FATE OF HYPERTENSION (HTN) BEFORE AND AFTER KIDNEY TRANSPLANTATION IN PATIENTS WITH END STAGE RENAL DISEASE (ESRD)
C. Venkata S. Ram, Robert Toto, David Nesser, Lannette Harris, Patricia Lemons, Elisith Hatfield. Clinical Research Institute, Dallas Nephrology Associates, Dallas, TX, United States.

Systemic hypertension (HTN) is a major problem in patients with ESRD and contributes to significant morbidity and mortality in this high-risk population. While kidney transplantation (KTx) can restore renal function, hypertension remains a serious problem after transplantation and cardiovascular disease is the number one cause of death in renal transplant recipients. Numerous factors including rejection episodes, immunosuppressive drugs, weight gain, and other factors raise BP levels post transplant, thus perpetuating HTN risk. Few studies have examined the impact of renal transplantation on blood pressure control. Accordingly, we assessed the fate of BP control and its burden before KTx and during the first 12 months of KTx in 50 consecutive patients with ESRD. Common causes of ESRD among the patients included glomerulonephritis, hypertension, diabetes, PKD, lupus, and glomerulosclerosis. This is a retrospective review. BP was measured by conventional sphygmomanometry. The results are shown in the Table (mean ± SD): To maintain adequate control of HTN after KTx the dosage and number of anti-hypertensive drugs had to be increased. The body weight increased as expected and, therefore, may be a confounding variable. The number of anti-hypertensive drugs needed to maintain BP control actually increased despite the restoration of renal function by KTx. Three cardiovascular complications occurred after KTx during the first 12 months - left subclavian vein thrombosis, non-Q wave MI, and CHF exacerbation. On the basis of the above results and observations, it is concluded that the burden of HTN in ESRD is not alleviated despite KTx. Studies are urgently needed to explore pathophysiological and therapeutic avenues to control and prevent persistent HTN in KTx population. Otherwise, these patients remain at risk for cardiovascular complications.

<table>
<thead>
<tr>
<th></th>
<th>Sitting BP (mm/Hg)</th>
<th>Weight (lbs)</th>
<th>Creatinine (mg/dL)</th>
<th>BUN (mg/dL)</th>
<th>Number of BP Meds</th>
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<tbody>
<tr>
<td>Pre-Transplant (Dialysis)</td>
<td>143/81</td>
<td>78.4</td>
<td>9.6</td>
<td>57.6</td>
<td>1.7</td>
</tr>
<tr>
<td>6 Months Post KTx</td>
<td>133/82</td>
<td>83.5</td>
<td>1.5</td>
<td>22.9</td>
<td>1.9</td>
</tr>
<tr>
<td>12 Months Post KTx</td>
<td>133/81</td>
<td>84.6</td>
<td>1.5</td>
<td>23.1</td>
<td>2</td>
</tr>
</tbody>
</table>

Key Words: Renal Function, Renal Transplant, Persistent Hypertension

SERUM CYSTATIN C LEVEL IS A SENSITIVE MARKER FOR EARLY RENAL DAMAGES IN PATIENTS WITH ESSENTIAL HYPERTENSION
Sumei Watanabe, Takafumi Okura, Jun Liu, Yasunori Tokata, Michtingu Nakamura, Zhao-Hui Yang, Yutaka Kitami, Kunio Hivoda. The Second Department of Internal Medicine, Ehime University School of Medicine, Onsen-gun, Ehime, Japan.

High level of urinary albumin excretion rate (AER) has been associated with the presence of atherosclerotic vascular damages and is an independent risk factor for all causes of death and cardiovascular morbidity and mortality in essential hypertensive patients. Measuring AER is indispensable for hypertensive subjects, however, requires a cumbersome method of 24-hour urine collection. Serum cystatin C, a nonglycosylated 13-kD basic protein, is a member of the cystatin superfamily of endogenous cysteine proteinase inhibitors. Recently, it has been reported to be a useful marker of glomerular filtration rate. In the present study, we investigated the relationship between serum cystatin C level and other markers of renal function in patients with essential hypertension.

