DIETARY AND URINARY EXCRETION OF SODIUM AND POTASSIUM ASSOCIATED WITH BLOOD PRESSURE CONTROL IN TREATED HYPERTENSIVE KIDNEY TRANSPLANT PATIENTS

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Background:

In kidney transplant recipients (ktr),hypertension is not only a major risk for cardiovascular (cv) complications but also for graft failure. Blood pressure (BP) control is therefore mandatory.

Office BP (OBP) remains the most frequently used for clinical decision, but out-of-clinic measurements such as home BP (HBP) have brought a significant improvement in the management of hypertension. Although still on debate, sodium is a modifiable risk factor since many studies accounted for a decrease of BP with a sodium (Na+) restricted diet. Increased potassium (K+) intake has also been recommended for prevention and treatment of hypertension. We investigated the relations between BP control (in office and at home) and the urinary Na+ and K+ excretion as well as their diet consumption in treated hypertensive ktr.

Methods:

Baseline characteristics of ktr are described in **table1.**

BP control was defined by an OBP<140/90 mmHg and a HBP<135/85 mmHg (<130/80 if diabetes for both techniques) in 70 ktr treated with antihypertensive drugs. OBP (mean of 3 measurements) and HBP (7 days recording;2x in the morning and the evening; mean HBP= mean from day 2 to day7) were measured with validated oscillometric device (Omron M6). Na+ and K+ excretion was measured on two 24-h collections 7 days apart. While collecting urine,recipients wrote on a 24-h food record their food and beverage consumptions (free diet).Diet quantitative analysis was done with Nubel Belgian food table.

Non parametric Mann–Whitney *U* test was used in table 2

Table 1: Baseline characteristics of kt recipients

	Mean ± sd [min-max]
N	70
Men/women (N)	43/27
Age (years)	56 ± 11.5 [33-76]
Graft survival (years)	7.0 ± 6.6 [1-25]
Hemodialysis vintage (years)	2.7 ± 3.7 [1 m-7.9 y]
GFR (ml/min)	65.6 ± 24 [26-133]
BMI (kg/m²) BMI ≥30	25.8 ± 4.7 [16-37] 15 (21%)
Diabetes (N,%)	19 (27)
Current smokers (N,%)	9 (13)
Office SBP (mmHg)	136 ± 14 [107-175]
Office DBP (mmHg)	83 ± 12 [50-108]
24h urinary Na+ (mmol)	167 ± 83 [45-463]
24h urinary K+ (mmol)	62 ± 24 [25-134]
NaCl (g/24h)	9.9 ± 4.9 [2.6-27]
Urinary Na+/K+ ratio	2.9 ± 1.2 [0.8-6]

Table 2: : Comparisons between controlled and uncontrolled patients- reference to office and home BP.

	OBP<140 and <90 **and** HBP<135 and <85	OBP ≥140 and/or ≥90 **and** HBP≥135 and/or ≥85	P
N	15	34	
Age (years)	53.4 ± 9.8	56.8 ± 11	>0.05
Graft survival (years)	5.3 ± 4	6.3 ± 6	>0.05
Nephrectomy (native kidney)(N)	1	5	>0.05
Donor			
Age (years)	40 ±12.4 [17-54]	42.7 ±16.5 [15-67]	>0.05
Hypertension, (N)	3	5	>0.05
Cadaveric, (N)	14	34	>0.05
Living, (N)	1	0	>0.05
Kt recipients			
Office SBP (mmHg)	125 ± 9	146 ± 12	<0.00001
Office DBP (mmHg)	77 ± 8	87 ± 13	0.004
Office heart rate (beat/min)	67 ± 9	69 ± 12	>0.05
Home SBP (mmHg)	123 ± 7	141 ± 10	<0.00001
Home DBP (mmHg)	77 ± 6	85 ± 11	0.0005
Home heart rate (beat/min)	68 ± 5	70 ± 12	>0.05

