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Research Article

24 hour ambulatory blood pressure monitoring and left ventricular ejection fraction- prognostic markers in chronic kidney disease

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ABSTRACT

Background: Left ventricular systolic dysfunction i.e. <55% is common and an important predictor of risk of cardiac death in chronic kidney disease patients. Ambulatory blood pressure monitoring assists in targeting this population.
Methods: Total 160 non diabetic hypertensive patients were enrolled between 10/04/14 to 15/05/15 at M.L.N. Medical College and SRN Hospital, Allahabad, India. Out of them 80 were CKD patients who were taken as cases and remaining 80 patients were non CKD and taken as controls. Ambulatory blood pressure monitoring, clinic BP, eGFR, %LVEF (left ventricular ejection fraction) and other clinical data were collected for 1 year duration. Statistical analysis was done by using unpaired t-test for independent variables.
Results: Out of 160 non diabetic hypertensive patients, 48 (60%) cases and 28 (35%) controls were found non-dippers, while remaining 32 (40%) cases and 52 (65%) controls were have dipper BP pattern. Patients with non-dipping BP pattern and low %LVEF had the worst renal function and severe cardiovascular damages ($P<0.05$). The eGFR and %LVEF shown significant relationship with the rate of decline in nocturnal BP.
Conclusions: Ambulatory blood pressure measurement allows a better risk stratification compared to clinic blood pressure measurement. Non-dipping status and low LVEF is closely related to severe renal and cardiovascular damage in CKD patients. Low LVEF and 24-hour ABPM can be used as prognostic markers in non-diabetic CKD patients, and lowering of nocturnal BP will reduce the renal and cardiovascular risk in these patients.

Keywords: Left ventricular ejection fraction, Ambulatory blood pressure monitoring, CKD patients, Prognostic marker

INTRODUCTION

Hypertension and loss of diurnal BP variation i.e. non-dipping pattern is responsible for rapid progression of cardiovascular and renal disease. Normally, nocturnal dip (% fall in night time systolic blood pressure compared to daytime systolic blood pressure) of more than 10% occurs in night-time and this nocturnal dip is due to fall in sympathetic nervous system activity during night.[1] Patients having less than normal nocturnal decline in night-time systolic blood pressure have been termed non-dippers, while those with normal diurnal BP variation are termed dippers. Dipping ratio (mean night time SBP to mean day time SBP ratio) is more than 0.9 for non-dippers while it is 0.8 to 0.9 for dippers.[2]

Blunting or loss of this diurnal variation of BP (i.e. non-dipping) occurs in CKD patients due to increased sympathetic nervous system activity, volume expansion, sleep apnoea, low level of physical activity during daytime, poor sleep quality and use of antihypertensive drugs.[3-9] Hence twenty four hour mean systolic BP remains high in subjects with non-dipper BP pattern. Hence, some of the increased target organ damage risk i.e. renal and cardiovascular disease progression associated with elevated nocturnal blood pressures (non-

dipping blood pressure profile) may simply be due to the greater 24 hour BP load in non-dippers as compared to dippers.[10-13] And 24 hour ambulatory blood pressure monitoring (ABPM) is superior to clinic BP monitoring in predicting the risk in hypertensive CKD patients.[14-16]

METHODS

The present case control study was conducted in M.L.N. Medical College and associated SRN hospital, Allahabad, India between 10/04/14 to 15/05/15 to analyse the impact of diurnal blood pressure profile variation (dipping or non-dipping pattern) on target organ damage risk in non diabetic hypertensive chronic kidney disease (CKD) patients.

