REVIEW ARTICLE

NOCTURNAL BLOOD PRESSURE ASSESSMENT, AN ENTITY OF HIGH PROGNOSTIC VALUE, NOT UTILIZED TO ITS POTENTIAL IN CLINICAL PRACTICE

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Abstract: Hypertension, a disease of epidemic proportion, is assessed by measuring blood pressure (BP). Various methods are employed, the most common being "clinic blood pressure measurement (CBPM)." However, environment and technical errors can confound it. Other methods include home blood pressure measurement (HBPM), which shows readings of awake time only, and 24-hour ambulatory blood pressure monitoring (ABPM). A recent technique that causes less sleep disturbance is "timed HBPM." Blood pressure is a 24-hour phenomenon, and prognostically night time blood pressure, being more important, must be assessed properly. Besides providing the precise mean of 24-hour blood pressure, it extends knowledge regarding many other parameters of clinical importance like dipping pattern and morning surge etc. For nocturnal BP assessment, 24-hour ABPM and time-triggered HBPM are utilized. The importance of nocturnal blood pressure assessment for diagnostic and prognostic evaluation of various cardiovascular and non-cardiovascular conditions would be highlighted in this review article.

Keywords: Nocturnal hypertension, HBPM, ABPM, time triggered HBPM, hypertension phenotypes

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INTRODUCTION

Hypertension is a disease of epidemic proportion with an estimated 1.27 billion people affected globally, and around 1.52 billion people expected to have it by 2025.[1]

The cornerstone method for its diagnosis is measurement of BP, in a clinic or hospital usually. This CBPM is fraught with many fallacies regarding the technique and the patient's response so that actual readings may differ considerably from the measured ones. HBPM is another technique in which 14 measurements are recorded at home. The readings of the last six days are averaged to get a mean value but, HBPM doesn't give any hint about nighttime BP. By ABPM, a 24-hour assessment of BP is obtained. It provides the mean BP (along with variability) of entire day, and other phenotypes of BP. To reduce disturbance during sleep, a time triggered HBPM device has been introduced recently.

Assessment of nighttime BP is of immense value regarding the diagnosis and prognosis in various cardiovascular diseases (CVD). Normally, night BP decreases by 10-20% as compared to daytime BP, a phenomenon called "dipping," and there are many patterns of it (as detailed below). Evaluation of the rise in BP as one comes out of sleep known as "morning surge" (normally < 20 mmHg) has also shown prognostic value.

Nighttime BP is less affected by climate change as compared to daytime BP and has been regarded as the most reproducible phenotype of BP.[2]

Nocturnal hypertension can lead to adverse cardiovascular (stroke, ischemic heart disease, and peripheral arterial diseases) and non-cardiovascular events (kidney failure and cognitive dysfunction) although the patient may still be labelled "normotensive" due to its non-detection by routine clinical assessment.[3]

Nocturnal systolic BP predicts end points more accurately than awake BP. A 10% increase in night-to-day ratio enhanced all CV end points. For every 10-mmHg escalation in mean nighttime BP the mortality risk increases by 21%.[4]

Association of nocturnal BP parameters with cardiovascular outcomes is more significant than with office BP. This has been demonstrated by a sub-analysis of 6083 off-treatment patients of the Australian National Blood Pressure 2 study (ANBP2 study) in which 702 patients were analyzed by 24-hour ABPM. The study showed that nocturnal BP (HR 1.26; 95% CI 1.10–1.45, p= 0.001) and nocturnal pulse pressure (HR 1.18; 1.06–1.31, p = 0.003) were the

most significant parameters for 11-year cardiovascular mortality. Similar findings were observed for all-cause mortality.[5]

Thus, the importance of the assessment of nocturnal BP regarding mean (and variability) along with many other parameters (phenotypes) like dipping pattern, morning surge, isolated nocturnal hypertension, and morning hypertension etc. is quite clear. In this regard, two methods are currently available viz. 24-hour ABPM (easily available), and timed-HBPM (not widely available). This review article will highlight the acquisition of various nocturnal BP parameters by these methods and elaborate the diagnostic and prognostic importance of each in various cardiovascular and non-cardiovascular states.

METHODS

A literature survey of PUBMED, Google Scholar and PakMediaNet was done with search terms, blood pressure, nocturnal hypertension, prognosis, HBPM, ABPM, and hypertension phenotypes which fetched 85 results. Full text of all articles were evaluated for authenticity of data. By manual evaluation 48 were found pertinent to the topic (describing either the definition of various terms or results regarding diagnosis and prognosis of nocturnal hypertension) from which the material for this article has been derived.

