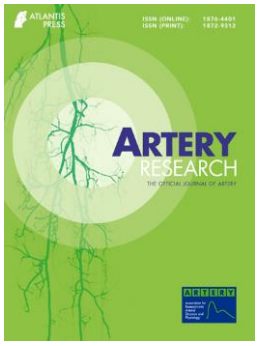

Artery Research

ISSN (Online): 1876-4401 ISSN (Print): 1872-9312
Journal Home Page: https://www.atlantis-press.com/journals/artres

PO-04: REBOUND WEIGHT GAIN AND BLOOD PRESSURE CONTROL AFTER LIVING KIDNEY DONATION AND KIDNEY TRANSPLANTATION

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To cite this article: Ekamol Tantisattamo, Weera Sukhumthammarat, Prapaipan Putthapiban, Wasawat Vutthikraivit, Siwadon Pitukweerakul (2016) PO-04: REBOUND WEIGHT GAIN AND BLOOD PRESSURE CONTROL AFTER LIVING KIDNEY DONATION AND KIDNEY TRANSPLANTATION, Artery Research 16:C, 90–91, DOI: https://doi.org/10.1016/j.artres.2016.08.012

To link to this article: https://doi.org/10.1016/j.artres.2016.08.012

Published online: 7 December 2019

Table 1 Cardiovascular responses to mental-stress among older men and women. (Mean ± SD).

Variable	*Men (n=46)*		*Women (n=45)*		*Effects*		
	Rest	Mental-stress	Rest	Mental-stress	Sex	Time	SxT
Brachial SP (mmHg)	124 ± 12	139 ± 16	127 ± 14	145 ± 20	**0.035**	**0.001**	0.545
Brachial DP (mmHg)	79 ± 7	86 ± 7	79 ± 7	86 ± 9	0.769	**0.001**	0.677
Brachial PP (mmHg)	45 ± 8	53 ± 12	49 ± 9	59 ± 12	**0.004**	**0.001**	0.558
Carotid SP (mmHg)	116 ± 12	129 ± 17	118 ± 13	131 ± 16	0.306	**0.001**	0.769
Carotid PP (mmHg)	37 ± 9	43 ± 14	39 ± 9	45 ± 10	0.218	**0.001**	0.903
HR ($b \cdot min^{-1}$)	60 ± 10	66 ± 10	63 ± 9	70 ± 13	**0.023**	**0.001**	0.735
PWV ($m \cdot s^{-1}$)	10.2 ± 2.6	11.2 ± 2.6	9.3 ± 2.7	10.1 ± 3.1	**0.020**	**0.023**	0.763
CCA Ep (kPa)	100.58 ± 35.40	125.01 ± 50.23	107.89 ± 46.99	123.64 ± 55.57	0.674	**0.005**	0.540

SP, systolic pressure; DP, diastolic pressure; PP, pulse pressure; HR, heart rate; PWV, pulse wave velocity; CCA, common carotid artery; Ep, elastic modulus; SxT, sex-by-time interaction.

PO-03
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 (1.33.73+/-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.

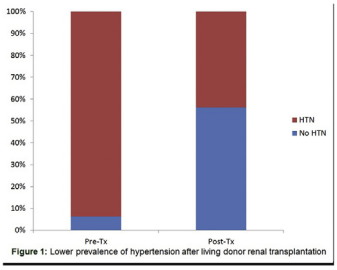

Figure 1: Lower prevalence of hypertension after living donor renal transplantation

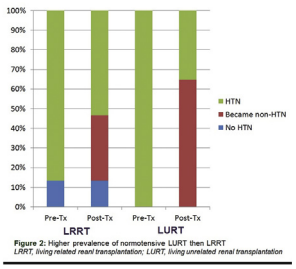

Figure 2: Higher prevalence of normotensive LURT then LRRT
LRRT, living related renal transplantation; LURT, living unrelated renal transplantation

