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Original Article

Is ambulatory blood pressure measurement a new indicator for survival among advanced heart failure cases

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ABSTRACT

Background: Ambulatory blood pressure monitoring (ABPM) in heart failure is not well defined. However, from the limited studies available, ABPM may be used to optimize heart failure therapy, and as a prognostic marker in this patient group. We analyzed the ABPM values with survival in advanced heart failure with reduced ejection fraction (HFrEF) patients who are on optimal guideline directed medical therapy (GDMT).

Methods and results: Hundred patients of advanced HFrEF were followed up for one year. Baseline left ventricular ejection fraction (LVEF), left ventricular end diastolic diamension (LVEDD) and ABPM values were measured and they were analyzed with survival. Deceased patients (n = 36) have lower ABPM values and are dippers as compared to living patients (n = 64) [24 hr systolic blood pressure (SBP24hr) = 97.6 ± 12.5 mmHg, 24 hr diastolic BP (DBP24hr) = 64.6 ± 10.2 mmHg, decrement in systolic BP (dipSBP) = 9.9 ± 5.2 mmHg and decrement in diastolic BP (dipDBP) = 11.1 ± 6.5 mmHg Vs SBP24hr = 109.4 ± 16.9 mmHg, DBP24hr = 71.7 ± 17 mmHg, dipSBP = 1.6 ± 5.9 mmHg and dipDBP = 2.7 ± 6.3 mmHg] and they were statistically significant with p values < 0.001, 0.025, <0.001, and <0.001 respectively. A logistic regression analysis was done to predict one year survival using age, sex, LVEF, LVEDD, SBP24hrs, DBP24hrs, dipSBP, dipDBP and dipMAP as independent predictors. When SBP24hrs is raised by one unit the chances of survival are 1.145 times more(Exp(B) = 1.145). One unit dip in SBP and DBP will reduce the chances of survival by 0.697 times and 0.586 times respectively.

Conclusion: In advanced HFrEF patients with Lower SBP & DBP and dippers have lesser survival compared to those with higher SBP & DBP and non-dippers.

1. Introduction

Heart failure (HF) is a syndrome complex with varied clinical features, etiology and pathophysiology. With such heterogeneity, it is difficult to assess severity and prognosis. Two most commonly used prognostic indices are left ventricular ejection fraction (LVEF)[1] and New York Heart Association (NYHA) functional class.[2] While NYHA functional class is subjective, LVEF is evaluated once and so it does not detect dynamic changes. In some studies, LVEF did not correlate with survival time in advanced heart failure patients. Several dynamic indeces such as stress testing, maximum myocardial oxygen consumption, maximum heart rate at effort etc are used. European Society of Cardiology defines advanced HF indexes[3] as NYHA class III or IV symptoms, objective evidence of severe cardiac dysfunction (EF < 30%), severely impaired functional capacity and HF hospitalization more than once in the past 6 months despite optimal guideline directed medical therapy. HF is associated with alterations in sympathetic and parasympathetic nervous system, renin-angiotesin system and vasopressin/atrial natriuretic peptide[4] secretion. Indeed, patients with severe congestive heart failure have increased sympathetic nervous system activity and impaired baroreceptor function, which will directly influence diurnal blood pressure profile.

Ambulatory blood pressure monitoring (ABPM) is capable of evaluating multiple aspects of blood pressure (BP) including 24-hr BP, nocturnal BP, dipping patterns, morning surge BP, postprandial hypotension and BP variability. There is an abundance of data on ABPM in hypertension, stroke, diabetes and chronic kidney disease but relatively little data on ABPM in heart failure. Several studies correlated ABPM variables with lesions in target organs in hypertensive patients. These studies used left ventricular

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hypertrophy[5], microalbuminuria,[6,7] retinal alterations and cerebrovascular diseases[8] as variables. However, few studies used ABPM to investigate heart failure. Some small studies have suggested that ABPM, specifically nocturnal blood pressure may be superior to office blood pressure measurement in predicting hospitalisation for heart failure[9]. During night retained fluid redistribute, resulting in increase in central venous pressure which in turn activates cardiopulmonary baroreflex. Hence there is decrement in night time BP. But in heart failure it is blunted resulting in non dipping pattern.[10–13]

2. Aims and objectives

- To determine the difference in mean baseline ambulatory BP measurement of advanced HF with reduced ejection fraction (HF_rEF) cases who died and who survived during one year of follow up.
- To determine the correlation of baseline ambulatory BP values with baseline LVEF and left ventricular end diastolic dimension (LVEDD).