TEXT

The description of "nocturnal hypertension" starts from physiological understanding of blood pressure variation and the various factors which cause it. Methods for its elaboration and the assessment of its phenotypes are explained next. Thirdly, the prognostic importance regarding cardiovascular diseases and target organ damage have been elucidated. Lastly, the association with certain non-cardiac conditions and impact of nocturnal hypertension on treatment has been emphasized.

FACTORS CAUSING DIURNAL VARIATION IN BLOOD PRESSURE AND NOCTURNAL HYPERTENSION

Like other physiological parameters, BP also shows diurnal variation in a way that two peaks are noted. The first peak occurs at the time of rising from bed (morning surge) and the second is noted in the evening. During sleep, BP goes down and reaches a trough one to two hours before rising again. This fall in night BP is about 10-20% of the daytime BP and represents the basal BP. External and internal factors are responsible for this circadian variation in BP. Smolensky et al. elaborated several factors responsible for day-night variation in BP by mentioning: "Nyctohemeral cycles of ambient temperature, light, noise and behaviorally driven food, liquid, salt, and stimulant consumption, mental/emotional stress, posture, and physical activity intensity plus circadian rhythms of wake/sleep, pineal gland melatonin synthesis, autonomic and central nervous, hypothalamic-pituitary-adrenal, hypothalamic-pituitary-thyroid, renin-angiotensin aldosterone, renal hemodynamic, endothelial, vasoactive peptide, and opioid systems constitute the key regulators and determinants of the 24h BP profile. Environmental and behavioral cycles are believed to be far more influential than circadian ones".[6]

Different mechanisms have been postulated for nocturnal hypertension and its prevalence varies from 30 to 60%. Increasing age, diabetes, obesity, hypercalcemia, hyperuricemia, homocystinemia, increased levels of serum Creatinine and Cystatin along-with sleep apnea syndrome enhance the likelihood of nocturnal hypertension.[7] Salt intake, salt sensitivity and Asian origin also increase the chances of nocturnal hypertension.

It is quite clear that multiple factors regulate nocturnal BP in the same way as they modulate daytime BP. As these factors show diurnal variation themselves hence their effect follows this trend. Prevalence of nocturnal hypertension in different populations also varies as per the different prevalence of these factors across the world.

MEASUREMENT OF NOCTURNAL BLOOD PRESSURE

Routine clinic, and home BP measurements cannot indicate the parameters of nocturnal blood pressure. 24-hour ABPM and time triggered HBPM are utilized for this purpose.

ABPM, first devised by Hinman et al. in 1962 is regarded as the "gold standard" for diagnosis, and subsequent evaluation of BP control in patients on anti-hypertensive medications.[8,9] ABPM is programmed to obtain at least 20, daytime and 7 nighttime readings. At least 70% valid readings should be obtained for analysis.[10] Sleep and awake times are marked.

Repeated BP measurements at night by ABPM disturbs sleep, rendering BP readings other than actual. It has been found by Yang et al. in their analysis of the IDACO study that 3-4 nighttime BP measurements are sufficient for analysis of nocturnal parameters.[11] Jaegar et al. calculated the diagnostic accuracy (i.e., Kappa statistic) of 2-4 readings from 74

sampling approaches of ABPM records. Sampling at 1, 2, 4, and 5 hours after falling asleep showed the highest kappa statistic of 0.84.[12]

Kollias et al used an HBP measuring device which is so timed that only three readings per night are obtained at hourly intervals for three consecutive nights, so that a minimum of six readings are obtained and used for analysis. Correlation with 24-hour ABPM measurements and with pre-clinical target organ damage was calculated. A good correlation was found for detection of nocturnal hypertension, non-dipper status, and pre-clinical target organ damage.[13] Mokwatsi GG et al. in their study of 1005 patients of J-HOP (Japan Morning Surge-Home Blood Pressure) trial compared a timer equipped home BP measuring device with 24-hour ABPM. Only systolic BP with a cutoff of 120 mmHg was compared for future CV events with a follow-up period of 7.6±3.4 years. Time triggered HBPM detected 564 (56.1%) whereas, ABPM detected 469 (46.7%) subjects to have nocturnal hypertension. Total CV events and stroke occurred significantly more often in hypertensive group detected by HBPM (CV event HR 1.78 95% CI 1.00–3.15, p = 0.001: stroke HR 2.65 95% CI 1.14–6.20, p = 0.002). This indicates the superiority of time triggered HBPM for detection of nocturnal hypertension and subsequent CV events.[14]

