RESEARCH ARTICLE

Office Assessed Blood Pressure and Ambulatory Blood Pressure Monitoring in Chronic Kidney Disease Patients Versus Kidney Transplant Recipients

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How reliable is office assessed blood pressure (BP) in chronic kidney disease (CKD) patients and kidney transplant (KTx) recipients is yet to be determined, although the diagnosis of arterial hypertension has been based on these measurements. The aim of this study was to investigate the potential differences between office assessed BP and ambulatory blood pressure monitoring (ABPM) in CKD patients and KTx recipients. We conducted a prospective study which enrolled 45 patients. Morning and evening seated office BPs were assessed using a sphygmomanometer at 5 consecutive outpatient visits. A mean systolic BP (SBP) and diastolic BP (DBP) was calculated. Ambulatory blood pressure was measured over 24 hours using a Meditech ABPM-05 device. Office SBP was statistically significant higher in CKD patients than KTx recipients both in the morning and evening (p=0.0433 and p=0.0066 respectively). ABPM showed higher night-time SBPs (p=0.0445) and higher overall, day-time and night-time DBPs in KTX recipients (p=0.0001, p=0.0006, p<0.0001 respectively). In CKD patients, office SBPs and DBPs are significantly higher than overall SBPs and DBPs as assessed by 24hr ABPM. Office BP monitoring as assessed by clinician is acceptable but tends to overestimate BP in both CKD and KTx study groups.

Keywords: office blood pressure, ambulatory blood pressure monitoring, chronic kidney disease, kidney transplant

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Introduction

It is a well known fact that arterial hypertension is prevalent following renal transplantation and its presence could predict chronic allograft dysfunction and patient survival rates [1-2]. Usually, the diagnosis of arterial hypertension is set using measurements performed at the physician's office, recordings traditionally called office blood pressure (BP). How reliable is office assessed blood pressure in chronic kidney disease (CKD) patients and kidney transplant (KTx) recipients is yet to be determined, as it was shown before that office BP tends to overestimate blood pressure control in renal transplantation [3]. On the other hand, ambulatory blood pressure monitoring (ABPM) seems to offer a more reliable view over arterial hypertension both in CKD and KTx patients [4-5] and also a prognostic superiority [6]. Due to the pro-hypertensive potential of immunosuppressive drugs [7-8], blood pressure control is one of the major targets for outcome management in KTx patients [9]. The aim of this study was to investigate the potential differences between office assessed BP and ambulatory blood pressure monitoring (ABPM) in CKD patients and KTx recipients.

Methods

We conducted a prospective observational study enroling 45 patients who presented between February and November 2014 at the Nephrology Unit of the County Clinic Hospital Tirgu Mures. Among these patients, 21 were diagnosed with CKD and 24 patients had a history of kidney transplantation. Office BP was assessed by the same attending physician in a proper and quiet environment, using a calibrated mercury sphygmomanometer, with the patient in a sitting position, at 5 consecutive outpatient visits, both in the morning and evening, after a prior rest period of 10 minutes. In patients whose arteriovenous fistula was still functioning, the measurement was performed at the contra lateral arm. A mean systolic blood pressure (SBP) and diastolic blood pressure (DBP) was further calculated. Twentyfour hours ambulatory blood pressure monitoring was performed using a Meditech ABPM-05 device. Three daytime (6 am-10 pm) measurements per hour and two night-time (10 pm-6 am) measurements were recorded. The cuff was adjusted according to the circumference of the arm. We considered valid only recordings with at least 85% successful readings. Dipping, non-dipping and rising blood pressure patterns were defined according to the ESH Practice Guidelines for ambulatory blood pressure monitoring as nocturnal systolic and diastolic BP fall >10% of daytime values for the dipping pattern, no reduction of BP values for the non-dipping pattern and as an increase in nocturnal systolic and/or diastolic BP values for the risingpattern. Also, definitions for ABPM derived parameters (BP load, morning surge, BP variability) were considered accordingly [10]. Demographic, clinical and laboratory data were collected for all selected patients. Statistical analysis was performed using Graph Pad Prism 6.0, comparisons between groups were tested by Student's T test and statistical

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significance was defined as p<0.05. The design of this study was approved by the Ethics Committee of the hospital and all participants have provided written informed consent prior to being enrolled in the study.

Results

We included in our study 45 adult patients, out of which 24 patients who underwent kidney transplantation, with no limit regarding the amount of time elapsed from surgery, average time being 75.42±10.41 months. At inclusion, mean serum creatinine was 1.565±0.1265 mg per dl. The main cause for end stage renal disease (ESRD) in KTx patients was glomerular nephropathy (58.33%). We had 54.17% of recipients with a brain dead donor graft.

Twentyone patients which were previously diagnosed with CKD were included in the study. Mean serum creatinine was 2.756±0.4536 mg per dl at inclusion. The main causes of ESRD in CKD group were diabetes and arterial hypertension, closely followed by glomerular nephropathies. Demographic, clinical and laboratory characteristics of both groups are presented in Table I and II.

Serum creatinine levels were significantly higher in CKD group than KTx group (p=0.0105). Although the mean excretion of proteins was higher in CKD group, no statistical significant relationship was found between the two groups.

