid bulbus (far wall) in 10-mm segments at the end of diastole, always in segments free of plaque. If a plaque was present at the IMT measurement site, an acceptable neighbouring part was selected. The studies were conducted using the mean of the right and left carotid IMT, which was 6 points overall. Diffuse common carotid artery thickening,

indicated by an average IMT >0.9 mm, was used to diagnose carotid atherosclerosis.

Albumin Excretion in the Urine: Fasting blood and urine samples were taken in the morning. A spot urine sample was used to assess the urinary albumin level, which was then expressed as UACR (mg/gCr) using a turbidimetric immunoassay. Enzymatic assay was used to quantify the creatinine in urine. UACR 22 mg/gCr in males and 31 mg/gCr in women was used to identify albuminuria.

Statistical Analyses: All collected data were entered in Microsoft excel sheet, data presented in percentage, mean and standard deviation and compared by appropriate test.

RESULTS

In this study, we discovered 493 new cases of hypertension throughout the study period. Out of these, 17 cases were refused permission for a follow-up period, and 14 cases were moved to a different city. So, 462 newly discovered hypertensive patients were used as the basis for our data analysis.

Table 1 show that demographic and clinical variables mean age was 67.7 years, while male contribute 61% of total population, furthermore mean BMI was 24.1 kg/m^2

Table 1: Clinico-demographic profile of cases included in the study

Variables	Cases (n=462)
Demographic variables	
Age (Years)	67.7±12.0
Male sex %	61
Body mass index, kg/m^2	24.1±4.1
Waist circumference, cm	83.2±9.9
Hypertension duration, years	5.2±6.9
Smoking, %	20.76
Habitual drinking, %	36.33
Diabetes mellitus, %	11.67
Hyperlipidaemia, %	40
Clinical variables	
Mean SBP, mm Hg	141.4±17.7
Mean DBP, mm Hg	78.6±10.2
Maximum SBP, mm Hg	164.3±21.1
Maximum DBP, mm Hg	91.1±11.7
baPWV, m/sec	18.2±4.2
Target organ damage	
LVMI, g/m^2	117.3±34.1
Proportion of LVH, %	51
Carotid IMT, mm	0.89±0.19
Proportion of CAS, %	48
UACR, mg/g Cr	23.0 (19.5–25.0)
Prevalence of albuminuria, %	42

SBP – Systolic blood pressure (mm Hg);
DBP – Diastolic blood pressure (mm Hg)
baPWV -Brachial ankle pulse wave velocity (m/sec);
LVMI – Left ventricular mass index (g/m^2);
LVH - left ventricular hypertrophy
IMT – Intima media thickness (mm);
CAS - carotid atherosclerosis;
UACR – Urinary albumin (mg)/creatinine ratio (g) (mg/gCr)

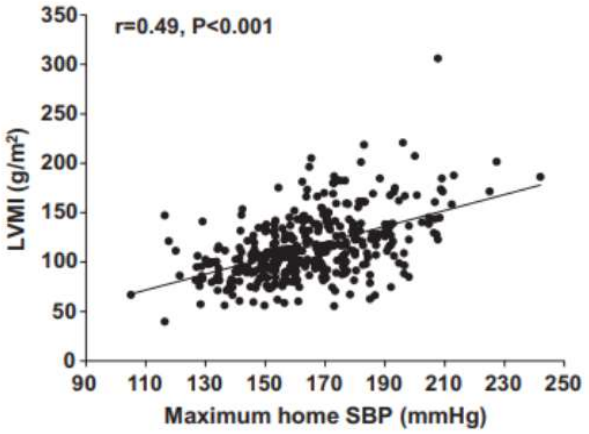

Figure 1: Univariate correlation between the maximum Self measured SBP and left ventricular mass index

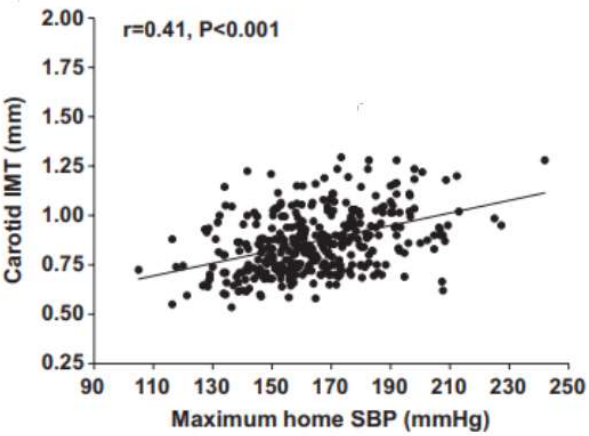

Figure 2: Univariate correlation between the maximum Self measured SBP and carotid intima media thickness