Although multiple robust studies have been done especially in Far east countries with newer less disturbing devices for BP measurement but a standard protocol from reliable guidelines forwarding authority is still awaited. In fact, there is a difference in marking the day and night time as some of them use the subject's rising and sleep time whereas others have used fixed timings. The impact of this discrepancy needs to be explored. Furthermore, the duration and number of measurements are not uniform till the time of this writing.

Other devices for nighttime BP assessment under investigation are: cuffless BP monitors (using pulse transit time) and wearable surge BP monitors (wrist-type tonometry devices) which are not only light-weight and cuff-inflation is also not disturbing but, their validity remains to be determined.

PHENOTYPES OF BLOOD PRESSURE OBTAINED BY MEASURING NOCTURNAL BLOOD PRESSURE

Assessment of nocturnal BP includes the usual parameters which are obtained by daytime BP such as mean BP along with standard deviation (SD) and co-efficient of variability but certain other notable features which have great clinical relevance regarding diagnosis and prognosis in hypertensive patients are dipping pattern, isolated nocturnal hypertension, and morning surge. Furthermore, clues are obtained for modification of treatment regime so that control of BP spans the entire twenty-four hours. These features are:

Mean BP: All the leading guidelines on hypertension agree that the cut-points for the diagnosis of nocturnal hypertension are systolic > 120 mmHg and diastolic > 70 mmHg except that forwarded by American heart association (AHA)/American college of cardiology (ACC). Their cut points are 110 mmHg for systolic and 65 mmHg for diastolic blood pressure.[15] This difference follows the same trend like the classification of BP and hypertension by various guidelines. A bit of controversy can be created until it is clearly mentioned in the report of 24-hour ABPM which guidelines have been followed. The impact of these differing cut-points also need to be explored.

For outcome prediction nocturnal BP is superior to daytime BP has been demonstrated in certain populations and conditions and clinic BP has the least value in this regard.[16]

Blood pressure variability: The difference in BP recorded at different time intervals represents variability. It could be short term, intermediate term and long-term (visit-to-visit).

Variability is represented by the standard deviation (SD) of recorded BPs and the co-efficient of variation (CV). SD and CV are affected by the stiffness of large arteries (atherosclerosis), the activity of vasomotor center, autonomic system activity, baroreflexes and treatment adherence.[17]

From ABPM study short term BP variability can be calculated by the SD of individual readings. As the diurnal BP variability is affected mostly by nocturnal BP the SD is corrected by the following formula:

$$\text{Weighted 24-hour SD} = \{(\text{daytime SD} \times \text{hour of daytime}) + (\text{nighttime SD} \times \text{hour of nighttime})\} / (\text{hour of daytime} + \text{hour of nighttime})$$

Strong prognostic influence of BP variability determined by SD of nocturnal BP has been observed in clinical studies. The Ohasama study group noted a significant association of nighttime SD (corrected by nighttime BP) with carotid plaque.[18] In a meta-analysis of 6 studies (7112 participants) for all-cause and cardiovascular mortality along with cardiovascular events SD of nighttime BP was found to be an independent prognostic marker.[19] 744 patients studied in the Syst-Eur trial showed that variability in nocturnal SBP carries more significance than variability in daytime BP as assessed by standard

deviation (SD) of BP measurements. Stroke risk increased by 80% CI 17-176%, for every 5 mmHg increase in the SD of nocturnal SBP.[20]

Another more robust parameter of variability is coefficient of variation, derived from SD. This indicates the dispersion of data around mean and is better suited to compare one data set from another even though the means are vastly different. Due to less interference by bodily and emotional activities nighttime BP shows less CV as compared to daytime CV. It is calculated as follows:

$$\mathrm{CV} = \frac{Standard\ Deviation\ of\ blood\ pressure\ (SD)}{Mean\ blood\ pressure}\ X\ 100$$

Average real variability (ARV), another parameter of variability is calculated as the mean difference (absolute) between adjacent BP measurements. This indicates the true variability of BP measurements recorded by reducing the signal noise of BP recordings. It is calculated by the formula:

$$\mathrm{ARV} = \frac{1}{N-1}\sum_{k=l}^{N-1} |\mathrm{BP}_{k+l} - \mathrm{BP}_k|$$

Where N denotes the number of valid BP readings by ABPM.