The subjects included in the current study were non diabetic hypertensive patients between the age group of 18 to 60 years of either sex attending nephrology OPD in SRN Hospital Allahabad and consented for the study. Out of total enrolled patients, cases taken in our study were non diabetic hypertensive (clinic blood pressure >140/90 mmHg, while ambulatory 24 hour blood pressure >130/80 mmHg) CKD patients of stage 2 to 5 as per KDIGO guidelines 2012, while control group comprised of age matched non-diabetic hypertensive non CKD volunteers. Out of total 160 enrolled subjects, 80 were cases and 80 were controls. Twenty four hour ABPM, eGFR (estimated glomerular filtration rate), and LVEF (left ventricular ejection fraction) were monitored for total duration of 1 year. Statistical analysis was done by using unpaired t test for independent variables to find out the association between non dipping blood pressure pattern and raised risk of target organ damage in non diabetic hypertensive CKD patients.

Any patient having changes in antihypertensive therapy 2 weeks before ABPM, true normotensive persons (BP<130/80 mmHg without antihypertensive therapy), patients on dialysis treatment or renal transplantation, diabetic CKD patients, patients with renal transplant, inadequate ABPM (number of recordings <14 and during day and <7during night respectively) and patients having established cardio vascular disease (valvular heart disease, cardiomyopathy, acute coronary syndrome) except hypertension were excluded from the study.

On the first visit to the OPD, clinic BP of all the participants was measured 3 times at 5 minute intervals. The clinic BP taken in this study was a mean of the 6 values recorded in the 2 consecutive days in which the ABPM device for 24-hr ambulatory BP monitoring was installed and removed. The installed ABPM device recorded systolic blood pressure and diastolic blood pressure (SBP and DBP) every 30 min between 7 am to 11 pm (active period) and every 60 min between 11pm to 7 am (passive period). The BP was considered at target when daytime and night-time values were less than 135/85mmHg and less than 120/70mmHg respectively. After this; dipping status (ratio of mean night time to mean day time SBP) was calculated. Then cases and controls were classified into dipper and non-dipper category depending upon the dipping ratio. Dippers have dipping ratio between 0.8-0.9 while for non-dippers this ratio is 0.9-1.0. Investigations collected on their first visit (zero month) were serum creatinine, serum urea, eGFR (Cockcroft-Gault formula), 2D-Echocardiography (%LVEF calculated by Simpson method), USG abdomen (renal size and echo-texture), fasting blood sugar, HbA1C, serum intact PTH, and haemoglobin. They followed up every month for complete 1 year. To assess the effect of hypertension on renal and cardiovascular functions, their eGFR and %LVEF were recorded monthly. The graph pad software version 6.0 was used for statistical analysis. The numerical data was compared by using unpaired t test for independent variables and the level of significance was considered at P value of 0.05.

RESULTS

Baseline characteristics of patients

The demographic and clinical data of the patients at the time of starting the study are listed in Table 1. Among the total 160 age matched hypertensive non-diabetic patients of either sex, 80 were CKD (cases), while 80 were non CKD (controls).

Prevalence of non-dipping pattern and increased risk of renal and cardiovascular events in study population

Out of 80 cases, 48 were non-dippers (60%) while among 80 controls only 28 (35%) were non-dippers (Table 1). During follow up, 22 cases (4 dippers and 18 non-dippers) and 4 controls (all 4 were non-dippers) were lost to follow up after their first clinic visit due to having either renal or cardiovascular end points (Table 2). Non-dipping pattern of BP is more prevalent in CKD patients and incidences of renal & cardiovascular events are high in non-dippers compared to dippers and even more in CKD patients compared to non CKD patients. Among cases 4 dippers and 12 non-dippers had to undergo haemodialysis while 4 non-dippers developed MI (myocardial infarction) and 2 non-dipper developed AF (atrial fibrillation), while among controls 2 dipper and 6 non-dipper turned into CKD during follow up while 2 non-dipper developed MI and 2 non-dipper had to undergo haemodialysis due to ARF (acute renal failure).

