# Immediate Impact of Kidney Transplantation on Blood Pressure – Single Center Experience

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### Abstract

Introduction and Aims: Hypertension (HTN) can be either a cause or a consequence of chronic kidney disease (CKD). Kidney transplantation is the most preferred mode of treatment of end stage kidney disease (ESKD). Long term outcome of the renal allograft after kidney transplantation largely depends upon good control of blood pressure (BP). The aim of this study was to observe the status of blood pressure and anti-hypertensive requirement of the kidney transplant recipients in post-kidney transplant hospital stay period.

Methods: This cross-sectional study was done in BIRDEM General Hospital from January 2007 to July 2014. A questionnaire was formed and data were collected from the hospital records. We divided the patients according to primary cause of ESKD in group 1 (due to diabetes mellitus), group 2 (due to chronic glomerulonephritis) and group 3 (patients with hypertension).

Results: Total number of patients was 82, male were 56 and female were 26. Mean age was  $38.49 \pm 10.58$  years. Most of the patients belonged to group 2 (35, 42.68%) followed by

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group 1 (25, 30.48%) and group 3 (22, 26.83%). After kidney transplantation both mean systolic and mean diastolic BP decreased significantly (p-value 0.000). To control BP only 9(10.98%) patients required increased dose of antihypertensive drugs after transplantation. In patients with HTN as primary disease change in number of antihypertensive drugs was not significant (p-value 0.069) unlike to the other two groups. Before kidney transplantation in only 9.76% patients BP could be kept within acceptable reference range with or without anti-hypertensive medication but after transplantation it was 68.29% with less number of drugs.

Conclusion: In this study population, the blood pressure could be kept within acceptable reference range with less number of anti-hypertensive drugs during discharge after kidney transplantation.

*Key words: Anti-hypertensive drugs, hypertension, kidney transplantation.* 

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## Introduction:

The relationship between abnormal blood pressure (BP) and kidney dysfunction was first established in the 19<sup>th</sup> century. The prevalence of hypertension and chronic kidney disease (CKD), and the associated burden of cardiovascular morbidity and mortality, has been dramatically increasing worldwide.<sup>1</sup> High blood pressure or hypertension (HTN) can be either a cause or a consequence of CKD and/or end stage kidney disease (ESKD). HTN may develop early in the course of CKD and can be associated with adverse outcomes such as worsening renal function and development of cardiovascular disease.<sup>1-3</sup> It is a major promoter of the decline in glomerular filtration rate (GFR) in both diabetic and nondiabetic kidney disease. Hypertension is a strong independent risk factor for ESKD.<sup>2,3</sup>

Kidney transplantation is the most preferred mode of treatment for the patients with ESKD. The long term outcome of the kidney allograft largely depends on good control of blood pressure. Normal blood pressure is a good marker of graft survival after renal transplantation, and effective anti-hypertensive treatment reduces the progression of graft damage.<sup>4,5</sup> Our aim was to describe

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and keep record of the status of BP and antihypertensive drug requirement of the kidney recipients in post-kidney transplant hospital stay period to set a baseline parameter for further long term evaluation.

### Methods:

This cross-sectional study was conducted in transplant unit of BIRDEM General Hospital. Data were collected in a preformed questionnaire from the hospital record with the permission from the Director General of the institute. A total number of 82 patients who had undergone live related donor kidney transplantation in BIRDEM General Hospital from January 2007 to July 2014 were included in this study. They were divided into 3 groups according to primary cause of ESKD. Group 1 consisting of patients with diabetes mellitus (DM), group 2 with chronic glomerulonephritis (CGN) and group 3 with HTN as cause of ESKD.

## **Results:**

Among the 82 study subjects, male were 56 and female were 26. Mean age was  $38.49 \pm 10.58$  years. Most of the patients belonged to group 2 (35, 42.68%) followed by group 1 (25, 30.48%) and group 3 (22, 26.83%). Following kidney transplantation there was significant decrease in both systolic (p = 0.000) and diastolic BP (p = 0.000) and requirement of number of anti-hypertensive drugs (table I and II respectively) in all groups during posttransplant discharge from hospital. Increased dose of anti-hypertensive drugs was only required in 9 (10.98%) patients (table III). In patients with HTN as primary disease, the change in the number of antihypertensive drugs was not significant unlike to the other two groups (table IV). Before kidney transplantation, the blood pressure could be kept within acceptable reference range (with or without anti-hypertensive drugs) in only 9.76% patients, but after transplantation it was 68.29% with less number of drugs (table V).