Diurnal variation in BP assessed by measuring dipping. Dipping is defined as decrease in mean nocturnal BP as compared to daytime BP. Normally it ranges from 10-20% and is calculated as follows:

$$\text{Dipping (\%)} = 1 - \frac{\text{Mean nocturnal systolic BP}}{\text{Mean daytime systolic/ BP}}\ \text{X } 100$$

The same formula is used for calculation of diastolic BP dipping. Four patterns of dipping are recognized, Figure 1: normal (10-20%, ratio 0.8-0.9), non-dipper (0-10%, ratio 0.9-1.0), excessive dipper (> 20%, ratio <0.8), and riser (<0%, ratio > 1.0).[21]

The dipping status, depends "on environmental (season, temperature, etc.) and genetic cues, daytime activity and stress, sleep quality, timing of intake and duration of action of antihypertensive drugs, position of the arm relative to the heart, nocturnal enuresis, differences in the cardiovascular risk profile, and many other factors".[22] Brein (1988) coined the term non-dipper when he noted that 20% of hypertensive patients don't drop their BP normally at night and there is an increased risk of stroke in such patients as compared to dippers (23.8% vs. 2.9%).[23] A non-dipper status has been found to increase several cardiovascular morbidities like left ventricular hypertrophy, atrial fibrillation, chronic kidney disease, and stroke. Even non-dipper normotensives show an enhanced risk of cardiovascular diseases.[2]

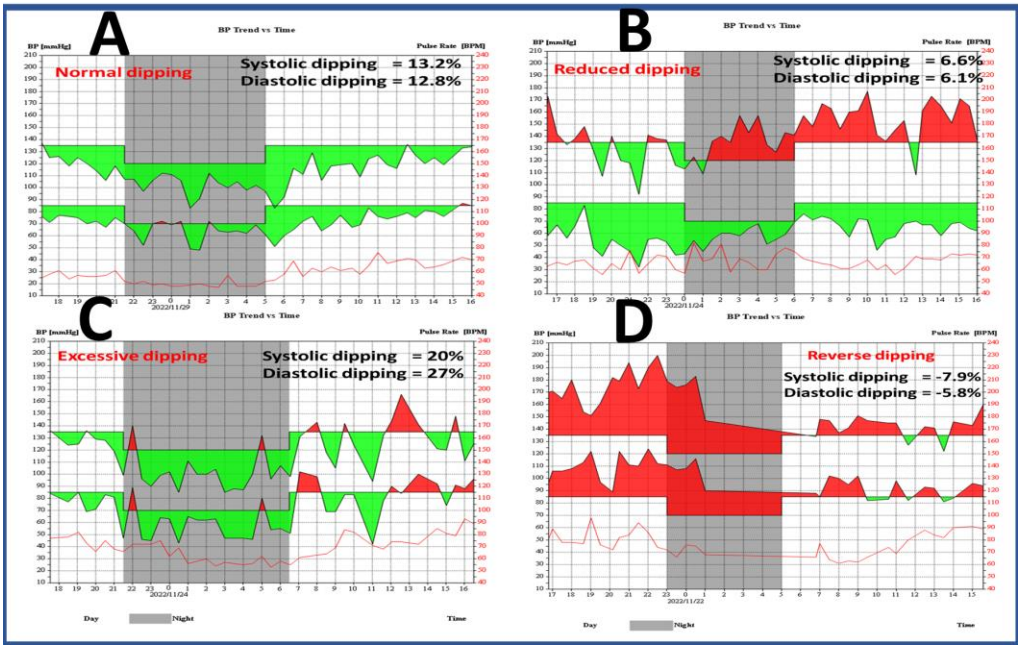

Figure 1: 24- Hour ABPM records showing the four dipping patterns: A- normal, B- reduced, C- excessive, and D- reverse dipping. Time is shown on X-axis and BP on Y-axis. Readings on white background are daytime whereas on gray background are nighttime. Green color indicates normal BP whereas red shows high BP measurements