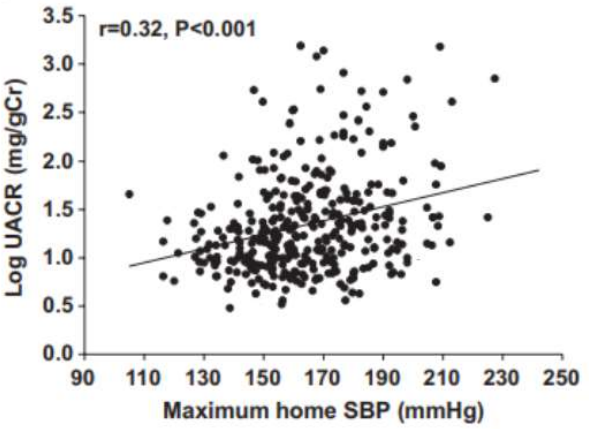

Figure 3: Univariate correlation between the maximum Self measured SBP and Log UACR (mg/gcr)

Table 2: Univariate correlations between bps and measures of target organ damage

TOD Variables	P/r value	Mean SMSBP	Mean SMDBP	Maximum SMSBP	Maximum SMDBP
LVMI	**r**	0.49	0.13	0.53	0.23
	P	<0.001	0.02	<0.001	<0.001
Carotid IMT	**r**	0.41	0.09	0.43	0.13
	P	<0.001	0.1	<0.001	0.012
Log UACR	**r**	0.32	0.08	0.27	0.08
	P	<0.001	0.15	<0.001	0.16

TOD – Target Organ Damage; SMSBP-Self measured systolic blood pressure; SMDBP-Self measured diastolic blood pressure; LVMI – Left ventricular mass index (g/m^2); IMT – Intima media thickness (mm); UACR – Urinary albumin (mg)/creatinine ratio (g) (mg/gCr)

Table 3: Maximum SBP and Target Organ Damage in the Total Population and Subgroups Divided by Mean BP Levels: Multivariate Regression Analysis

Dependent Variable	Total Population (n=462)			Mean BP <135/85 (n=175)			Mean BP > 135/85 (n = 287)		
	β (SE)	*P*	R^2	β (SE)	*P*	R^2	β (SE)	*P*	R^2
LVMI*, g/m^2									
Maximum SBP	0.593 (0.091)	<0.001	0.29	0.510 (0.185)	0.006	0.23	0.657 (0.147)	<0.001	0.21
Carotid IMT†, mm									
Maximum SBP	0.002 (<0.001)	<0.001	0.25	0.002 (0.001)	0.006	0.27	0.002 (0.001)	<.001	0.26
Log UACR‡, mg/gCr									
Maximum SBP,	0.003	0.02	0.19	0.002 (0.002)	0.65	0.41	0.002 (0.001)	0.19	0.16

This model was adjusted by age, sex, habitual drinking, and mean SBP.
This model was adjusted by age, sex, hypertension duration, smoking, diabetes mellitus, and mean SBP. This model was adjusted by age, sex, diabetes mellitus, and mean SBP.
β indicates partial regression coefficient; R 2, multiple coefficients of determination; LVMI, left ventricular mass index; IMT, intima-media thickness; UACR, urinary albumin/creatinine ratio; SBP, systolic blood pressure

In clinical variables mean SBP was 141.4±17.7 mm Hg while mean DBP was 78.6±10.2 mm Hg, Maximum SBP was 164.3±21.1 mm Hg, Maximum DBP was 91.1±11.7 mm Hg, while ba PWV, mean was 18.2±4.2 m/sec.

Furthermore, target organ damage LVMI mean was 117.3±34.1 g/m^2, while prevalence of left ventricular hypertrophy was noted in 51% of cases. While Carotid IMT mean 0.89±0.19 mm, Prevalence of carotid atherosclerosis was noted in 48% of cases. While UACR 23 mg/gCr, furthermore Prevalence of albuminuria was noted in 42% of total cases.

Table 2 show that the maximal Self measured SBP was strongly and linearly linked with LVMI and carotid IMT in univariate analysis, and all SBP measurements were substantially connected with all TODs. The univariate correlations between Self measured BP indices and TODs remained the same. The correlation coefficients were substantially different for LVMI (0.49 against 0.53, P=0.008) and carotid IMT (0.41 versus 0.43, P = 0.001) but not for UACR (0.32 versus 0.27, P=0.16).