Effect of non-dipping blood pressure pattern on renal (eGFR) functions of study population

Table 3 shows the effect of non-dipping blood pressure pattern on eGFR after 1 year of follow up. Among cases at baseline (0 month), non-dippers had the mean eGFR (ml/min/1.73m^2) of 80.46±4.45 and dippers had 82.28±3.19. After 1 year of follow up, the mean eGFR of non-dippers and dippers decreased to 53.33±4.68 and 66.35±4.19 respectively. The decrement in mean eGFR of cases after 1 year of follow up was more in non-dippers as compared to dippers and was statistically

significant also (p value = 0.001). Among controls at baseline (0 month), non-dippers had the mean eGFR (ml/min/1.73m2) of 102.3±4.63 and dippers had 104.73±5.72. After 1 year of follow up, the mean eGFR of non-dippers and dippers decreased to 91.42±7.73 and 98.88±7.52 respectively. The decrement in mean eGFR of controls after 1 year of follow up was greater in non-dippers than dippers and was statistically significant also (p value = 0.007).

Data given in Table 3 shows that decrement in mean eGFR after 1 year of follow up was more in CKD patients as compared to non CKD patients. The Non-dipping pattern of blood pressure in hypertensive CKD patients has more significant relationship with decline in eGFR or progression of CKD as compared to dipping pattern of BP.

Table 1: Demographic parameters of patients.

Parameters	CKD patients (cases) n=80	Non CKD patients (controls) n=80
Number of males	58	64
Number of females	22	16
Mean age in years ±SD	48.58±7.94	47.35±9.78
Mean clinic BP (mmHg) ±SD	146 ±17/82±10	142±15/83±12
Mean ambulatory BP (mmHg) ±SD Mean 24hr Day-time Night-time	 **136±12/78±8** **144±19/81±9** **130±22/76±11**	 **133±9/75±6** **136±16/79±7** **126±17/71±13**
Dippers (n)	32	52
Non-dippers (n)	48	28

Table 2: Prevalence of renal and cardiac events in CKD (cases) and non CKD (controls) patients.

Dipping status	CKD (cases)		Non CKD (controls)	
	Renal events	Cardiac events	Renal events	Cardiac events
Dippers (n)	4 HD	None	2 turned into CKD	None
Non-dippers (n)	12 HD	4 acute MI 2 AF	2 HD 6 turned into CKD	2 acute MI

Effect of non-dipping blood pressure pattern on cardiovascular (%LVEF) functions of study population

Table 3 shows the effect of non-dipping blood pressure pattern on LVEF after 1 year of follow up. Among cases at baseline (0 month), non-dippers had the mean %LVEF of 54.53±3.54 and dippers had 60.36±4.9. After 1 year of follow up, the mean %LVEF of non-dippers and dippers decreased to 41.47±7.06 and 53.50±4.2 respectively. The decrement in mean %LVEF of cases after 1 year of follow up was more in non-dippers as compared to dippers and was statistically significant also (p value = 0.0001). Among controls at baseline (0 month), non-dippers had the mean %LVEF of 57.66±2.10 and dippers had 62.5±3.58. After 1 year of follow up, the mean %LVEF of non-dippers and dippers decreased 50.5±4.44 to and 58.65±4.92 respectively. The decrement in mean %LVEF of controls after 1 year of follow up was greater in non-dippers than dippers and was statistically significant also (p value = 0.001).

Table 3: Mean eGFR (ml/min/1.73m^2) and mean %LVEF values at zero month and at the end of 1 year and the decline in mean eGFR and mean %LVEF at the end of 1 year in dippers and non-dippers of CKD and non CKD patients.

