Assessment of Hypertension Control in Chronic Kidney Disease Patients by Ambulatory Blood Pressure Monitoring

S Prakash*, Sumedha K Chibber**, Smita Prakash+, DP Pande***, S Joshi****, KK Gupta***** , DS Rana++

Abstract
Background: Inadequate control of blood pressure (BP) increases cardiovascular mortality and morbidity in chronic kidney disease (CKD) and renal transplant patients. 24 hour ambulatory BP was recorded to evaluate the adequacy of BP control in these patients.

Methods: 60 CKD patients (25 conservative therapy, 16 maintenance hemodialysis, 19 renal transplant patients) were studied prospectively. After achieving clinic BP control, 24 hour ambulatory BP was recorded at 1 and 6 months. The patients were followed up for one year.

Results: Mean daytime and nighttime systolic blood pressure (SBP) both at 1 month and at 6 month was higher in non-survivors than in survivors. The survivors had better control of their daytime (p=0.018) as well as nighttime SBP levels (p=0.018) at 6 months compared to those at 1 month. Survivors achieved nocturnal dipping of SBP at 1 and 6 months (p=0.047, p=0.025, respectively). Non-survivors failed to achieve lower daytime (p=0.375) or nighttime SBP (p=0.254) at 6 months as compared to SBP at 1 month in spite of optimizing antihypertensive therapy. Daytime (p=0.022) and nighttime (p=0.029) diastolic BP (DBP) in the non-survivors was higher than in survivors. Nocturnal dip in DBP was not seen in either survivors at 1 (p=0.177) and 6 months (p=0.434) or non-survivors at 1 (p=0.408) and at 6 months (p=0.081). Renal transplant patients did not exhibit nocturnal dipping of BP.

Conclusion: We conclude that, unlike survivors, there was worsening of 24 hour BP control in non-survivors. ABPM has a role in better management of total BP burden in CKD patients.©

INTRODUCTION

Control of hypertension is one of the key strategies for reducing the declining rate of glomerular filtration and retarding the progression toward end-stage renal disease.1 In the post-transplant period, tight control of hypertension is essential for the longevity of both, the graft and the patient. Several prospective clinical studies, as well as epidemiological studies, have indicated that the incidence of cardiovascular events is predicted by blood pressure (BP) as measured conventionally or with ambulatory methods,2 even after adjustment for a number of established risk factors.3 Therefore having reliable BP measurements on regular basis is crucial in the management of renal diseases.4 Hypertension should be viewed in total temporal profile as a persistent quantitative cardiac load. As ambulatory blood pressure monitoring (ABPM) yields multiple blood-pressure readings during all types of patient activities, including sleep, ABPM gives a far better representation of the “total blood-pressure burden” on the heart than what might be obtained by a single BP recording in the clinic.5 In some studies, 24-hour ambulatory blood pressure monitoring (ABPM) predicted cardiovascular events even after adjustment for conventional blood-pressure measurements.6 Normally the BP tends to fall during sleep as compared to daytime non-sleeping BP.7 This is called dipping and Ritz 8 describes it as a phenomenon in which decrease in nocturnal BP is 10 per cent or more. Attenuation of this physiological lowering of BP is non-dipping. Non-dipping of BP has been attributed to over-hydration, impaired functioning of autonomic nervous system, particularly parasympathetic nervous system, and sleep apnea.8 Abnormal blood pressure diurnal
rhythm (non-dipping) is significantly more common in secondary than in primary hypertension. This abnormality is independent of the underlying renal disease. Most, but not all authors, have noted an attenuated nocturnal decline in BP and an increased proportion of 'non-dippers' even in early renal disease.

ABPM is non-invasive, easily reproducible, portable, accurate and affordable. Yet for reasons not clearly understood, it remains underutilized in clinical practice. There is, therefore, a lack of data on the prognostic value of ABPM in patients of chronic kidney disease (CKD) and renal transplantation who are receiving antihypertensive treatment — a scenario that more closely reflects day-to-day clinical practice. This prompted us to initiate this prospective study with the following aims: (1) to evaluate adequacy of BP control in patients of CKD not only in the clinic, but over 24 hours; (2) to study the prognostic significance of 24 hour BP load; and (3) to see if the loss of physiological reduction of BP (non-dipping) during night had any bearing on patient outcomes.