Table 3 show that the results of the multivariate linear regression analyses are shown in the table, which was divided by the mean Self measured blood pressure level of 135/85 mm Hg to assess the independent connection of the highest Self measured SBP with TODs in the entire population and subgroups. In none of the models we discovered any multicollinearity. Even after adjusting for important variables, the maximal Self measured SBP remained independently linked with the LVMI and carotid IMT in all groups. The highest Self measured SBP, on the other hand, was independently linked with the UACR in the entire population but not in either subgroup.

Discussion

The highest SBP was independently linked with LVMI and carotid IMT, even when the mean blood pressure level was normal; and third for the occurrence of LVH and carotid atherosclerosis. These were the key findings of this investigation. This is the first study to show how the maximal SBP in people with untreated hypertension affects clinical outcomes.

Although earlier studies suggested that averaging additional measures would increase the accuracy of Self measured blood pressure as a predictor of cardiovascular disease, the current study showed that the highest SBP value was more closely connected with LVMI and carotid IMT than the mean SBP.[4,10] In the current investigation, the first day was when the maximum Self measured SBP was most frequently recorded. The strong correlations between the maximal Self measured SBP and LVMI/carotid IMT persisted even when day 1 blood pressure data were excluded.

Even though maximum Self measured SBP is distinct from BP variability in that it only pertains to brief fluctuations in BP, these two variables frequently show comparable relationships with other clinical indicators, such as arterial stiffness and baroreceptor dysfunction.[1,17,18] Age and PWV, two factors that may contribute to the deterioration of the arterial baroreflex, were independent predic-

tors of the magnitude of the maximum Self measured SBP in the current investigation.[21,22]

Consequently, a baroreceptor malfunction may play a role in the processes underlying larger-value maximal Self measured SBP. The tight correlations between maximal Self measured SBP and cardiac/vascular damage may be explained by the fact that a baroreceptor malfunction itself has been linked to LVH and carotid atherosclerosis.[22,23]

It is still unknown if the maximal Self measured SBP contributes to the pathogenesis of TOD or if it is only a by-product of subclinical vascular damage. The current findings demonstrated that information concerning possible kidney injury inherent in the mean Self measured SBP level was not improved by the maximum Self measured SBP. This conclusion may be related to other studies that found a modest correlation between awake SBP variability and albuminuria as opposed to substantial correlations between awake SBP variability and LVMI or carotid IMT.[17,24]

In addition, despite a substantial association between SBP variability and LVMI, Lantelme et al. discovered that there was no significant correlation between baroreceptor sensitivity and albuminuria.[22] Renal autoregulation has been shown to mitigate glomerular damage by preventing the transmission of acutely increased BP fluctuations to the glomerular capillary circulation in an experimental research employing the rat kidney model.[25] These data suggest that, in the presence of normally operating autoregulatory systems, a brief rise in blood pressure may not have any negative effects on the kidney.

Age and regular drinking were associated with patients in the current research who had higher peak Self measured SBP levels. These findings are in line with earlier research, which showed that ageing and binge drinking were important predictors of Self measured SBP variability, defined as either the fluctuation between morning and evening. SBP values, also known as daily SBP values. Age-related declines in baroreceptor sensitivity have been recorded, and this mechanism can account for elderly people's temporary BP rises.[16,19,20]

We have noted that frequent nighttime alcohol use raises morning blood pressure and lowers late-night blood pressure.[26] This biphasic action may raise the morning maximum Self measured BP level but not the mean Self measured BP level, resulting in a higher level of peak size in the Self measured SBP.[26] Even after adjusting for confounders like mean Self measured SBP, Self-measured SBP variability was a significant predictor of LVMI in the current research. This is the first investigation to show a direct relationship between TOD and daily Self measured SBP fluctuation. On the other hand, daily fluctuation of the Self measured SBP was not a significant factor in carotid IMT. Our findings imply that transitory BP fluctuations (BP instability) may be more strongly linked to carotid atherosclerosis than daily BP variability, which represents more usual oscillations.

Limitations

As a result of the cross-sectional aspect of the current investigation, it is still unclear in what direction the cause-and-effect relationship between the highest Self measured SBP and TODs operates. Second, whereas the highest office SBP was repeatable and not a random occurrence, the maximum Self measured SBP's repeatability is uncertain.

Conclusion

According to our study, transiently high blood pressure measurements recorded at Self measured shouldn't be dismissed as noise but rather taken seriously as significant warning signs of hypertensive TOD in the heart and arteries. These results further highlight the need of monitoring maximal Self measured SBP severity together with mean Self measured BP levels during the first assessment of hypertension. To investigate the physiological causes of inflated Self measured BP swings and to prospectively evaluate the clinical ramifications of this BP phenomenon, more research is required.

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