3. Inclusion criteria

Patients presenting with advanced HF with reduced ejection fraction (EF) (NYHA IV), and on medical therapy were included. An exclusion criteria is applied before selection of patients for study. Evidence based treatment is optimised as per the ACC/AHA guidelines and at maximal tolerable doses.

4. Exclusion criteria

Patients with acute HF syndrome, hemodynamically unstable terminally ill patients, irregular heart rhythm, HF with normal EF, congenital heart disease, acute coronary syndrome, revascularization within past six months, endocarditis, pericarditis, myocarditis, peripheral arterial disease and patients on cardiac resynchronization therapy were excluded.

5. Material and methods

One hundred eligible patients of HF with reduced EF (NYHA IV) admitted in the department of cardiology, SMS Medical College, Jaipur were enrolled. After stabilization and decongestion, 24 h ambulatory BP monitoring, measurement of LVEF (Simpson's method) and LVEDD were done. To exclude peripheral arterial disease palpation of all peripheral pulses, recording of BP in all four limbs and auscultation for any bruit were done.

Medtech ambulatory BP instrument was used for 24 hr ABPM recording. EasyABPM software was used to analyze the ABPM values. ABPM was done by putting cuff on non dominant arm in the morning and removed at the same time next morning. Patients were given diary to record any unexpected events and instructed to relax the cuffed arm at the time of insufflations. The monitor was programmed to record BP every 30 min during day time and hourly in the night time. The following ABPM variables were obtained, mean 24 h systolic BP (SBP24hr), mean 24 h diastolic BP (DBP24hr), mean 24 h mean arterial pressure (MAP24hr), mean wake systolic BP (SBP_W), mean wake diastolic BP (DBP_W), mean wake mean arterial pressure (MAP_W) mean sleep systolic BP (SBP_S), mean sleep diastolic BP (DBP_S), mean sleep MAP (MAP_S), decrements in systolic BP (dipSBP), decrements in diastolic BP (dipDBP) and decrements in MAP (dipMAP).

Ischemic etiology was ruled in based on history of myocardial infarction or prior revascularization (coronary artery bypass graft or percutaneous coronary intervention). Patients with risk factors for coronary artery disease, coronary angiography was done to rule out ischemic etiology. First patient was enrolled on January 2015 and last patient was enrolled on February 2016. ABPM monitoring was done within one to two weeks of enrolment. No patients died before ABPM measurement. As patients were on HF medications only optimisation was done during follow-up. Patients were followed up for one year with regard to mortality. Follow-up was completed on February 2017. Death if occurred was confirmed by death certificate or by first degree relatives' information. Correlation between ABPM values and LVEF and LVEDD was done.

6. Statistical analysis

Categorical variables were expressed as percentages and continuous data as mean ± standard deviation (SD). Student's *t*-test was used to analyze the difference in the baseline ABPM values (SBP24hr, SBP_W, SBP_S, DBP24hr, DBP_W, DBP_S, MAP24hr, MAP_W, MAP_S, dipSBP, dipDBP and dipMAP) in both groups. Correlation between baseline ABPM values (SBP24hr, DBP24hr, dipSBP and dipDBP) with LVEF and LVEDD was done using Pearson correlation coefficient. Logistic regression was done for prediction of survival on the basis of independent predictors (age, sex, SBP24hr, DBP24hr, dipSBP, dipDBP, dipMAP, LVEF and LVEDD).

7. Results

Hundred patients of advanced heart failure were enrolled and were followed up for one year. At the time of enrolment 2D echocardiogram and 24hr ambulatory BP monitoring was done. Characteristics of the participants are shown in the Table 1. During the one year follow-up, 36 (36%) deaths occurred. Of 36 deaths, 7 (20%) deaths occurred in hospital (4 deaths due to worsening of heart failure and 3 deaths due to sudden death), 29 deaths occurred at home (25 deaths were due to worsening of HF and four deaths were due to sudden death). There was no statistically significant difference between living and deceased patients' baseline characteristics with regard to age, sex, diabetes, hypertension, smoking, electrocardiographic abnormalities, serum creatinine and medications.

Table 1
Characteristics of the participants.