**Methods**

The hospital ethics committee approved the study. Written informed consent was obtained from the patients. They retained their right to withdraw from the study at any time they so desired. The patients, who did not give their informed consent, were unable or unwilling for regular follow up, and those with a past history of non-compliance were excluded from the study.

Forty-one patients of CKD who were attending the nephrology division of Central Hospital were selected. Of these, 25 patients were on conservative therapy and 16 patients were on maintenance hemodialysis (MHD). Nine post renal transplant patients were also studied.

Detailed clinical examination and laboratory tests were done. Baseline creatinine clearance was calculated by 24-hour urinary collection and cardiac evaluation included X-ray chest, electrocardiogram (ECG) and 2D echocardiogram. The patients on conservative therapy received 35 K-calorie, 0.6gm/kg high biological value protein diet containing 5 gram salt. Amlodipine, enalapril, losartan, frusemide, metoprolol, clonidine and prazosin were the commonly employed antihypertensive medications. The target clinic BP was <140/90 mmHg. Patients receiving enalapril and/or losartan were regularly followed up for any evidence of hyperkalemia. In the event of any clinical, biochemical or electrocardiographic evidence of hyperkalemia, these patients are nearest to their dry body weight. Ambulatory blood pressures were then recorded over a 24-hour period during the patient’s normal daily activities, in the first month, with a properly validated and calibrated monitor (Toshiba TM-2424/2020). The monitor was programmed to obtain readings at 30 min intervals between 6 AM and 10 PM and at one hourly interval between 10 PM and 6 AM. A decrease of nocturnal BP by <10 per cent was considered as non-dipping. ABPM values >135/85 mmHg were considered as hypertension. The ABPM recording having 90% or more BP measurements was considered adequate. Any recording less than 90% of expected BP recordings were considered insufficient and the patient had to undergo another 24 hour ambulatory BP recording. The ABPM values were studied and anti hypertensive therapy was reinforced on the next patient visit, if the mean ABPM values were > 135/85 mmHg. The patients were followed up on a regular basis in the clinic. Twenty-four hour ABPM was repeated at the end of 6months. At the end of one year the data was statistically analyzed by using SPSS 10.0 Windows statistical package. Students’ paired t test was used. P values <0.05 were considered significant.

**Results**

The patient population comprised of 30 men and 30 women, mean age being 47.5 ± 14.9 years. The etiology of CKD was chronic glomerulonephritis in 24 (40%) patients, diabetes mellitus in 22 (36.6%), chronic interstitial nephritis in 7 (11.6%), hypertensive nephropathy in 2 (3.4%), autosomal dominant polycystic kidney disease in 2 (3.4%) and not known in 3 (5%) patients. Dyslipidemias were seen in 35 patients (58%).
The biochemical data and ABPM values of the transplant patients were separately analyzed and their values were excluded from the following analysis to prevent the confounding effect of renal transplantation on the mortality/survival profile of non-transplanted CKD patients. Therefore, the following analysis has been done on the data on 41 patients on conservative management and those on MHD (Table 1). The data of transplant patients is shown in Table 2.

### Systolic blood pressures

In the first month, 18 (43.9%) patients had mean ambulatory systolic BP >140 mmHg. In 12 of these patients another antihypertensive drug was added and in 6, the dose of existing drug was increased. At 6 months, 7 patients (17%) still had SBP > 140 mmHg. Mean daytime as well as nighttime SBP, both at 1 month and at 6 months was higher in non-survivors than in survivors (Table 3). Survivors showed better control of their daytime SBP (p=0.018) as well as nighttime SBP levels (p=0.018) at 6 months as compared to that at 1 month SBP levels (Table 4). Conversely, non-survivors did not achieve lower daytime (p=0.375) or nighttime SBP (p=0.254) at 6 months as compared to SBP at 1 month (Table 3, 5); even after attempts to intensify their antihypertensive therapy; rather the SBP in non-survivors were higher both during the day as well as during the night, at 6 months as compared to the SBP levels recorded at 1 month. The survivors had lower nocturnal SBP levels as compared to their daytime SBP levels both at 1 month (P=0.047) and at 6 months (P=0.025); that is, the survivors demonstrated nocturnal dipping of SBP. Nocturnal dipping was not observed in non-survivors.