PO-04
REBOUND WEIGHT GAIN AND BLOOD PRESSURE CONTROL AFTER LIVING KIDNEY DONATION AND KIDNEY TRANSPLANTATION

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Objective: Overweight and obesity are known risk factors of hypertension in both donors and recipients after kidney donation and transplantation, respectively. We aim to study the correlation between blood pressure (BP) and body mass index (BMI) in donor post-donation and in recipients post-transplantation.
Methods: A consecutive 24 paired living kidney donors and recipients were reviewed. Demographic data, systolic blood pressure (SBP), diastolic blood pressure (DBP), and BMI were collected.
Results: Of all 24 donors and recipients, donor's trends to be younger than their recipients (mean age 46.54±2.81 vs. 50.32±3.16 years old). Half of the donors and 54.17% (13/24) of the recipients were male. In donor group, mean SBP, but not DBP decreased overtime after donation (SBP 125.58±2.9 vs. 123.69±1.97; p=0.5924 vs. 121.33±3.02; p=0.3181. DBP 74.92±1.7 vs. 75.73±1.12; p=0.6926 vs. 76.85±1.82; p=0.4437). However, BMI decreased at 2-week post-donation, but rebounded above pre-donation BMI at 6 months (BMI 28.19±0.87 vs. 28±0.82; p=0.8750 vs. 28.92±1.03; p=0.5884) (Figure

1A). For recipient group, mean SBP, DBP, and BMI trended down after transplantation. However, these values increased to almost the same levels of pre-transplantation at 3-month post-transplant, and only DBP and BMI trended up beyond pre-transplant values at 6-month post-transplant (Figure 1B). Among 24 donors, 13 and 11 patients were living-related (LRD) and living unrelated donors (LUD), respectively. SBP, but not DBP continuously decreased in both LRD and LUD. Conversely, BMI was up trending in LRD, but decreased at 2-week post-donation, and then rebounded at 6-month (Figure 2A and 2B). Of all 24 recipients, 13 and 11 patients were living-related (LRR) and living unrelated renal transplant recipients (LUR), respectively. SBP, DBP, and BMI in LRR decreased until 1-month post-transplant and increased to above pre-transplant levels at 6-month post-transplant without statistical significance (Figure 2C). LUR group had the same patterns of SBP, DBP, and BMI, but SBP and DBP at 1-week and 1-month post-transplantation almost significantly decreased from the pre-transplant levels (Figure 2D).

Conclusion: BP and BMI in both donors and recipients seem to be positively correlated, and BMI rebounded beyond the pre-donation and pre-transplant levels. Early post-transplant SBP and DBP appear to be better improved in LUR than LRR group.

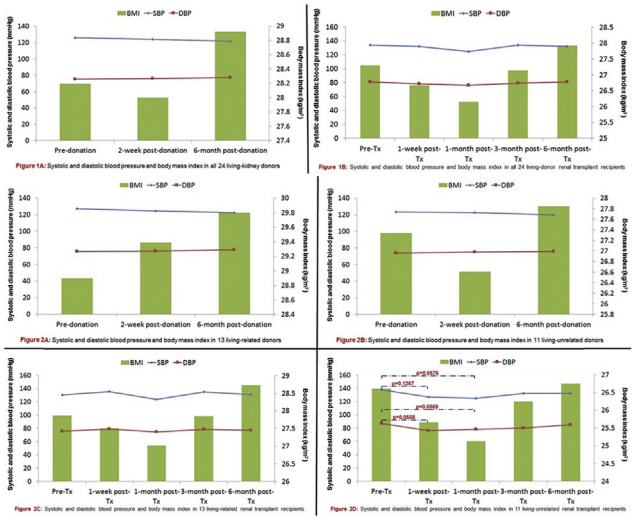

Figure 1A: Systolic and diastolic blood pressure and body mass index in all 24 living-kidney donors

Figure 1B: Systolic and diastolic blood pressure and body mass index in all 24 living-donor renal transplant recipients

Figure 2A: Systolic and diastolic blood pressure and body mass index in 13 living-related donors

Figure 2B: Systolic and diastolic blood pressure and body mass index in 11 living-unrelated donors

Figure 2C: Systolic and diastolic blood pressure and body mass index in 13 living-related renal transplant recipients

Figure 2D: Systolic and diastolic blood pressure and body mass index in 11 living-unrelated renal transplant recipients

young obese adults with normal metabolic profile still exhibited comparable central hemodynamics and arterial stiffness as normal-weight adults, suggesting preserved vascular health despite initial carotid vascular remodeling.

Table 1 Comparisons of central hemodynamics and arterial stiffness in normal-weight and obese adults.