	Deceased (n = 36)	Survived (n = 64)	p
Age (years)	54.3(14.4)	54.4(10.1)	0.968
Sex			
Male	22	38	>0.05
Female	14	26	>0.05
Diagnosis			
ICMP	21	39	>0.05
DCMP	15	25	>0.05
Diabetes	10	17	0.918
Hypertension	0	10	0.077
Smoking	11	27	0.349
ECG			
LBBB	10	19	>0.05
RBBB	2	5	> 0.05
IVCD	10	16	> 0.05
Bifascicular block	1	3	> 0.05
Sr.creatinine (mg/dl)	1.3(0.2)	1.2(0.3)	0.077
DRUGS			
Beta blocker	35	63	>0.05
ARB/ACEI	32	63	>0.05
Diuretics	36	64	> 0.05
Digoxin	10	8	>0.05

LBBB-left bundle branch block, RBBB- right bundle branch block, IVCD-intraventricular conduction block, ICMP- ischaemic cardiomyopathy, DCMP- dilated cardiomyopathy, ARB/ACEI-angiotensin receptor blocker/angiotensin convertase enzyme inhibitor.

Table 2
Comparison of baseline ABPM values at the time of enrollment between deceased and survived patients in one year (values in mean(sd)).

variable	Survival status		p
	Died(n = 36)	Survived(n = 64)	
LVEF (%)	19(4)	23(5)	<0.001
LVEDD (mm)	63.4(9.4)	60.5(9.4)	0.034
SBP24hr (mmHg)	97.6(12.5)	109.4(16.9)	<0.001
SBPw (mmHg)	97.9(14.3)	107(19)	0.014
SBPs (mmHg)	92.5(12.7)	106(17.3)	<0.001
DBP24hr (mmHg)	64.6(10.2)	71.7(17)	0.025
DBPw (mmHg)	64.5(9.4)	69.1(10.7)	0.034
DBPs (mmHg)	59(8.1)	68(12)	<0.001
MAP24hr (mmHg)	75.3(9.4)	84.1(14.8)	0.002
MAPw (mmHg)	76.4(10.2)	81.6(12.4)	0.035
MAPs (mmHg)	70.5(9.13)	81(12.2)	<0.001
dipSBP (mmHg)	9.9(5.2)	1.6(5.9)	<0.001
dipDBP (mmHg)	11.1(6.5)	2.7(6.3)	<0.001
dipMAP (mmHg)	9.4(5.8)	1.9(5.8)	<0.001

Echocardiographic parameters are shown in Table 2. Deceased patients had LVEF of 19 ± 4% and living patients had LVEF of 23 ± 5% with statistically significant difference ($p < 0.001$). Similarly LVEDD in deceased patients were 63 ± 9.4 mm as compared to living patients 60.5 ± 9.4 mm with statistically significant difference ($p = 0.034$)

Mean and standard deviations of living patients systolic BP values (SBP24 hr, 109.4 ± 16.9 mmHg; SBP_W, 107 ± 19 mmHg; SBP_S 106 ± 17.3 mmHg) were higher than that of deceased patients (SBP24 h, 97.6 ± 12.5 mmHg; SBP_W, 97.9 ± 14.3 mmHg; SBP_S 92.5 ± 12.7 mmHg) values. The difference is statistically significant with p values < 0.001, 0.014, <0.001 respectively and is shown in Table 2.

Mean and standard deviations of living patients diastolic BP values (DBP24hr, 71.7 ± 17 mmHg; DBP_W, 69.1 ± 10.7 mmHg; DBP_S 68 ± 12 mmHg) were higher than that of deceased patients (DBP24hr, 64.6 ± 10.2 mmHg; DBP_W, 64.5 ± 9.4 mmHg; DBP_S 59 ± 8.1 mmHg) values and is statistically significant with p values 0.025, 0.034, <0.001 respectively (Table 2).

Mean and standard deviations of living patients night decrements in pressure (dipSBP, 1.6 ± 5.9 mmHg; dipDBP, 2.7 ± 6.3 mmHg; dipMAP 1.9 ± 5.8 mmHg) were lower than that of deceased patients (dipSBP, 9.9 ± 5.2 mmHg; dipDBP, 11.1 ± 6.5 mmHg; dipMAP 9.4 ± 5.8 mmHg) values and is statistically significant with p values < 0.001, <0.001, <0.001, respectively (Table 2).