### Table 1: Clinical characteristics of non-transplanted CKD patients

<table>
<thead>
<tr>
<th></th>
<th>Non-survivors (n=10)</th>
<th>Survivors (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>44.8 ± 10.0</td>
<td>48.2 ± 16.0</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>2 /8</td>
<td>16 / 15</td>
</tr>
<tr>
<td>Duration of hypertension (yr)</td>
<td>2.7 ± 0.29</td>
<td>4.6 ± 0.48</td>
</tr>
<tr>
<td>Duration of uremia (yr)</td>
<td>1.2 ± 0.88</td>
<td>2.4 ± 0.25*</td>
</tr>
<tr>
<td>Clinic SBP 1 month (mmHg)</td>
<td>136 ± 19</td>
<td>134 ± 17</td>
</tr>
<tr>
<td>Clinic DBP 1 month (mmHg)</td>
<td>77 ± 12</td>
<td>74 ± 11</td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>8.0 ± 2.4</td>
<td>8.3 ± 2.0</td>
</tr>
<tr>
<td>Blood urea nitrogen (mg/dl)</td>
<td>76 ± 34.1</td>
<td>71 ± 32.3</td>
</tr>
<tr>
<td>Serum creatinine (mg/dl)</td>
<td>4.2 ± 2.7</td>
<td>3.8 ± 1.9</td>
</tr>
<tr>
<td>Total protein (g/dl)</td>
<td>6.4 ± 0.5</td>
<td>6.3 ± 0.6</td>
</tr>
<tr>
<td>Serum albumin (g/dl)</td>
<td>3.6 ± 0.3</td>
<td>3.8 ± 0.5</td>
</tr>
<tr>
<td>Urinary albumin (mg/day)</td>
<td>1.3 ± 0.3</td>
<td>1.9 ± 0.1</td>
</tr>
<tr>
<td>LV mass (gm)</td>
<td>290.6 ± 86.2</td>
<td>211.0 ± 52.5*</td>
</tr>
<tr>
<td>LVH on echocardiogram (n/%)</td>
<td>7 (70)</td>
<td>16 (39.2)</td>
</tr>
<tr>
<td>LVH on electrocardiogram (n/%)</td>
<td>8 (80)</td>
<td>11 (26.8)*</td>
</tr>
<tr>
<td>Non-dippers (n)</td>
<td>8</td>
<td>14</td>
</tr>
<tr>
<td>Dippers (n)</td>
<td>2</td>
<td>17</td>
</tr>
</tbody>
</table>

*P<0.05. Values are mean ± SD where appropriate.

