

Improvement in Post-Transplant Hypertension in Living Donor Renal Transplantation

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Objectives: Since genetic factor determines part of hypertensive phenotype, we aim to demonstrate the role of transplanted kidney from normotensive living donors in post-transplant hypertension (HTN).

Methods: From 1.5-year-medical record review yielded 103 kidney transplant recipients in whom living-donor renal transplantation (LDRT) was performed in 32 (15 living-related renal transplantation (LRRT) and 17 living-unrelated renal transplantation (LURT)).

Results: Of all 32 recipients, mean age was 51.30 years old (21.42-79.53) and 50% were male. Mean duration of follow-up was 8.4 months (0.63-16.33). Up to 93.75% of recipients had pre-transplant hypertension, and 56.25% became non-hypertensive after transplantation, which was defined as SBP≤140, DBP≤90, or being on ≤2 BP agents regardless SBP or DBP (Figure 1). Mean post-transplant systolic blood pressure (SBP) was lower than pre-transplant SBP but not statistically significant (132.88±2.54 vs.134.75±3.01, p= 0.6366) as same as mean DBP (77.84±1.88 vs. 82.25±2.39, p= 0.1520). The number of pre- and post-transplant blood pressure medications was 1.94 and 1.28, respectively. in LRRT group, 5 of 13 (38.46%) pre-transplant hypertensive patients became normotensive while 11 of 17 (64.71%) patients in LURT group were non-hypertensive (Figure 2). Mean post-transplant SBP was higher than mean pre-transplant SBP in LRRT group (133.73±3.33 vs.129.67±4.40, p=0.4680); however, mean post-transplant DBP in LRRT group (77.93±2.68 vs.79.40±3.20, p=0.7273) as well as mean SBP (132.12±3.85 vs. 139.24±3.93, p=0.2049) and mean DBP (77.76±2.71 vs. 84.76±3.48, p=0.1223) in LURT were lower than those during pre-transplant periods. The mean number of antihypertensive medications was decreased in post-transplant compared to pre-transplant in both LRRT (1±0.24 vs.1.73±0.33, p=0.0844) and LURT (1.53±0.12 vs.2.12±0.28, p=0.0616) groups.

Conclusion: Hypertension was resolved in more than half of the pre-transplant hypertensive patients after kidney transplantation. Since higher number of LURT recipients becomes normotensive, the possibility of hypertensive genotype in living-related donor kidneys may contribute to post-transplant HTN in some LRRT recipients.

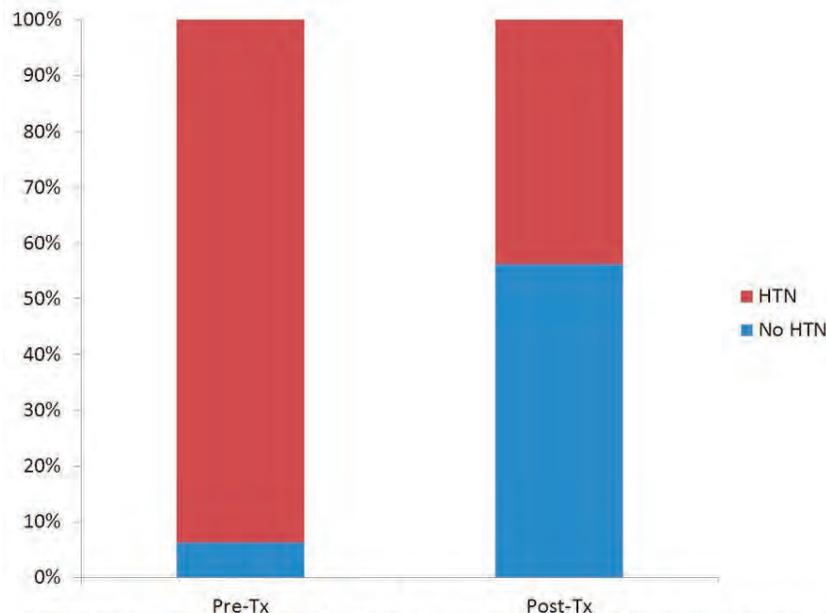


Figure 1: Lower prevalence of hypertension after living donor renal transplantation