Fifty-seven patients with essential hypertension participated in the present study. Patients with diabetes mellitus were excluded. Serum cystatin C level was measured by a particle-enhanced turbidimetric assay. Each patient collected his or her own urine for 24 hours for the determination of AER and creatinine clearance. Twenty-four-hour blood pressure was measured by a cuff-oscillometric method.

Serum cystatin C level was positively correlated with serum creatinine concentration and was negatively correlated with creatinine clearance (r=0.660, p<0.0001; and r=-0.565, p=0.0001, respectively). It was also significantly correlated with AER (r=0.569, p=0.0001), and mean 24-hour systolic blood pressure (r=0.407, p=0.0027). Mean 24-hour systolic blood pressure was independently associated with both AER and serum cystatin C level, whereas HDL-cholesterol was associated only with AER, but not with serum cystatin C level.

The present study first demonstrated that serum cystatin C level is a useful and convenient parameter of the renal function, and may become a sensitive marker of severity for early renal damages in patients with essential hypertension.

Key Words: Essential Hypertension, Cystatin C, Albumin Excretion Rate

THE EFFECT OF STRESS INDUCED CHANGES IN ANGIOTENSIN II ON SODIUM EXCRETION AND BLOOD PRESSURE DURING AND FOLLOWING STRESS IN AFRICAN-AMERICAN YOUTHS

Background: Previous investigators demonstrated (a) enhanced antinatriuresis in response to angiotensin II (Ang II) infusion and (b) correction of impaired sodium regulation during mental stress with angiotensin converting enzyme inhibition. Purpose: The purpose of this study was to determine the relationships between Ang II during an extended period of mental stress (one hour) and both sodium excretion (U Na V) and systolic blood pressure (SBP) during and following the stress period.

Methods: The subjects included 22 African-American youths aged 15-18 years. The subjects were brought into similar levels of sodium balance prior to testing. The five hour protocol included a one hour video game stress period which was preceded and followed by a two hour rest period. Blood samples were collected at minutes 0, 60, 120, 125 (5 minutes into the stress period, 150 (midway through stress period), 180 (end of stress period), 240, and 300. Urine samples were obtained hourly and blood pressure at 15 minute intervals.

Results: U Na V during stress was inversely correlated with Ang II at minutes 125 (r=-0.39; P<0.04), 150 (r=-0.44; P<0.04), and 180 (r=-0.46; P<0.02). The correlations between each of the measures of AII and U Na V during the first hour following stress were also negative but did not reach significance. However, U Na V during the second hour following stress was inversely correlated with Ang II at minutes 125 (r=-0.61; P<0.001), 150 (r=-0.46; P<0.007), and 180 (r=-0.60; P<0.002). SBP during the stress period was inversely related to Ang II at minutes 125 (r=-0.42; P<0.03), 150 (r=-0.56; P<0.01), and 180 (r=-0.38; P<0.05). SBP during the first hour following stress was inversely correlated with Ang II at minutes 125 (r=-0.55; P<0.004), 150 (r=-0.49; P<0.05), and 180 (r=-0.54; P<0.007). Finally, SBP during the second hour following stress was inversely correlated with Ang II at minutes 125 (r=-0.59; P<0.002), 150 (r=-0.54; P<0.01), and 180 (r=-0.51; P<0.01).

Conclusion: These results support the findings of previous studies that Ang II contributes to the control of sodium handling and blood pressure during stress. The results extend this research by demonstrating that Ang II during stress is related to sodium handling and blood pressure following the cessation of the stress. The findings provide support for the hypothesis that stress induced changes in sodium regulation contributes to the development of hypertension in African-Americans.

Key Words: Stress, Sodium, Blood Pressure