### Table 2: Clinical characteristics of transplant patients

<table>
<thead>
<tr>
<th></th>
<th>Non-survivors (n=10)</th>
<th>Survivors (n=31)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>47.6 ± 7.2</td>
<td></td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>12 / 7</td>
<td></td>
</tr>
<tr>
<td>Duration of hypertension (yr)</td>
<td>3.9 ± 0.31</td>
<td></td>
</tr>
<tr>
<td>Duration of uremia (yr)</td>
<td>2.7 ± 1.5</td>
<td></td>
</tr>
<tr>
<td>Clinic systolic BP at 1 month (mmHg)</td>
<td>130 ± 10.2</td>
<td></td>
</tr>
<tr>
<td>Clinic diastolic BP at 1 month (mmHg)</td>
<td>81 ± 12.3</td>
<td></td>
</tr>
<tr>
<td>Hemoglobin (g/dl)</td>
<td>11.9 ± 2.7</td>
<td></td>
</tr>
<tr>
<td>Blood urea nitrogen (mg/dl)</td>
<td>20.3 ± 11.3</td>
<td></td>
</tr>
<tr>
<td>Serum creatinine (mg/dl)</td>
<td>1.5 ± 1.2</td>
<td></td>
</tr>
<tr>
<td>Total protein (g/dl)</td>
<td>6.3 ± 0.5</td>
<td></td>
</tr>
<tr>
<td>Serum albumin (g/dl)</td>
<td>3.6 ± 0.3</td>
<td></td>
</tr>
<tr>
<td>Urinary albumin (mg/day)</td>
<td>0.56 ± 0.16</td>
<td></td>
</tr>
<tr>
<td>Left ventricular mass (gm)</td>
<td>233 ± 39.6</td>
<td></td>
</tr>
<tr>
<td>LVH on echocardiogram (n/%)</td>
<td>13 (68.4)</td>
<td></td>
</tr>
<tr>
<td>LVH on electrocardiogram (n/%)</td>
<td>10 (52.6)</td>
<td></td>
</tr>
<tr>
<td>Non-dippers (n)</td>
<td>15 (75.9)</td>
<td></td>
</tr>
<tr>
<td>Dippers (n)</td>
<td>4 (21.1)</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD where appropriate.

### Table 3: Comparison of day and night time systolic and diastolic blood pressure levels at 1 month and 6 months in survivors, non-survivors and transplant patients

<table>
<thead>
<tr>
<th></th>
<th>Survivors (n=31)</th>
<th>Non-survivors (n=10)</th>
<th>Transplants (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic BP (mmHg)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Day-time 1 month</td>
<td>140 ± 20</td>
<td>164 ± 32</td>
<td>136 ± 19</td>
</tr>
<tr>
<td>Day-time 6 month</td>
<td>130 ± 15</td>
<td>174 ± 29</td>
<td>127 ± 13</td>
</tr>
<tr>
<td>Night-time 1 month</td>
<td>137 ± 19</td>
<td>162 ± 30</td>
<td>132 ± 14</td>
</tr>
<tr>
<td>Night-time 6 month</td>
<td>123 ± 14</td>
<td>179 ± 32</td>
<td>134 ± 18</td>
</tr>
</tbody>
</table>

Diastolic BP

<table>
<thead>
<tr>
<th></th>
<th>Survivors (n=31)</th>
<th>Non-survivors (n=10)</th>
<th>Transplants (n=19)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Day-time 1 month</td>
<td>83 ± 13</td>
<td>81 ± 8</td>
<td></td>
</tr>
<tr>
<td>Day-time 6 month</td>
<td>77 ± 13</td>
<td>89 ± 11</td>
<td></td>
</tr>
<tr>
<td>Night-time 1 month</td>
<td>82 ± 10</td>
<td>82 ± 12</td>
<td></td>
</tr>
<tr>
<td>Night-time 6 month</td>
<td>77 ± 16</td>
<td>91 ± 13</td>
<td></td>
</tr>
</tbody>
</table>

Values are mean ± SD where appropriate.