Association of different dipping patterns with cardiovascular events is not the same. Lo L et al. assessed 1199 hypertensive subjects with 24-hour ABPM for dipper status and cardiovascular outcomes. Survival was inferior in non-dippers and reverse dippers for total cardiovascular events (HR 1.320 95% CI 0.814-2.141) and coronary events (HR 1.476 95% CI 0.783- 2.784) but not for cerebrovascular events.[25] Riser pattern of nocturnal BP is associated with adverse effect, so much that every 10 mmHg rise in mean nocturnal BP is associated with 21% increased risk of mortality.[26]

Isolated nocturnal hypertension (INH):

INH is noted in 7% of hypertensive patients and can be diagnosed only by assessment of nocturnal BP, Figure 2. Salazar MR et al. studied 1344 individuals to detect the prevalence of INH and found that amongst the phenotypes of masked hypertension nocturnal hypertension was the most common and one-third of these patients had INH.[27]

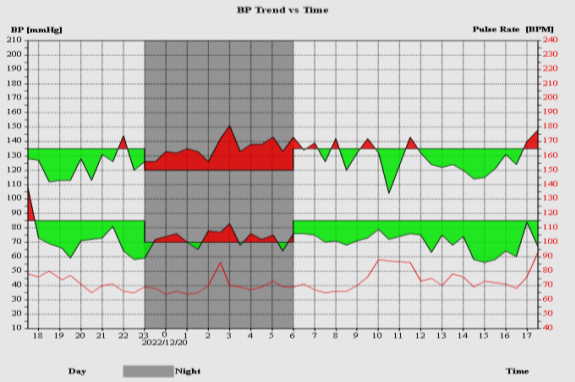

Figure 2: Isolated nocturnal hypertension. Day time and nocturnal BP records are seen with light and dark gray backgrounds respectively

Association of INH with target organ damage (TOD) has been studied. Kim SH et al. evaluated 1734 patients with 24-hour ABPM dividing them into three groups of normotension, INH, and diurnal hypertension. They compared the three groups regarding TOD for "arterial stiffness, left ventricular mass, left ventricular diastolic dysfunction and cerebrovascular insufficiency". 18.1% of the subjects had INH, mostly diastolic (71.3%). The occurrence of higher pulse wave velocity ($p<0.001$), higher central systolic pressure ($p<0.001$), higher left ventricular mass index ($p=0.026$) and worse diastolic dysfunction ($p<0.001$) was seen significantly more commonly in INH group after multivariable analysis.[28]

Isolated nocturnal hypertension is a type of masked hypertension and like later its elaboration is very important for management of patient. This can only be done by ABPM hence, every patient must be evaluated for this phenotype.

Morning surge assessment:

A rise in BP is noted as an individual leaves the bed and starts usual daily chores. The nadir of BP (an average of 3 readings centered around the lowest nighttime BP) compared with the average BP readings of the first hour of morning tells the 'morning surge' in BP, Figure 3.

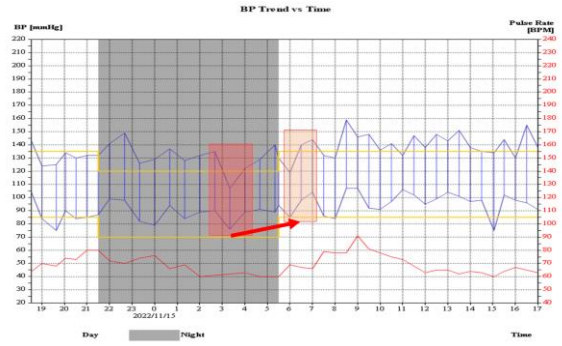

Figure 3: Morning surge (red arrow), lowest nighttime BP (pink rectangle) and mean BP of first hour of morning (light pink rectangle). Day time and nocturnal BP records are seen with light and dark gray backgrounds respectively

Asian population has higher morning surge with resultant increased incidence of stroke (salt sensitivity also plays part) than westerners.[29] Every 10 mmHg increase in morning surge (normal < 20 mmHg) is associated with a 22% enhanced risk of stroke (ischemic). Pierdomenico et al. in a study of 391 patients (with normal 24-hour BP on ABPM record) followed for 9.3 ± 4.6 years highlighted the role of dipping pattern and morning surge. They found that as "compared to dippers with normal morning surge, dippers with high morning surge (HR 2.52, 95% CI 1.29–4.93, P = 0.007), and non-dippers (HR 2.09, 95% CI 1.19–3.68, P = 0.01) remained at higher cardiovascular risk".[30]

Guideline recommended normal cut-points of morning surge are still awaited (an area of future research) but its importance can be discerned from the higher occurrence of cardiovascular events during morning hours (time after awakening).