	OBP<140 and <90 **and** HBP<135 and <85	OBP ≥140 and/or ≥90 **and** HBP≥135 and/or ≥85	P
N	15	34	
Hemodialysis vintage (years)	4.0 ± 7	2.6 ± 2	>0.05
Diabetes (N)	1	13	0.04
BMI (kg/m²)	24 ± 4.6	26 ± 4.9	>0.05
Antihypertensive drugs (N)	2.1 ± 1	2.1 ± 1	>0.05
Diuretics	4 (26.7%)	10 (29%)	>0.05
GFR(ml/min)	63.2 ± 28	61.5 ± 21	>0.05
Calcineurin inhibitors N(%) Cyclosporine Tacrolimus	14 (93) 2 (13) 12 (80)	31 (91) 14 (41) 17 (50)	> 0.05 0.045
Prednisone (daily dose)	3.9 ± 1.2	4.7 ± 1	> 0.05
K+ (blood;mmol/l)	3.9 ± 0.3 [3.1-4.5]	4 ± 0.4 [3.2-5]	>0.05
Urinary excretion			
Diuresis (ml)	2431 ± 719	2457 ± 919	>0.05
Na^+ (mmol/24h)[mg]	154 ± 93 [3542]	162 ± 88 [3726]	>0.05
K^+ (mmol/24h)[mg]	68 ± 14[2659]	54 ± 20[2111]	0.029
Na^+/K^+ ratio	2.3 ± 1.2	3.2 ± 1.3	0.052
NaCl (g/24h)	9.1 ± 5.4	9.6 ± 5.2	>0.05
Diet intakes			
Na^+ (mg/24h)*	2339 ± 1067	1766 ± 695	>0.05
K^+ (mg/24h)	3279 ± 753	2208 ± 720	0.009

* Without added salt for cooking and at table

Results:

Using an agreement between OBP and HBP, 15 ktr (21%) were identified as controlled while 34 (49%) remained hypertensive **[table 2]** (disagreement between both BP techniques was observed in 16 ktr with masked HT and 6 with white-coat HT). Major confounding parameters susceptible to interfere with the BP regulation did not differ between groups except that a higher proportion of uncontrolled recipients was treated with cyclosporine (P=0.045) and the majority of ktr with diabetes were uncontrolled. Although both groups consumed higher amounts of salt (between 9 and10g) than recommended (5-6g/day), they excreted a similar quantity of Na+. Uncontrolled ktr excreted less K+ (P=0.029) and had significantly lower k+ intakes (P=0.009), resulting in a higher urinary Na+/K+ ratio. Proportions of ktr treated with diuretics (60 to 67% were thiazide) did not differ between groups. Home systolic BP (but not diastolic) was inversely and significantly correlated to urinary K+ after controlling for age, BMI and urinary Na+ **[table 3],** and a positive relation was noted with urinary Na+ after controlling for age, BMI and urinary K+.

Two 24-h urine collections as well as food intakes being available,at baseline visit and at the end of the HBP recording, a comparison of Na+ and K+ has been performed to assess the short term reproducibility.

The paired comparisons between first and second urine collections and between first and second food recall did not show any statistical difference either for urinary Na+ and K+ and for Na+ and K+ consumption.

Table 3:Correlations (Pearson correlation)	Partial correlation coefficient	P
N=49		
Home SBP with urinary Na+ (Controlled for: age, BMI and urinary K+)	0.32	0.042
Home SBP with urinary K+ (Controlled for: age, BMI and urinary Na+)	- 0.46	0.002

Conclusions:

Half of the treated hypertensive ktr remained uncontrolled in office and at home. Although it is known that not all individuals are salt-sensitive and therefore will not respond to a restricted salt diet, our results support the idea of a potential relation between blood pressure and the urinary and dietary sodium/potassium ratio in treated hypertensive uncontrolled kidney transplant recipients. Restoring a well-balanced sodium/potassium ratio intakes could be a non-pharmacological opportunity to improve the BP control.