Parameters	CKD (cases)		Non CKD (controls)	
	Dippers	Non-dipper	Dippers	Non-dippers
Mean eGFR ±SD at 0 month	82.28±3.19	80.46±4.45	104.73±5.7	102.3± 4.63
Mean eGFR ±SD after 1 year follow up	66.35±4.19	53.33±4.68	98.88±7.52	91.42± 7.73
Decline in mean eGFR after 1 year	15.65	27.13	5.85	10.91
Mean %LVEF ±SD at 0 month	60.36±4.9	54.53±3.54	62.5±3.58	57.66± 2.10
Mean %LVEF ±SD after 1 year follow up	53.50±4.2	41.47±7.06	58.65±4.92	50.5± 4.44
Decline in mean %LVEF after 1 year	6.68	13.06	3.85	7.16

Data given in Table 3 shows that decrement in mean %LVEF after 1 year of follow up was more in CKD patients as compared to non CKD patients. The Non-dipping pattern of blood pressure in hypertensive CKD patients has more significant relationship with decline in %LVEF or cardiovascular function as compared to dipping pattern of BP.

Night-time SBP compared to daytime SBP causes more target organ damage risk

After analysing the effect of daytime and nigh-time SBP on renal and cardiovascular function of cases and controls we found that eGFR and LVEF declined significantly (p value<0.05) among cases who had mean daytime and night-time SBP more than 135 and 120 mm Hg respectively as compared to cases having mean daytime and night-time SBP in the range of 125-135 and 110-120 respectively. And among controls, patients who had mean daytime and night-time SBP more than 145 and 135 mm Hg respectively had significant decline (p value <0.05) in both eGFR and LVEF compared to controls having mean daytime and night-time SBP in the range of 120 – 145 and 110-135 mm Hg.

Table 4: Mean eGFR (ml/min/1.73m^2) and mean %LVEF values at zero month and at the end of 1 year and the decline in mean eGFR and mean %LVEF at the end of 1 year in CKD and non CKD patients having mean daytime SBP of 125 – 135 mm Hg. (eGFR in ml/min/1.73m^2).

Parameters	Cases (CKD) n=15	Control (non CKD) n=28
Mean eGFR±SD at 0 month	81.23±3.12	103.14±2.50
Mean eGFR±SD after 1 year	78.21±4.04	101.22±3.45
Decline in mean eGFR after 1 year	3.02	1.92
P value	0.82	0.47
Mean LVEF±SD at 0 month (%)	62.88 ± 6.04	64.65±3.80
Mean LVEF±SD after 1 year	57.33±4.09	61.48 ±5.11
Decline in mean LVEF after 1 year	5.55	3.17
P value	0.07	0.935

Table 5: Mean eGFR (ml/min/1.73m^2)and mean %LVEF values at zero month and at the end of 1 year and the decline in mean eGFR and mean %LVEF at the end of 1 year in CKD and non CKD patients having mean daytime SBP of 135 – 145 mm Hg (eGFR in ml/min/1.73m^2).

Parameters	Cases (CKD) n=28	Control (non-CKD) n=40
Mean eGFR±SD at 0 month	79.29±3.66	99.74±2.32
Mean eGFR±SD after 1 year	75.26±3.57	97.40±1.43
Decline in mean eGFR after 1 year	4.03	2.34
P value	0.04	0.169
Mean LVEF±SD at 0 month (%)	56.19±2.62	60.79±2.14
Mean LVEF±SD after 1 year	45.55±3.85	56.72±3.26
Decline in mean LVEF after 1 year	10.64	4.07
P value	0.017	0.547

Table 6: Mean eGFR (ml/min/1.73m^2) and mean %LVEF values at zero month and at the end of 1 year and the decline in mean eGFR and mean %LVEF at the end of 1 year in CKD and non CKD patients having mean daytime SBP of >145 mm Hg (eGFR in ml/min/1.73m^2).

Parameters	Cases (CKD) n=15	Control (non CKD) n=8
Mean eGFR±SD at 0 month	77.58±3.62	99.88±2.20
Mean eGFR±SD after 1 year	52.0 ±1.88	85.48±3.86
Decline in mean eGFR after 1 year	25.51	14.4
P value	0.00069	0.003
Mean LVEF±SD at 0 month (%)	56.03±2.99	58.25±2.22
Mean LVEF±SD after 1 year	42.18±2.46	48.12±2.80
Decline in mean LVEF after 1 year	13.85	10.13
P value	0.007	0.028

Table 7: Mean eGFR (ml/min/1.73m^2) and mean %LVEF values at zero month and at the end of 1 year and the decline in mean eGFR and mean %LVEF at the end of 1 year in CKD and non CKD patients having mean night-time SBP of 110 - 120 mm Hg (eGFR in ml/min/1.73m^2).