Correlation was done between SBP24hr, DBP24hr, dipSBP and dipDBP with LVEDD and LVEF. LVEF showed moderate positive correlation with dipSBP (correlation coefficient r = 0.33) and dipDBP (r = 0.32) whereas SBP24hr (r = 0.11) and DBP24hr (r = 0.18) showed slight positive correlation. LVEDD showed moderately negative correlation with dipSBP(r = −0.18) and dipDBP (r = −0.35) whereas SBP24hr (r = 0.18) and DBP24hr (r = 0.09) showed slight positive correlation as shown in Table 3.

Kaplan Meier and log rank tests of nonparametric analysis showed SBP 24hr, DBP 24hr, dip SBP, dip DBP are significant for prediction of survival (Figs. 1–4). The group of patients with SBP24hr > 105 mmHg (n = 45), DBP > 69 mmHg (n = 38), nondipper systolic BP (n = 68) and nondipper diastolic BP (n = 67) has longer survival time. We took an arbitrary cut-off value of SBP24hr 105 mmHg which is the mean SBP24hr of the study population similarly we took arbitrary cut-off value of DBP24hr 69 mmHg which is mean DBP24hr of study population in the Kaplan Meier survival analysis.

Table 3
Correlation between SBP24hr, DBP24hr, dipSBP and dipDBP with LVEF and LVEDD.

Parameters	LVEF	LVEDD
SBP24hr	r = 0.11	r = 0.18
DBP24hr	r = 0.18	r = 0.09
dipSBP	r = 0.33	r = −0.18
dipDBP	r = 0.32	r = −0.35

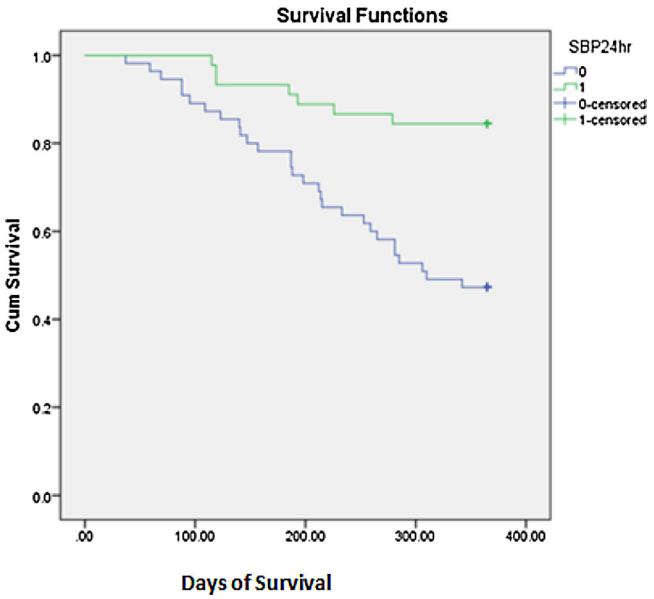

Fig. 1. Kaplan Meier survival curve: SBP24 h more than 105mmHg (green) vs less than 105mmHg (blue).

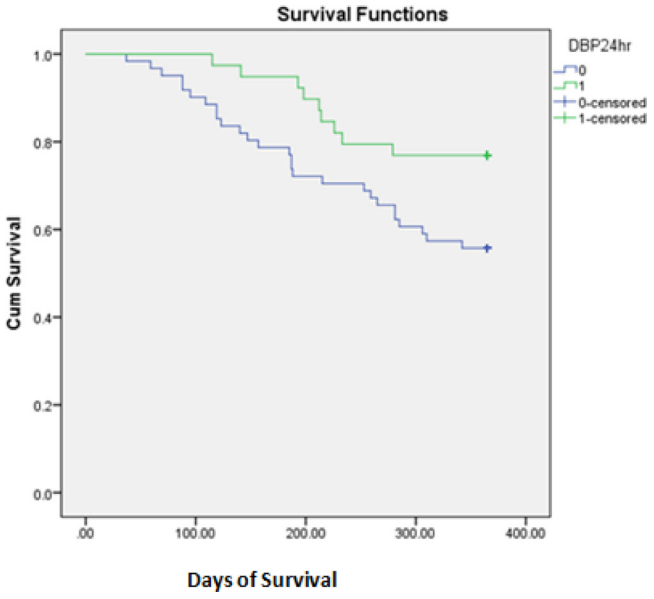

Fig. 2. Kaplan Meier survival curve: DBP24 h more than 69mmHg (green) vs less than 69 mmHg (blue).