# Table I

Pre and Post-transplant status of blood pressure and requirement of anti-hypertensive drug (N=82)

	Pre-transplant (Pre-Tx)	Post-transplant (Post-Tx)	P Value
Mean Systolic BP(mm of Hg)	$152.14 \pm 17.89$	$130.42 \pm 10.53$	0.000
Mean Diastolic BP(mm of Hg)	$89.70 \pm 9.92$	$80.54 \pm 4.85$	0.000
Mean number of drug(s)	$2.11 \pm 1.12$	$1.69 \pm 0.99$	0.000

**Table II** 

Change in blood pressure in patients with different primary cause of ESKD ( $N=82$ )						
Causes of ESKD	Pre-Tx mean	Post-Tx mean	р	Pre-Tx mean	Post-Tx mean	р
	Systolic BP	Systolic BP	Value	Diastolic BP	Diastolic BP	value
DM (n=25)	$158 \pm 19.68$	133.8±11.11	< 0.0001	88.2±8.5	$79.6 \pm 2.5$	< 0.0001
CGN(n=35)	148.57±17.0	126.29±9.02	< 0.0001	90.43±10.60	80.14±5.88	< 0.0001
HTN (n=22)	150.91±17.16	132.73±10.66	0.0003	89.32±10.50	$82.27 \pm .06$	0.0070

Table III
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Dose changes of anti-hypertensive drugs after kidney transplantation			
Dose of anti-hypertensive drugs	Number of patients (%)		
Unchanged	39 (47.56%)		
Decreased	34 (41.46%)		
Increased	9 (10.98%)		

reduced in those patients.<sup>4</sup> In our study population we also found that immediately after kidney transplantation (on discharge after transplant) BP could be kept well controlled with less number of anti-hypertensive drugs. As control of BP is a good marker of graft survival in kidney transplantation, we could expect better graft function in our patients in the long run. The study had some limitations. It was a single center study, sample size was small and the observation period was only the immediate post-transplant hospital stay periods. A larger multi-center study including large number of patients can be done and still there is scope to observe the long term effect of BP status on renal allograft function among these patients.

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Changes in number of anti-hypertensive drugs in ESRD patients with different primary disease.				
Causes of ESKD(no. of patient) Pre-Tx mean number of		Post-Tx mean number of	Р	
	anti-hypertensive drugs	anti-hypertensive drugs	Value	
DM (25)	$2.28 \pm 1.02$	$1.64 \pm 0.86$	0.005	
CGN(35)	$2.14 \pm 1.19$	$1.83 \pm 1.07$	0.039	
HTN (22)	$1.91 \pm 1.11$	$1.59 \pm 1.05$	0.069	

#### **Table IV**

Blood pressure control before and after kidney transplantation <sup>6</sup>					
No of antihypertensive	BP before tr	ansplant (no of patient)	BP after transplant (no of patient)		
used	>140/90 mm of Hg	140/90 mm of Hg or less	>140/90 mm of Hg	140/90 mm of Hg or less	
0	05	04	00	09	
1	11	00	03	08	
2	32	03	13	22	
3	18	01	08	11	
4	08	00	02	06	
Total	74 (90.24%)	8(9.76%)	26(31.71%)	56(68.29%)	

# Table V

## **Discussion:**

Under normal conditions, renal blood flow (RBF) varies very little within a broad range of systemic mean arterial pressure (80 to 160 mmHg). Increases in BP within this range lead to vasoconstriction of the glomerular afferent arteriole, thereby maintaining RBF and glomerular capillary pressure constant.<sup>3</sup>

The increase in pressure load to the kidney vasculature results in a mechanical stretch of the glomerular capillaries and mesangial cells, which induces a repair response that is mediated by fibrogenic cytokines and angiotensin II. Repetitive injuries and repairs can result in glomerulosclerosis, which is worsened further by local factors such as proteinuria. Hypertension-related mechanisms that are involved in the progression of renal damage include the systemic BP load, the degree to which it is transmitted to the renal microvasculature (i.e. renal autoregulation), and local susceptibility factors to barotrauma, which is the degree of damage for any degree of BP load.<sup>1-3,7</sup>

HTN after kidney transplantation is an important factor in cardiovascular mortality, as well as a risk factor for graft loss. It has not been established whether this is due to the deleterious effect of HTN on the graft or HTN is a marker of an underlying disease.<sup>8</sup>

In healthy person BP follows a characteristic pattern throughout the 24 hour cycle with daytime pressure being higher than the night time pressure. In case of CKD patients on conservative management or patients on haemodialysis or patients on continuous ambulatory peritoneal dialysis, this nocturnal pattern of blood pressure drop is lost.<sup>9-11</sup> Patients lacking this normal nocturnal decrease in BP have a higher incidence of end organ damage.<sup>12,13</sup>

Following successful kidney transplantation several body parameters return to normal physiological level. The prevalence of HTN is high in spite of normally functioning graft.<sup>10,11</sup> Gatzka et al first reported return of normal BP after kidney transplantation in 1995.<sup>14</sup> In 2002, Ravichandran R et al found in his study that before kidney transplantation, BP could be kept in normotensive range (with or without anti-hypertensive drugs) in only 33.33% of their study subjects but after transplantation it was 70.11% and drug requirement also