### Table 4: Statistical analysis of blood pressure values in survivors

<table>
<thead>
<tr>
<th></th>
<th>Pair</th>
<th>Mean d</th>
<th>Standard deviation</th>
<th>95% Confidence interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP1D &amp; SBP1N</td>
<td>3.6765</td>
<td>12.4334</td>
<td>(0.0067, 7.2851)</td>
<td>0.047</td>
<td></td>
</tr>
<tr>
<td>SBP6D &amp; SBP6N</td>
<td>4.2121</td>
<td>11.9288</td>
<td>(0.6947, 7.7296)</td>
<td>0.025</td>
<td></td>
</tr>
<tr>
<td>SBP1D &amp; SBP6D</td>
<td>7.9091</td>
<td>20.8032</td>
<td>(1.7749, 14.0433)</td>
<td>0.018</td>
<td></td>
</tr>
<tr>
<td>SBPIN &amp; SBP6N</td>
<td>8.1515</td>
<td>21.3192</td>
<td>(1.8652, 14.4379)</td>
<td>0.018</td>
<td></td>
</tr>
<tr>
<td>DBP1D &amp; DBP1N</td>
<td>1.0588</td>
<td>6.5687</td>
<td>(-0.8477, 2.9653)</td>
<td>0.177</td>
<td></td>
</tr>
<tr>
<td>DBP6D &amp; DBP6N</td>
<td>0.3333</td>
<td>11.4227</td>
<td>(-3.0349, 3.7015)</td>
<td>0.434</td>
<td></td>
</tr>
<tr>
<td>DBP1D &amp; DBP6D</td>
<td>6.3333</td>
<td>17.3793</td>
<td>(1.2087, 11.4579)</td>
<td>0.022</td>
<td></td>
</tr>
<tr>
<td>DBP1N &amp; DBP6N</td>
<td>5.5758</td>
<td>16.3727</td>
<td>(0.7480, 10.4035)</td>
<td>0.029</td>
<td></td>
</tr>
</tbody>
</table>

(S=Systolic, D=Diastolic, BP= blood pressure, 1-first month, 6-sixth month, D-daytime, N-nighttime, Mean d-mean value of differences, p-probability of rejection)
Table 5: Statistical analysis of blood pressure levels of non-survivors

<table>
<thead>
<tr>
<th>Pair</th>
<th>Mean d</th>
<th>Standard deviation</th>
<th>95% Confidence interval</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>SBP1D &amp; SBP1N</td>
<td>0.2222</td>
<td>18.3697</td>
<td>(-11.1642, 11.6086)</td>
<td>0.486</td>
</tr>
<tr>
<td>SBP6D &amp; SBP6N</td>
<td>-9.3692</td>
<td>6.3692</td>
<td>(-14.4062, -3.9271)</td>
<td>0.008</td>
</tr>
<tr>
<td>SBP1D &amp; SBP6D</td>
<td>-3.667</td>
<td>26.8229</td>
<td>(-25.7322, 18.3969)</td>
<td>0.375</td>
</tr>
<tr>
<td>SBP1N &amp; SBP6N</td>
<td>-6.667</td>
<td>22.9056</td>
<td>(-25.5097, 12.1764)</td>
<td>0.254</td>
</tr>
<tr>
<td>DBP1D &amp; DBP1N</td>
<td>-0.60</td>
<td>7.9331</td>
<td>(-3.1989, 3.986)</td>
<td>0.408</td>
</tr>
<tr>
<td>DBP6D &amp; DBP6N</td>
<td>-2.428</td>
<td>6.0356</td>
<td>(-5.3925, 0.5354)</td>
<td>0.081</td>
</tr>
<tr>
<td>DBP1D &amp; DBP6D</td>
<td>-10.00</td>
<td>10.8321</td>
<td>(-17.9556, -2.0444)</td>
<td>0.025</td>
</tr>
<tr>
<td>DBP1N &amp; DBP6N</td>
<td>8.00</td>
<td>14.5945</td>
<td>(-18.7190, 2.7190)</td>
<td>0.098</td>
</tr>
</tbody>
</table>

(S-Systolic, D-diastolic, BP-Blood Pressure, 1-first month, 6-sixth month, D-daytime, N-nighttime, Mean d-mean value of differences, p-probability of rejection)

the non-survivors at 1 month (P=0.486) or at 6 months. Instead, the nocturnal SBP at 6 months had risen significantly compared to their daytime levels in non-survivors (P= 0.008). The mean nocturnal SBP was significantly lower in survivors as compared to non-survivors at 1 month as well as at 6 months (Table 3).

**Diastolic blood pressures**

In the survivors, mean daytime DBP was lower at 6 months compared to DBP at 1 month (p= 0.022). Similarly, mean nighttime DBP was lower at 6 months as compared to 1 month in the survivors (p=0.029). However, nocturnal dipping was not observed in DBP at 1 month (P=0.177) or at 6 months (P= 0.434) in the survivors (Table 3, 4).