8. Logistic regression for prediction of survival of advanced heart failure patients

A logistic regression analysis shown in Table 4 was done to predict one year survival using age, sex, LVEF, LVEDD, SBP24 h, DBP24 hr, dip in SBP, dip in DBP and dip in mean arterial pressure as independent predictors. A test of full model was statistically significant indicating that the predictors as a set reliably distinguish between the patients who survived and who died.

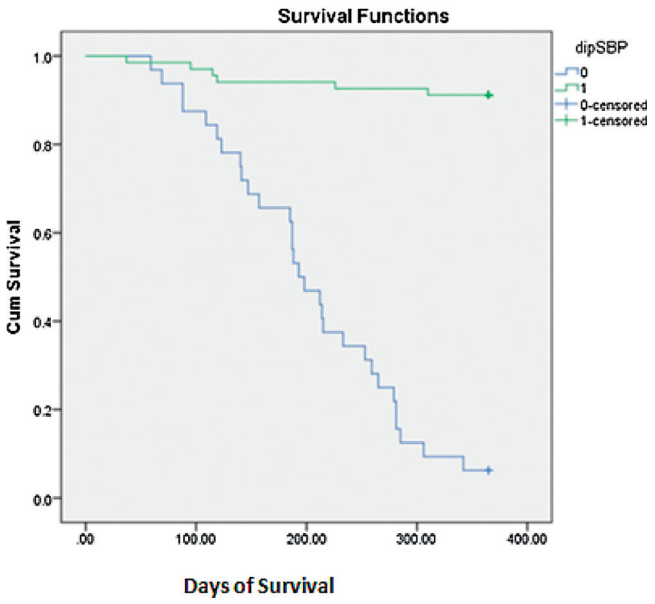

Fig. 3. Kaplan Meier survival curve: dipSBP dippers (blue) vs nondippers (green).

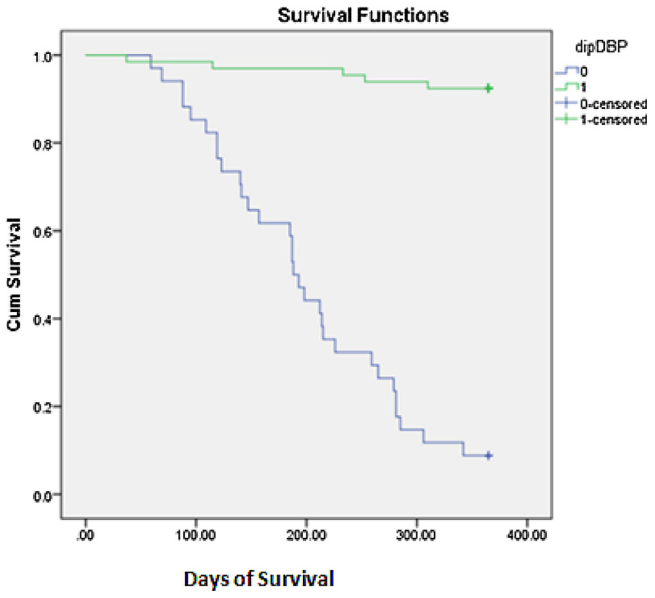

Fig. 4. Kaplan Meier survival curve: dipDBP dippers (blue) vs nondippers (green).

Table 4
Logistic regression for prediction of survival in advanced HF.

		p	Exp(B)	95% C.I.for EXP(B)	
Lower	Upper				
	age	0.140	0.950	0.888	1.017
	sex(1)	0.670	0.648	0.088	4.788
	LVEF	0.053	1.395	0.996	1.953
	LVEDD	0.787	0.974	0.802	1.182
	SBP24	0.008	1.145	1.035	1.266
	DBP24	0.430	0.973	0.908	1.042
	dipSBP	0.037	0.697	0.497	0.979
	dipDBP	0.015	0.586	0.381	0.900
	dipMAP	0.258	1.309	0.821	2.089
	Constant	0.257	0.000		

Also Hosmer and Lemeshow test shows a good fit by high p value (0.551) and low chi-square value (6.865) at degrees of freedom (df-8). Prediction success overall was 90.9%. The Wald criteria demonstrates that only SB24 hr, dip SBP and dip DBP made a significant contribution to predictors. Statistical value Exp (B) 1.145 for SBP24hr indicates that when SBP24 h is raised by one unit the chances of survival are 1.145 times more. One unit dip in SBP reduces the chances of survival by 0.697 times. Similarly one unit dip in DBP reduces the chances of survival (Exp B = 0.586).