ENHANCED PROGNOSTIC ROLE OF NOCTURNAL BP PARAMETERS AS COMPARED TO DAYTIME PARAMETERS WITH REGARD TO TARGET ORGAN DAMAGE:

Nocturnal hypertension and various specific parameters of it are associated with target organ damage more often than daytime parameters of hypertension has been exemplified by several clinical studies.

In a pediatric population of 145 diabetic (type I) patients, Atabeck ME et al. found that Carotid intima media thickness (CIMT) was significantly more in subjects showing nocturnal hypertension as compared to normotensives (0.063 ± 0.010 mm vs.0.061 ± 0.009, P = 0.003). Nocturnal non-dipping pattern also showed a similar trend of higher CIMT.[31]

Kario K et al. in JAMP study of 6359 patients found that, nighttime systolic BP was associated with significantly elevated risk of atherosclerotic cardiovascular disease (ASCVD) and heart failure (HF) (increase 21% [p = 0.017] and 36% [p = 0.009] respectively), whereas daytime systolic BP rise is associated with ASCVD risk only and not total CVD. A significantly higher risk of CVD (1.48 [95% CI, 1.05–2.08]; P=0.024) and heart failure (2.45 [95% CI, 1.34–4.48]; P=0.004) was noted in patients showing riser pattern, and nighttime BP higher than daytime.[32]

Fabian F et al. in their study of 1521 patients (followed-up for 518 ± 120 days) found that of the ABPM parameters higher nighttime SBP was associated with MACE (HR: 1.018, 95% CI: 1.001–1.037; P = 0.044) more than any other parameter independently.[33] Similar results were noted by Fujiwara T et al. by time triggered HBPM in 2475 Japanese patients of J-HOP ((Japan Morning Surge-Home Blood Pressure) trial. They assessed the CVD risk in four groups by home BP monitoring of morning and evening BP and three automated nightly measurements for seven days. The groups were normotensives, daytime hypertensives, nocturnal hypertensives, and sustained hypertensives. Over a median follow-up of 7.6 years, hazard ratio (HR) for events was higher in nocturnal hypertensives (HR 1.57 95% CI 1.10-2.47) than normotensives (HR 1) and daytime hypertensives (HR 0.99 95% CI 0.47-2.10), but less than in those with sustained hypertension (HR 1.97 95% CI 1.26-3.10).[34]

Xu J et al. prospectively analyzed 1996 hypertensive patients for stroke occurrence, as compared to a validation cohort of stroke patients for recurrence, by clustering 24-hour ABP parameters making profile groups. Profile 1 comprised of patients with normal diurnal variation, profile 2 consisted of dippers with increased morning surge and profile 3 of nocturnal hypertensives with non-dipping. After removing the covariates only profile 3 patients showed significantly increased hazard ratios for stroke as compared to profile 1 (HR 1.76, 95% CI: 1.00 to 3.09), while no such difference was noted between profile 2 and profile 1 (HR 1.22, 95% CI: 0.66 to 2.25).[35] Yamamoto et al. studied 105 patients by ABPM and cerebrovascular findings and found that patients who showed increasing lacunar infarcts or white matter hyperintensities or cerebral hemorrhages had increased nocturnal blood pressure and reduced nocturnal dipping over a follow-up of 3.2 years.[36] This association was found by others only if reduced dipping is associated with sustained hypertensive response on ABPM.

Delsart et al. followed 281 type 2 diabetic patients (median 9.4 years) for lower limb events (revascularization or amputation). 20 lower limb events and 45 all-cause deaths were noted. Reverse dipping (noted in 35 patients) was associated significantly more with lower limb events (HR 3.61 95%CI, 1.16-11.2, p=.026).[37]

These clinical studies clearly show the superiority of nocturnal hypertension and its phenotypes regarding target organ damage over not only normotensives but over daytime hypertensives as well.

ASSOCIATION OF NOCTURNAL BLOOD PRESSURE WITH NON-CARDIAC CONDITIONS

Certain non-cardiac conditions have been found to be either affected by nocturnal hypertension or diagnosis of nocturnal hypertension indicates their concurrent presence.