	Normal-Weight (n=11)	Obese (n=13)
Percent body fat (%) *	31.1±1.7	41.9±1.7
Total cholesterol	180±14	176±11
High density lipoprotein (mg/dL)	62±3	51±5
Low density lipoprotein (mg/dL)	103±14	110±12
Triglycerides (mg/dL)	100±19	85±11
Glucose (mg/dL)	96±4	98±5
Brachial SBP (mmHg)	109±1	109±3
Brachial DBP (mmHg)	70±2	73±2
Aortic SBP (mmHg)	93 ±3	96±3
Aortic DBP (mmHg)	65±2	69±2
HR (bpm)	62±2	58±3
cIMT (mm) *	0.37±0.01	0.44±0.02
CAVI	6.0±0.2	6.0±0.2
β-Stiffness	5.5±0.4	5.2±0.4
Ep (kPA)	66.3±5.3	62.8±5.3
AC (%)	1.02±0.07	1.20±0.09
AIx (%)	7±4	6±3
AIx@75 (%)	0±3	-2±3
AP (mmHg)	2±1	2±1
FPH (mmHg)	25±1	25±1
RPH (mmHg)	38±6	34±6
RI (%)	19±4	24±9

Data are mean±SE. BMI, body mass index; cIMT, carotid intima-media thickness; CAVI, cardio-ankle vascular index; β-stiffness, beta stiffness; Ep, elastic modulas; AC, arterial compliance; SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate; AIx, augmentation index; AIx@75, augmentation index normalized to heart rate of 75 bpm; FPH, forward pulse height; RPH, reflected pulse height; RI, reflection index. *significant group difference based on an independent *t*-test ($P<0.05$).

PO-09
CENTRAL HEMODYNAMICS AND ARTERIAL STIFFNESS IN YOUNG OBESE ADULTS: THE PRELIMINARY FINDING

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Changes in central hemodynamics and arterial stiffness are associated with augmented cardiovascular risks and have been reported in obese adults with metabolic syndrome. It is unclear whether this observation may also be present in young healthy obese adults with normal metabolic profile.

Objectives: To compare measures of central hemodynamics and arterial stiffness in young normal-weight vs. obese adults.

Methods: There were 11 normal-weight (female=6; age 25±2 yrs; BMI 22.4±0.6 kg/m^2) and 13 obese adults (female=6; age 27±1 yrs; BMI 32.7±0.6 kg/m^2). Central hemodynamics were measured using SphygmoCor and wave separation analysis. Ultrasonography was used to measure carotid intima-media thickness (cIMT) and arterial stiffness (beta stiffness (β), elastic modulus (Ep), arterial compliance (AC)). Cardio-ankle vascular index (CAVI) was measured using VaSera and is another index reflecting the stiffness of the artery from the heart to ankles. Percent fat was determined using DEXA.

Results: Obese adults exhibited higher percent body fat and cIMT than normal-weight adults ($P<0.05$), with no group differences in metabolic profile. No group differences were observed for brachial and aortic blood pressures, heart rate, arterial stiffness, and wave separation variables.

Conclusion: The larger carotid intima-media thickness in young obese adults suggest early remodeling of the vasculature as a result of obesity. However,

PO-10
VASCULAR FUNCTION IN INDIVIDUALS WITH DOWN SYNDROME

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Individuals with Down syndrome (DS) experience premature aging. Arterial stiffness increases with advancing biological age and predicts cardiovascular disease. However, only limited studies investigated arterial function in individuals with DS. Thus, the impact of DS on vascular function still remains poorly understood.

Purpose: To compare vascular function between individuals with and without DS (control).

Methods: Twenty-seven volunteers (DS=13, Control=14) participated in this study. Central arterial stiffness indices (β-stiffness, Ep and circumferential strain) were measured by carotid ultrasonography and analyzed with B-mode, echo tracking and strain analysis. Cardio-ankle Vascular Index (CAVI) and carotid blood pressure (carBP) were measured using a limb cuff system and applanation tonometry (SphygmoCor), respectively. In addition, heart rate (HR) was recorded by finger photoplethymography.

Results: There were significant differences in CAVI (lower) and circumferential strain (higher) in individuals with DS compared to individuals without DS ($p<0.05$). No group differences were observed for β-stiffness and Ep.

Conclusions: Our results suggest that individuals with DS have lower arterial stiffness than that of individuals without DS. Interestingly, circumferential carotid strain was greater in persons with DS, with no differences in β-stiffness, suggesting the greater strain may have been a function of greater pulse pressure in individuals with DS.