9. Discussion

In healthy subjects, BP is highest in the early morning hours and declines to its lowest level at night. Normal circadian rhythm is dictated by various mechanisms including the sympathetic nervous system, postural position, baroreflexes, physical activity, tobacco use, sodium intake, alcohol use and neurohormones.[14] The superiority of circadian BP, specifically nocturnal BP, has been repeatedly demonstrated for cardiovascular outcomes in many disease states including hypertension, diabetes, stroke and kidney diseases. In heart failure up-regulated neurohormones, increased sympathetic activity, salt and water retention, and impaired baroreceptor reflex may impact the normal circadian rhythm. HF pharmacologic therapies that modulate the neurohormonal milieu, such as beta-blockers and ACEIs, may also play a role in the altered circadian rhythm.

Several small studies have demonstrated different average daytime blood pressure ranging from 108/72 mmHg[15] to 131/77 mmHg.[16] The data obtained by Borne et al[17] demonstrated even lower ambulatory daytime and nocturnal blood pressure in NYHA class III–IV patients. These conflicting data highlight the need for large studies to assess the circadian BP patterns in the heart failure population, especially in the current era of evidence based medicine.

In the healthy controls, the normal circadian pattern is one of the nocturnal dipping or a decline in BP from ambulatory daytime BP. Typically, this decline is approximately 20% compared with awake reading; however, the general consensus is that a decline or a “dip” of <10% from day to night BP reading is considered abnormal and is associated with poor cardiovascular outcomes.[18] Current literature classifies patients based on their nocturnal dipping profile: (1) dippers,10%–20% decline in BP from day to night, (2) Non-dippers, 0%–10% decline in nocturnal BP, (3) Extreme dippers, those with >20% decline in BP, and (4) Risers, an increase in nighttime BP from daytime reading. Several reports from independent centers showed that prevalence of LV hypertrophy,[19] cerebrovascular disease,[18,20] and microalbuminuria[21] were higher among subjects with blunted or abolished fall in BP from day to night than individuals with normal day–night BP difference in hypertensive patients. Furthermore, day–night BP changes significantly refined cardiovascular risk stratification above office BP and other traditional risk markers. Yamamoto et al[22] demonstrated that the degree of ambulatory BP reduction from day to night at the baseline assessment was significantly ($p < 0.01$) smaller in the group with subsequent cerebrovascular events than in the group with no future events. In older patients with isolated systolic hypertension, the Syst-Eur study found that cardiovascular risk increased with a higher night:day ratio of systolic BP (i.e., in patients more likely to be non-dippers) independent of the average 24-h BP.[23] Similarly, Ohkubo et al[24] showed an increased cardiovascular mortality in non-dippers (relative risk [RR]: 2.56, $p = 0.02$) and reverse-dippers (RR: 3.69, $p = 0.004$) in comparison with dippers. While these definitions have been applied to heart failure patients, there is no consensus on what constitutes a normal dipping profile in heart failure. In a large cohort of NYHA class II–III heart failure patients the majority had an abnormal dipping profile

using the standard definitions. In the same cohort, the presence of an abnormal dipping profile was an independent predictor of HF outcome. Non-dippers and risers had a 1.6 and 2.7 times increased risk of death or hospitalization compared to dippers, respectively.[25] In fact, there is some data that suggests a normal dipping profile may be detrimental in HF. Canesin MF et al[26] studied the effect of dipping profile on survival in 38 NYHA IV HF patients. Patients who had ≤6 mmHg decline in nighttime mean BP (dipSBP) had better prognosis at 6 months. While these findings can't be extrapolated to patients with less severe heart failure (NYHA I–III), they do raise considerable questions regarding the normal dipping profile in HF patients. Large-scale assessment of dipping profiles in HF patients with varying severity is required. Establishing values to define dippers and nondippers in HF is essential; it is possible that a dip of <10% is beneficial in HF. Establishing these definitions are important in ultimately determining if pharmacotherapy can be used to normalize the dipping profile and improve outcome. In our study we found that deceased patients were dippers (dipSBP = 9.9 ± 5.2 mmHg and dipDBP = 11.1 ± 6.5 mmHg) as compared to survived patients who were nondippers (dipSBP = 1.6 ± 5.9 mmHg and dipDBP = 2.7 ± 6.3 mmHg).

