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Original Research Article

Ambulatory blood pressure monitoring in patients with ST- elevation myocardial infarction: one year follow-up study

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ABSTRACT

Background: The aim of this study was evaluation of nocturnal dipping of blood pressure in ST-elevation myocardial infarction (STEMI) patients and determining the effect of dipping on outcomes at 12 months follow-up.
Methods: This was an observational, single-centre, retrospective study that included STEMI patients, performed in a tertiary care hospital in India from November 2016 to October 2017. The primary endpoint of the study was the assessment of outcomes at 12 months. The patients were divided into two groups on the basis of blood pressure dipping, i.e., patients with positive dipping were considered in group 1 and patients with negative dipping were considered in group 2.
Results: Total 43 patients were included in the study. Group 1 consisted of 27 patients and Group 2 consisted of 16 patients. Mean 24 hr systolic blood pressure (SBP) and asleep SBP in Group 1 patients was 128.15±18.05 mmHg and 122.67±18.94 mmHg, respectively. Mean 24 hr diastolic (DBP) and asleep DBP in Group 1 patients was 78.07±10.73 mmHg and 73.41±12.35 mmHg, respectively. In the patients with non-dipping, mean 24 hr SBP and asleep SBP was 130.56±27.32 mmHg and 135.13±29.58 mmHg, respectively. Mean 24 hr DBP and asleep DBP was 76.00±15.40 mmHg and 79.69±17.05 mmHg, respectively. The mean percentage of asleep dipping of SBP was 5.7±6.7% in Group 1 and -4.6±6.82% in Group 2. Similarly, the mean percentage of asleep dipping of DBP was 7.6±9.0% in Group 1 and -6.3±9.1% in Group 2.
Conclusions: In view of the results, it can be concluded that ambulatory blood pressure monitoring in patients with STEMI can provide a significant prognostication of the future events.

Keywords: Ambulatory blood pressure, Diastolic blood pressure, Mortality, Systolic blood pressure, ST-elevation myocardial infarction

INTRODUCTION

Humans experience variation in blood pressure (BP) profile with circadian rhythm. The changes in BP pattern during day and night can be assessed using ambulatory blood pressure monitoring (ABPM). Recently, the use of ABPM is increasing due to its better ability to prognosticate significant cardiovascular events, such as myocardium infarction and stroke, as compared to the BP values obtained in consultation rooms.[1-3]

During ABPM, a variable to be highlighted is the BP drop at night, usually known as nocturnal dipping. Normally the BP gets reduced during sleep. Literature states that there is an inverse relation of BP dipping and cardiovascular outcomes, i.e. if the BP does not get reduced at night, then the probability of occurrence of a cardiovascular event increases.[3] The probable mechanisms behind this have been nocturnal autonomic dysfunction, disturbed baroreflex sensitivity, sleep apnea, abnormal sodium handling, endothelial dysfunction and nocturnal volume overload.[4] Endothelial cells have been associated with vasomotor balance and an imbalance in endothelial functioning will lead to alterations in vasodilation and vasoconstriction leading to altered blood pressures. Literatures also suggest that endothelium-dependent vasodilation is blunted through a decrease in nitric oxide release in non-dippers compared with patients who have nocturnal dipping of BP.[5]

The ABPM also has an important prognostic value in patients of acute MI.[6] The nocturnal dipping and non-dipping can pose effect on cardiovascular outcomes in such patients. Thus, the aim of the study was evaluation of nocturnal dipping of blood pressure in STEMI patients and determining the effect of dipping on outcomes at 12 months follow-up.

METHODS

This was an observational, single-centre, retrospective study performed in a tertiary care hospital in India from November 2016 to October 2017.

Inclusion and exclusion criteria

The patients who were of age 18 years and above, diagnosed with STEMI and who underwent thrombolysis were included in the study. The patients with cardiogenic shock, complicated arrhythmia and heart block, hepatic/renal dysfunction were excluded from the study.

The primary endpoint of the study was the assessment of outcomes at 12 months. Therefore the patients were divided into two groups on the basis of blood pressure dipping, i.e., patients with positive dipping were considered in group 1 and patients with negative dipping were considered in group 2.

Diagnosis of acute STEMI was made on the basis of at least two of the following criteria: an ST-segment elevation ≥1mm in at least two contiguous leads; chest pain lasting more than 30 minutes; peak creatine phosphokinase (CPK) values exceeding more than twice the upper limit of normal.[7]

Methodology

The ABPM was performed within 3 days after diagnosis of STEMI with a Suntech AccuWin Pro v3 software utilizing Oscar 2 system. Systolic and diastolic blood pressure, as well as heart rate (HR), mean arterial pressure (MAP) and pulse pressure measurements, were performed each 30 minutes during 6:30 am to 10:30 pm and during rest of the time at night, estimations were done hourly with a proper arm cuff. A mean of 24 hours of recording was obtained with a mean of 40 measurements per ABPM participant. The following variables derived from the ABPM were considered: mean 24 hours systolic and diastolic BP, mean 24-hour pulse pressure, and mean 24-hour arterial pressure. All these variables were also calculated for awake period, asleep period and white coat period. The enrolled patients were followed-up through an outpatient visits at 12 months after discharge. Cardiac death and development of signs and symptoms consistent with heart failure were considered as endpoints.