In the non-survivors daytime DBP at 6 months was higher than the daytime DBP at 1 month. (P= 0.025) Nighttime DBP at 6 months was higher than the DBP at 1 month in non-survivors. (P= 0.098)

At 6 months the non-survivors had higher daytime DBP levels (P< 0.05) and higher nocturnal DBP levels (P= 0.029) than survivors. Thus, there was a rise in day and night DBP at 6 months in the non-survivors whereas survivors attained lower daytime and nighttime DBP at 6 months (Tables 3, 5).

In the non-survivors there was an absence of nocturnal dipping in mean diastolic pressures at 1 month (P=0.408) as also at 6 months (P=0.081). Even in survivors, better control of DBP at 6 months had not resulted in restoration of nocturnal dipping of DBP.

**Mortality**

At one year 31 of the 41 patients (70.5%) were alive. There were 10 deaths, 4 (16%) of 25 patients on conservative therapy and 6 (37.5%) of 16 patients on MHD. The survivors and non-survivors were comparable in terms of age, clinic BP, hemoglobin level, renal functions, total protein serum albumin and 24 hour urinary albumin excretion (Table 1). The duration of uremia was significantly longer in survivors but the duration of hypertension was not significantly different in survivors and non-survivors.

Non-survivors had significantly higher left ventricular masses on 2-d echocardiogram as compared to survivors (P<0.018) (Table 1). There was greater prevalence of left ventricular hypertrophy (LVH) in echocardiogram as well as on ECG in non-survivors compared to survivors. Significantly, the cause of death in the majority was cardiovascular. Acute coronary syndrome was responsible for 4 deaths, 2 patients had dilated cardiomyopathy, one had septicemia, one had extensive pulmonary tuberculosis, one patient had diffuse pericarditis with multiorgan failure and one had sudden death at home.

**Transplant patients**

In the transplant group (Table 2, 3) daytime SBP at 6 months was significantly reduced compared to that at 1 month (P<0.05). However, the nighttime SBP remained unchanged at 6 months compared to that at 1 month (P>0.05). Although lower SBP levels were achieved at the end of 6 months, yet the transplant patients were unable to lower their nocturnal sleeping BP levels compared to daytime BP levels. There was no significant reduction in DBP at 6 months compared to that at 1 month, both in daytime as well as nighttime DBP ambulatory recordings (P>0.05) (Table 4). All nineteen patients were well with stable graft functions.

**DISCUSSION**

**24 hour blood pressure load and its reduction**

This study has brought out the difficulties associated with correct assessment of the adequacy of 24 hour BP control by clinic BP alone. Therefore, achieving tight blood pressure control on a long temporal period in CKD patients becomes difficult. In this study all patients at enrollment apparently had satisfactory BP control by clinic BP (<140/90 mmHg) on 3 occasions, but measurement of their 24 hour ambulatory BP in their home surrounding revealed that control was less than optimal. Initially 18 (43.9%) patients had mean ambulatory systolic BP >140 mmHg whereas at 6 months of intensifying therapy based on mean 24 hour SBP values, 7 patients (17%) had SBP>140 mmHg. CBP had failed to identify this occult hypertensive load. ABPM helped in the detection and subsequent reduction of SBP in 26.9% of patients. Reduction of 24 hour SBP load had bearing on the outcomes of these patients, as it plays a major role in determining cardiac damage in dialysis patients via LVH. Verdecchia et al have also reported that ambulatory blood pressure control is superior to office blood pressure control for prediction of individual cardiac events.
cardiovascular risk in treated hypertensive subjects.

We found positive correlation between raised SBP levels and mortality as non-survivors had significantly higher SBP levels compared to survivors both at 1 month and at 6 months. \( p = 0.006, p = 0.0001, \) respectively). Our patient numbers do not permit us to say unequivocally whether poorer SBP control in non-survivors is the cause of increased mortality or is just an epiphenomenon.