Li ZT et al. in 86 hypertensive patients, with symptoms of GERD noted the association of hypertensive episodes with pathological reflux. Significantly higher episodes of high BP in GERD patients were noted, especially nocturnal hypertension (p= 0.026). No such association was noted for daytime or mean BP. These episodes were reduced dramatically after 14-day course of Omeprazole.[38]

Ma Y et al. studied 56 Chinese with orthostatic sleep apnea (OSA) of varying grades (mild, moderate, and severe) based on apnea-hypopnea index. Of the 67.9% of patients who were found hypertensive by 24-hour ABPM, more had nocturnal (73.2%) hypertension than daytime hypertension (57.1%). Analysis for dipper pattern revealed that as the severity of OSA increased, significantly more non-dipper and riser

patterns were noted, although not statistically significant.[39] Sapina-Beltran E et al. in 131 subjects with OSA and normotension found that 34.3 % of such subjects had masked hypertension. They found a significant reduction in mean 24-hour BP (-3.65 (-6.76 to -0.54) mmHg (P = 0.021) only in subjects showing masked hypertension. Furthermore, the true normotensive patients showing a non-dipper status responded to CPAP treatment by reduction in their blood pressures significantly not seen in patients showing dipper pattern. This proves the role of ABPM use in CPAP treatment even in clinically normotensive patients.[40]

Certain eye conditions like normotensive glaucoma and non-arteritic anterior ischemic optic neuropathy have been found to be specifically related to nocturnal blood pressure. Excessive dipping of nocturnal BP can result in exaggeration of these disease states.[41]

An association of nocturnal BP with proteinuria has been noted. In a study of 356 elderly patients (median age 66) Xinro Guo et al. found an association between lowest nocturnal systolic blood pressure (LNSBP) and heavy proteinuria (OR 1.24; 95% CI 1.10–1.39; P< 0.001; per 10 mmHg). There was a 24% enhanced chance of proteinuria for every 10 mmHg increase in LNSBP with the maximum incidence seen in those who have LNSBP 130 mmHg or more.[42]

Hypertension is associated with increased occurrence of Pre-eclampsia and eclampsia and resultant morbidity and mortality. Salazar MR et al. assessed 87 pregnant ladies by ABPM for various phenotypes of hypertension and their association with pre-eclampsia and eclampsia. Office (13.8%) and ABPM (40.2%) hypertension had a low concordance (k=0.17, p=0.044). Nocturnal hypertension (35.6%) was noted more often than 24-hour hypertension (26.4%) with increased relative risk of pre-eclampsia and eclampsia (OR 5.32 95% CI 1.48-19.10), this was not associated with diurnal hypertension until nocturnal hypertension was included in analysis.[43]

Increased prevalence of CV complications has been noted in diabetic patients with nocturnal hypertension, non-dipping, and reverse dipping.

Thus not only in cardiovascular diseases, in certain non-cardiac diseases also the assessment of nocturnal blood pressure is of paramount importance.

BENEFITS OF TREATMENT BY NOCTURNAL BP DERIVED PARAMETERS

Anti-hypertensive medications are usually taken in the morning, and many of them can't keep their therapeutic level for the entire 24-hour period resulting in nocturnal BP not being effectively controlled. It has been suggested that some or part of the medicines should be taken in the evening thus ensuring control of BP for the entire 24-hour period. MAPEC study endorsed this hypothesis in which more than 2000 subjects were given at least one medicine at nighttime. Significant reductions in non-dipping pattern, nocturnal BP, and CV events were noted along with much better control of 24-hour BP.[44]

CONCLUSION

Hypertension, a 24- hour phenomenon, is not a static event. It shows variability and every parameter of hypertension has clinical importance. An effective 24-hour control of BP is mandatory to achieve significant reduction in morbidity and mortality. Nocturnal BP, although representative more of the basal BP of an individual, is not measured routinely in clinical practice. Clinical research has delineated the various phenotypes of nocturnal hypertension with their individual diagnostic and prognostic importance. Elucidation of nocturnal hypertension and its various parameters has been shown to be superior to daytime parameters. This review article is an attempt to apprise the treating physician and other health-care providers the importance of measurement and control of nocturnal hypertension along with its phenotypes.

AUTHORS' CONTRIBUTION

IH: Concept and design, data acquisition, interpretation, drafting, final approval, and agree to be accountable for all aspects of the work.

Conflict of interest: Authors declared no conflict of interest.

Disclosures: All the figures/pictures in this manuscript are author's own work.

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