Statistical analysis

The continuous data were presented as mean, standard deviation, minimum and maximum. All data were analysed using the Statistical Package for Social Sciences (SPSS; Chicago, IL, USA) program, version 15.

RESULTS

Table 1: Baseline demographics and lesion characteristics of all patients.

Variables	Dipper (N=27)	Non dipper (N=16)
Age (mean±SD, years)	48.9±12.1	58.9±11.7
Male, n (%)	18(66.6%)	9(56.3%)
Chest pain, n (%)	15(55.6%)	11(68.8%)
Alcoholic, n (%)	5(18.5%)	2(12.5%)
Diabetes mellitus, n (%)	5(18.5%)	6(37.5%)
Hypertension, n (%)	4(14.8%)	9(56.3%)
Smoker, n (%)	6(22.2%)	5(31.3%)
Cerebrovascular accident, n (%)	1(3.7%)	0
No risk factors, n (%)	3(11.1%)	1(6.3%)
Location		
Anterior wall myocardial infarction, n (%)	10(37.0%)	9(5.3%)
Inferior wall myocardial infarction, n (%)	7(25.9%)	5(31.3%)
Hemoglobin (mean±SD, %)	9.3±0.8	8.8±0.5
Ejection fraction (mean±SD,%)	43.9±10.2	40.3±6.3
No. of vessels involved		
Single vessel disease, n (%)	15(55.6%)	4(25%)
Double vessel disease, n(%)	8(29.6%)	9(56.2%)
Triple vessel disease, n (%)	4(14.8%)	3(18.8%)
Death at 12 months follow-up, n (%)	3(11.1%)	4(25%)
Symptoms of heart failure at 12 months follow-up, n (%)	2(7.4%)	2(12.5%)

Total 43 patients were included in the study. Group 1 consisted of 27 patients who experienced dipping of blood pressure at night and Group 2 consisted of 16 patients who did not experience dipping at night. The mean age of patients was 48.9±12.1 years in Group 1 and 58.9±11.7 years in Group 2. There were 18 and 9 males, respectively in Group 1 and Group 2. Diabetes, hypertension, smoking and alcohol were less incident in patients of Group 1 than in patients of Group 2. Single vessel disease was more predominant in Group 1(55.6%) and double vessel disease was more prominent in Group 2(56.2%) (Table 1).

Mean 24 hr SBP and asleep SBP in Group 1 patients was 128.15±18.05 mmHg and 122.67±18.94 mmHg, respectively. Mean 24 hr DBP and asleep DBP in Group 1 patients was 78.07±10.73 mmHg and 73.41±12.35 mmHg, respectively. Both mean SBP and DBP were increased during white coat period in Group 1 patients. Details about Group 1 are represented in (Table 2). In the patients with non-dipping, i.e. increase of blood pressure at night, mean 24 hr SBP and asleep SBP was 130.56±27.32 mmHg and 135.13±29.58 mmHg, respectively. Mean 24 hr DBP and asleep DBP was 76.00±15.40 mmHg and 79.69±17.05 mmHg, respectively (Table 3).

Table 2: Details of group 1 patients (Dipper (n=27)).

Description	Mean	Std. Deviation	Minimum	Maximum
Mean 24 hr SBP, mmHg	128.15	18.05	96	168
Mean 24 hr DBP, mmHg	78.07	10.73	58	99
Mean 24 hr HR, bpm	83.19	14.69	63	126
24 hr MAP, mmHg	94.70	11.95	72	115
Mean 24 hr PP, mmHg	49.89	13.59	34	87
Mean SBP awake period, mmHg	129.63	18.10	97	171
Mean DBP awake period, mmHg	79.37	10.72	59	99
Mean HR awake period, bpm	83.81	14.74	63	126
MAP awake period, mmHg	96.07	11.97	74	117
Mean PP awake period, mmHg	50.26	13.81	35	88
Mean SBP asleep period, mmHg	122.67	18.94	89	159
Mean DBP asleep period, mmHg	73.41	12.35	49	97
Mean HR asleep period, bpm	80.37	14.85	61	126
MAP asleep period, mmHg	89.85	13.32	63	111
Mean PP asleep period, mmHg	49.26	13.47	33	84
Systolic asleep dip (%)	0.05	0.05	-0.02	0.209
Diastolic asleep dip (%)	0.08	0.07	0.006	0.302

SBP-Systolic blood pressure; DBP-Diastolic blood pressure; HR-Heart rate; MAP-Mean arterial pressure; PP-Pulse pressure

Table 3: Details of group 2 patients (Non-dipper (n=16)).