Parameters	Cases (CKD) n=20	Control (non CKD) n=48
Mean eGFR±SD at 0 month	85.46±3.63	102.43±1.40
Mean eGFR±SD after 1 year	80.18±1.21	100.81±1.30
Decline in mean eGFR after 1 year	5.28	1.62
P value	0.07	0.27
Mean LVEF±SD at 0 month (%)	62.63±2.68	65.06±1.38
Mean LVEF ±SD after 1 year	59.78±2.54	63.21±1.60
Decline in mean LVEF after 1 year	2.85	1.85
P value	0.209	0.32

Table 8: Mean eGFR (ml/min/1.73m^2) and mean %LVEF values at zero month and at the end of 1 year and the decline in mean eGFR and mean %LVEF at the end of 1 year in CKD and non CKD patients having mean night-time SBP of 120 - 135mm Hg (eGFR in ml/min/1.73m^2).

Parameters	Cases(CKD) n=27	Control (non CKD) n=18
Mean eGFR±SD at 0 month	75.48±2.13	101.02±2.76
Mean eGFR±SD after 1 year	57.75±0.7	99.41±3.02
Decline in mean eGFR after 1 year	17.73	1.61
P value	0.0009	0.07
Mean LVEF±SD at 0 month (%)	55.56±4.33	59.45±4.11
Mean LVEF±SD after 1 year	45.35±5.02	56.49±5.04
Decline in mean LVEF after 1 year	10.21	2.96
P value	0.002	0.12

Table 4-9 shows that eGFR and LVEF in patients decreases as the mean daytime and night-time SBP increases but this decline is more in CKD patients as compared to non CKD patients, i.e. any degree of increment in blood pressure causes more target organ damage in CKD patients as compared to non CKD patients. Rise in night-time SBP leads to more increment in target organ damage risk when compared to similar level of rise in daytime SBP.

Table 9: Mean eGFR (ml/min/1.73m^2) and mean %LVEF values at zero month and at the end of 1 year and the decline in mean eGFR and mean %LVEF at the end of 1 year in CKD and non CKD patients having mean night-time SBP of >135mm Hg (eGFR in ml/min/1.73m^2).

Parameters	Cases(CKD) n=11	Control (non CKD) n= 10
Mean eGFR±SD at 0 month	75.45±3.76	98.50±4.95
Mean eGFR±SD after 1 year	46.87±2.35	80.23±3.05
Decline in mean eGFR after 1 year	28.58	18.27
P value	0.0005	0.007
Mean LVEF±SD at 0 month (%)	53.21±3.04	56.78±5.07
Mean LVEF ±SD after 1 year	38.49±4.76	45.76±4.83
Decline in mean LVEF after 1 year	14.72	11.02
P value	0.0001	0.003

DISCUSSION

Previous studies have demonstrated that 24-hr ambulatory blood pressure monitoring is an important prognostic marker in hypertensive patients.[14-16] Present study is done to assess the role of ABPM and LVEF as a prognostic marker in non-diabetic hypertensive CKD patients. The results of our study confirm the significance of 24-hr ambulatory BP monitoring in identifying or refuting the non-dipping pattern of BP or significant hypertension when compared with outpatient clinic BP measurement, as mentioned in a number of previous studies. Our study also shows that low LVEF is associated with increases risk of cardiac death in non diabetic hypertensive CKD patients. We found that left ventricular systolic dysfunction (low LVEF) which identifies patients at risk of cardiac death, increases with progression of renal failure and is particularly common with advanced CKD.