Regarding DBP evaluation at 1 month, there was no difference in daytime and nighttime DBP in both survivors and non-survivors. However, at 6 months the non-survivors had significantly raised daytime and nighttime DBP. This persistently raised BP load in our study, not withstanding our best efforts to control it, was an important predictor of mortality. Clemente et al.\(^2\) have also reported that higher ambulatory SBP or DBP levels predict cardiovascular events even after adjustment for classic risk factors including office measurements of blood pressure. In Macgregor's study \(^4\) of 24 home HD patients, anemia, increased 24 hour BP burden along with nocturnal non-dipping of BP were responsible for increased LVH in the non-survivors. Likewise, we also found significantly higher SBP, attenuation of nocturnal dipping, more LVH and left ventricular mass index (LVMI) in the non-survivors as compared to survivors. Moderately severe anemia was present in our patients, both in survivors and the non-survivors. Not withstanding the fact that anemia is strong predictor of LVH and other cardiac complications; we feel that elevated SBP was more important in the causation of LVH and adversely affecting the survival of our patients. This is corroborated by the fact that the hemoglobin levels were comparable in both the survivors as well as non-survivors, whereas the SBP load was significantly raised in the non-survivors in our study. Cost economically also, it is more prudent to achieve tighter BP controls, as it costs much less than hemoglobin correction to normal levels in CKD patients. Harnett et al.\(^5\) also found SBP to be significantly and independently associated with increased LVMI. In a large sample of a Japanese community comprising both treated and untreated subjects, ambulatory blood pressure predicted the risk of fatal cardiovascular events, even after adjustment for age, sex, risk factors, medication, cardiovascular history, and conventional blood pressure values. The correlation between LVMI and mean 24 hour ambulatory SBP was significantly stronger than with clinic BP.\(^1\)

By measuring 24 hour ABPM in our transplant patients we could ascertain their total BP burden and objectively emphasized, to the patients, the importance of keeping their BP under tight control. This has probably led to lower daytime SBP and DBP levels at 6 months compared to enrollment BP at 1 month.

Nocturnal non-dipping

In addition to BP control, we specifically looked for the nocturnal BP pattern in our patients. It has been shown that loss of nocturnal dipping precedes development of micro albuminuria in a certain percentage of diabetic patients.\(^6\) Therefore, non-dipping is now considered to be an earlier and more sensitive predictor of nephropathy and is recommended as a clinical marker of diabetic nephropathy.\(^7\) A strong correlation exists between non-dipping and albumin excretion in both, type I diabetes and type II diabetes.\(^8\) A significantly higher risk of cardiovascular complications has also been reported in nocturnal non-dippers.\(^9\) In our study also, nocturnal non-dipping was associated with increased mortality as 80% of non-survivors were non-dippers. Conversely, 55% of the survivors were non-dippers and 45% were dippers. It is a likely possibility that in the long run, those survivors with loss of nocturnal dip would have higher mortality than that subgroup of survivors who were dippers. However, this requires confirmation by studies with a longer follow up.

In spite of adequate BP control as validated by clinic BP recordings, majority of our transplant patients (78.9%) did not have restoration of nocturnal dipping. This is consistent with Farmer's study \(^10\) but at variance with Gatzka et al.\(^11\) who have reported normalization of BP rhythm, post renal transplant. This conflicting literature on post transplant BP control needs to be addressed by larger trials as untreated nocturnal non-dipping of longer duration may accelerate graft dysfunction.

The drawbacks of ABPM include the slight inconvenience of wearing the device and the fact that not every monitoring session is technically successful and not all monitors are accurate. Yet ABPM will remain the optimum benchmark for diagnosis of total hypertension load until alternative methods have proven their value. As for the cost, if ambulatory monitoring can provide a more accurate assessment of adequacy of blood pressure control or demonstrate the effectiveness of a particular antihypertensive medication or regime, the associated costs will be more than offset by the savings involved.\(^2\)

Our study concurs with Liu et al.\(^12\) that ambulatory BP monitoring is non-invasive, safe and reproducible technique which can be of great use in achieving better BP control, identifying high risk patients (non-dippers) and prognostication of outcomes along with traditional markers of end-stage renal disease.

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