Description	Mean	Std. deviation	Minimum	Maximum
Mean 24 hr SBP, mmhg	130.56	27.32	99	184
Mean 24 hr DBP, mmhg	76.00	15.40	60	112
Mean 24 hr HR, BPM	83.44	11.56	64	111
24 hr MAP, mmhg	94.19	18.95	74	136
Mean 24 hr PP, mmhg	54.63	15.64	31	89
Mean SBP awake period, mmhg	129.13	27.27	97	184
Mean DBP awake period, mmhg	74.81	15.15	59	109
Mean hr awake period, BPM	83.13	11.08	65	109
MAP awake period, mmhg	93.00	18.53	73	132
Mean PP awake period, mmhg	54.19	15.38	31	89
Mean SBP asleep period, mmhg	135.13	29.58	102	201
Mean DBP asleep period, mmhg	79.69	17.05	62	125
Mean hr asleep period, BPM	84.00	15.06	57	119
MAP asleep period, mmhg	98.25	20.77	76	151
Mean PP asleep period, mmhg	55.63	16.15	33	88
Systolic asleep dip (%)	-0.05	0.04	-0.129	0.008
Diastolic asleep dip (%)	-0.06	0.03	-0.145	-0.009

SBP-Systolic blood pressure; DBP-Diastolic blood pressure; HR-Heart rate; MAP Mean arterial pressure; PP-Pulse pressure

The mean percentage of asleep dipping of SBP was 5.7±6.7% in Group 1 and -4.6±6.82% in Group 2. Similarly, the mean percentage of asleep dipping of DBP was 7.6±9.0% in Group 1 and -6.3±9.1% in Group 2 (Table 4).

Table 4: Difference in mean asleep dipping percentage between both groups.

Asleep dipping (%)	Dipper (N=27)	Non dipper (N=16)	p value
Systolic (%)	5.7±6.7	-4.6±6.82	<0.001
Diastolic (%)	7.6±9.0	-6.3±9.1	<0.001

DISCUSSION

With fast advancing research, the evidence of ambulatory blood pressure as a predictor of cardiovascular events has been increasing. Literature depicts that the patients with reverse dipping, i.e, non-dippers or the patients who experience increase in BP at night have been associated with worse outcomes irrespective of any history of cardiovascular disease.[8] Normally, the blood pressure reduces at night; the exact mechanisms behind the abnormal variation of blood pressure remain unknown, possibly the compromising in the autonomic balance, which leads to a sympathetic hyperactivity during sleep period that may alter circadian rhythm would be responsible.[8]

In this study of 43 patients, 27 patients experienced dipping at night and 16 patients experienced non-dipping at night. The mean age was 48.9±12.1 years and 58.9±11.7 years, respectively in both groups, representing the probability that higher age would lead to non-dipping. However, the results were contrary to a previous study by Melo R et al., in which the dippers and non-dippers had mean age of 60.4±12.4 and 60.6±11.3 years, respectively.[9] In this study males were predominant in both the groups. Diabetes, hypertension, smoking and alcohol were less incident in patients of Group 1 than in patients of Group 2. Thus, it can be postulated that presence of such risk factors can increase the chances of nocturnal increase in blood pressure leading to escalation in probability of cardiovascular events.

Patients of Group 1 had lower mean 24 hr SBP than Group 2 but the mean DBP was higher in Group 1 than Group 2. Accordingly, literature states that a low mean 24 hr DBP leads to drop of the myocardial perfusion gradient, formed by the intracoronary pressure, opposed by extra coronary resistances and left ventricle filling pressure.[10]Thus, low 24 hr mean DBP in Group 2 patients also hereby indicates the probability of higher morbidity and mortality in patients with reverse or non-dipping of nocturnal blood pressure.

During the white coat period, the SBP and DBP were higher in Group 1 (132.56 and 81.19 mmHg) than in Group 2 patients (123.44 and 71.63 mmHg), which depicts that more or less white coat hypertension is allied with the nocturnal dipping of blood pressure.

The mean pulse pressure in Group 1 patients was less than that of Group 2. The PP is an independent indicator of mortality. With age, the arterial rigidity increases, which in turn leads to increased risk of coronary events due to modification in the arteries' walls and development of atherosclerotic plaques.[11]

At 12 months follow-up, the mortality was seen in 11.1% patients of Group1 and in 25% in Group 2. Moreover, symptoms of heart failure were developed in 7.4% and 12.5% patients of Group 1 and 2, respectively. In a study by Antonini L, et al, the patients who experienced evens during follow-up had higher mean 24hr DBP which is similar to our study.[6] Paralelly, Ben-Dov IZ, et al. have also stated in a study that percentage of dipping of SBP, DBP and PP in dead patients was statistically lower than in patients who were alive.[3]

CONCLUSION

In view of the results, it can be concluded that ambulatory blood pressure monitoring in patients with STEMI can provide a significant prognostication of the future events. Non-dipping of blood pressure at night poses an indication for cardiovascular abnormality that could lead to increased chances of mortality and morbidity.

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