Minutolo R et al evaluated the prognostic role of ambulatory blood pressure measurement in patients with non-dialysis chronic kidney disease and concluded that office measurement of BP did not predict the risk of the renal or cardiovascular end point. Patients who were non-dippers and those who were reverse dippers had a greater risk of both end points. The result of their study demonstrates that the predictive role of ABPM is independent of other risk factors, such as diabetes mellitus, cardiovascular disease, proteinuria, haemoglobin level, and GFR. They conducted a prospective cohort study in 436 CKD patients (non-dialysis) to show the prognostic efficacy of ABPM (day & night SBP & DBP) in comparison with office

measurements and they found that high nocturnal BP leads to increased renal and cardiovascular risk in CKD patients.[15] Similarly in the present case control study, ABPM correlated more significantly with worsening of renal function and left ventricular systolic dysfunction or low LVEF than clinic BP in non diabetic hypertensive CKD patients.

Davidson MB et al predicted the association of impaired diurnal blood pressure variation with a subsequent decline in glomerular filtration rate and concluded that blunted diurnal blood pressure variation is associated with a subsequent deterioration in renal function that is independent of SBP load and other risk factors for renal impairment.[17]

Tripepi et al found that, 24h systolic BP was also greater in subjects with higher night/day systolic ratios. Hence, some of the increased target organ damage risk associated with elevated nocturnal pressures (non-dipping) may simply be due to the greater 24h BP load associated with this elevation.[14] This is consistent with our findings that Non-dippers have overall increased risk for target organ damage because of the greater 24hr blood pressure load as a result of elevated nocturnal blood pressure.

Agarwal R et al also concluded that systolic ambulatory BP and non-dipping are independent predictors for ESRD after adjusting for clinic BP.[16]

In the present study we found that a non-dipper BP pattern was independently correlated with kidney damage and low LVEF in CKD patients. A high BP at night (non-dippers) impacts the heart, vasculature, and kidney, boosting damage and increasing risk of developing clinical events in CKD patients. In the present study, after 1 year of follow up it was not surprising to find that in CKD patients with non-dipper BP pattern the decrement in eGFR (ml/min/1.73m^2) and %LVEF was 27.13 and 13.06 respectively while the patients with dipping status had decrement of only 15.65 in eGFR & 6.68 in %LVEF. Therefore, lowering nocturnal BP might help to reduce cardiovascular and renal death risk in non diabetic CKD patients who have non-dipping pattern of BP. Our study results show that, LVEF shows rapid and significant decline in chronic kidney disease patients i.e. LVEF was found to be lower in patients having low eGFR or moderate to severe renal failure. In this study non-dippers were found to have lower eGFR or more rapid progression of CKD and more left ventricular systolic dysfunction in comparison to dippers. As low LVEF raises the risk of cardiac death, hence LVEF can be used as a helping parameter for adequate control of hypertension, and consequently control of nocturnal BP will improve LVEF which in turn will lead to reduction in the risk of cardiac death in CKD patients. From our study it is very clear that left ventricular systolic dysfunction and ABPM can be used as a prognostic marker in predicting the risk of morbidity and mortality in non diabetic hypertensive CKD patients.

CONCLUSION

In non-diabetic hypertensive CKD patients, ambulatory blood pressure measurement allows a better risk stratification compared to clinic blood pressure measurement by identifying the dipping or non-dipping pattern of BP. Non-dipping BP pattern and low LVEF is closely related to severe renal and cardiovascular damage in CKD patients. Rise in night-time SBP leads to more increment in target organ damage risk when compared to similar level of rise in daytime SBP. Thus Low LVEF (< 55%) and 24-hr ABPM both are important prognostic markers to determine the morbidity and mortality in non diabetic hypertensive CKD patients, hence special attention should be given to these CKD patients for better outcome and Lowering of nocturnal BP will reduce the renal and cardiovascular risk in these patients.

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