The link between blood pressure and outcome in heart failure can be made at a variety of levels. In a subset of 181 chronic HF (CHF) patients from the Rotterdam Heart Study, Mosterd et al[27] found that those community CHF patients with a higher BP had a better outcome. Canesin et al[26] studied 24-h ambulatory BP in 38 patients with advanced HF (NYHA IV) and also assessed their LVEF and LVEDD. These patients were then followed up for a minimum period of at least 6 months wherein 12 deaths occurred in this period. The mean 24-h, waking and sleeping systolic pressures of the living patients were higher than those of the deceased patients and were significant for predicting survival. Patients with a nocturnal dipDBP of less than 6 mmHg had longer survival. Conversely, patients with mean nocturnal dipSBP of <105 mmHg were 7.6 times more likely to die than those with SBP >105 mmHg. In this study, LVEF (35.2 ± 7.3%) and LVEDD (72.2 ± 7.8 mm) were not correlated with the survival.

In our study, analysis of LVEF showed moderate positive correlation with dipSBP (r = 0.33) and dipDBP (r = 0.32) whereas SBP24hr (r = 0.11) and DBP24hr (r = 0.18) showed slight positive correlation. LVEDD showed moderately negative correlation with dipSBP(r = −0.18) and dipDBP (r = −0.35) whereas SBP24hr (r = 0.18) and DBP24hr (r = 0.09) showed slight positive correlation. Canesin et at[26] showed positive correlation of LVEF with SBP24hr, SBP_W and SBP_S whereas LVEDD was negatively correlated with above ABPM variables. Caruana et al[28] did not observe correlation of LVEF with above parameters but positive correlation of LVEF with dipSBP and dipDBP similar to our study. Different findings of correlation between measures of BP and its variability with LVEF are probably due to heterogeneous characteristics in the disease evolutionary level, etiology and even the presence of associated diseases in patients of HF.

Franciosa et al[29] and Ghali et[30] al have individually shown in their studies that HF patient with higher BP had longer survival than those with lower BP. From these studies, there is shift of isolated BP measurements to continuous ABPM. Office BP measurements are prone to errors because of masked hypertension, white coat effect and white coat hypertension phenomenon.[31] Moreover dipping pattern can be better analyzed using ABPM measurement hence ABPM is superior to office BP measurement. In our study, deceased patients have lower BP and dipping pattern compared to living patients (deceased-SBP24hr = 97.6 ± 12.5 mmHg, DBP24hr = 64.6 ± 10.2 mmHg, dipSBP = 9.9 ± 5.2 mmHg and dipDBP = 11.1 ± 6.5 mmHg vs survived-SBP24hr = 109.4 ± 16.9 mmHg, DBP24hr = 71.7 ± 17 mmHg, dipSBP = 1.6 ± 5.9 mmHg and dipDBP = 2.7 ± 6.3 mmHg). So non-dippers and high SBP & DBP have more survival compared to dippers and low SBP & DBP. Canesin et al showed similar findings of improved survival in non dippers and high SBP & DBP. In the same study LVEF (35.2 ± 7.3%) and LVEDD (72.2 ± 7.8 mm) were not correlated with the survival. In Rotterdam Heart Study, Mosterd et al found that CHF patients with a higher BP had a better outcome.

Caruana et al[28] showed in their study in 20 patients of NYHA III/IV that non dippers were more in HF compared to controls but they did not analyse ABPM values with mortality. Portaluppi et al[32] also showed that circadian variability of BP altered in HF. Contrary to this Moroni et al[33] showed that there was no loss of circadian variability in advanced HF.

10. Conclusion

LVEF and NYHA functional class are the most frequently used prognostic indices in HF. In our study it is observed that lower SBP and dippers evaluated by 24-h ambulatory BP monitoring are predictors of higher mortality. Comparative analysis of survival plots suggest that parameters of SBP24hr, DBP24hr and night decrements of systolic/diastolic pressure obtained by ambulatory monitoring are predictors of mortality. So ABPM should be one of the new predictors of mortality together with the established ones.

Conflict of interest

None.

Funding

This study was not funded by anyone.

Ethical approval

all procedures performed in the study involving human participants were in accordance with ethical standard of the institutional and national research committee and with the 1964 Helsinki declaration and its later amendments or comparable ethical standards.

Informed consent

Informed consent was obtained from all individual participants included in study.

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