Morning and evening assessed mean office SBPs were significantly higher in CKD group than in KTx group (p=0.0433 and p=0.0066 respectively).

ABPM identified 42.85% non-dipping patterns in the CKD group and 66.67% in the KTx group, while rising BP pattern was found in 9.53% in the CKD group and 20.83% in the KTx group. Other results can be followed in Table III.

n=24 (% from total) Age (years) 46.13 \pm 2.103 Body weight (kg) 79.17 \pm 3.588 Gender (%males) 16 (66.66%) Time since transplantation (months) 75.42 \pm 10.41 Donor Type 79 Brain Dead 13 (54.17%) Living 11 (45.83%) Graft Position 75.42 \pm 10.41 Left Iliac Fossa 9 (37.5%) Right Iliac Fossa 9 (37.5%) Right Iliac Fossa 15 (62.5%) Cause for End Stage Renal Disease 6 Glomerular 14 (58.33%) Tubulo-interstitial 2 (8.33%) Cystic 4 (16.66%) Lupus nephropathy 1 (4.16%) Other 3 (12.52%) Creatinine(mg/dl) 1.565 \pm 0.1265 eGFR(ml/min/1.73m²) 55.37 \pm 4.668 Proteinuria(g/day) 0.7789 \pm 0.2464		
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Lupus nephropathy 1 (4.16%) Other 3 (12.52%) Creatinine(mg/dl) 1.565±0.1265 eGFR(ml/min/1.73m²) 55.37±4.668	Tubulo-interstitial	2 (8.33%)
Other 3 (12.52%) Creatinine(mg/dl) 1.565±0.1265 eGFR(ml/min/1.73m²) 55.37±4.668	Cystic	4 (16.66%)
Creatinine(mg/dl) 1.565±0.1265 eGFR(ml/min/1.73m²) 55.37±4.668	Lupus nephropathy	1 (4.16%)
eGFR(ml/min/1.73m ²) 55.37±4.668	Other	3 (12.52%)
	Creatinine(mg/dl)	1.565±0.1265
Proteinuria(g/day) 0.7789 ± 0.2464	eGFR(ml/min/1.73m ²)	55.37±4.668
	Proteinuria(g/day)	0.7789 ± 0.2464

Table II. Main characteristics of CKD patients (mean±SEM)

	n=21 (% from total)	
Age (years)	63.05±2.467	
Body weight (kg)	81.95±2.583	
Gender (%males)	11 (52.38%)	
Cause for End Stage Renal Disease		
Glomerular	4 (19.04%)	
Tubulo-interstitial	1 (4.76%)	
Hypertension	5 (23.81%)	
Diabetes	7 (33.33%)	
Hyperuricemia	2 (9.53%)	
Other	2 (9.53%)	
Creatinine(mg/dl)	2.756±0.4536	
eGFR(ml/min/1.73m ²)	36.96±5.853	
Proteinuria(g/day)	1.051±0.1939	

ABPM revealed mean night-time SBPs significantly higher in the KTx group (p=0.0445). Mean overall, daytime and night-time DBPs were statistically significant higher in the KTx group than in the CKD group (p=0.0001, p=0.0006 and p<0.0001 respectively).

Furthermore, as for the ABPM derived parameters, BP variability and morning surge were statistically signifficant higher in the CKD group (p=0,0458 and p=0,0059 respectively). However, no signifficant association could be established between blood pressure loads in the studied groups.

Table III. Blood pressure patterns(CKD vs KTx, mean ± SEM)

	CKD (n=21)	KTX (n=24)
Office SBP (mmHg)		
Morning	141.7 ± 3.600	132.5 ± 2.715
Evening	139.8 ± 2.756	130.0 ± 2.078
Office DBP(mmHg)		
Morning	81.90 ± 1.769	83.96 ± 1.340
Evening	81.86 ± 1.720	78.21 ± 0.8231
ABPM Average SBP (mmHg)	125.6 ± 2.136	130.2 ± 2.788
ABPM Daytime SBP (mmHg)	128.8 ± 2.341	131.9 ± 2.729
ABPM Night Time SBP (mmHg)	118.7 ± 2.244	127.0 ± 3.233
ABPM Average DBP (mmHg)	66.57 ± 2.285	79.58 ± 2.097
ABPM Daytime DBP (mmHg)	69.95 ± 2.506	81.96 ± 2.086
ABPM Night Time DBP (mmHg)	60.76 ± 2.098	74.79 ± 2.293
BP Patterns		
Dipping	10 (47.62%)	2 (8.33%)
Non-dipping	9 (42.85%)	16 (66.67%)
Rising	2 (9.53%)	5 (20.83%)
Morning surge	15,89 ± 2,732	6,36 ± 1,820

In CKD group, morning and evening mean office assessed SBPs were significantly higher than overall average SBPs on 24 hr ABPM (p=0.0003 and p<0.0001 respectively). Consecutively, morning and evening mean office assessed DBPs were also significantly higher than average overall DBPs as recorded by ABPM (p<0.0001). In KTx group, morning mean office DBP was significantly higher (p=0.0370) than overall average DBP as assessed by 24hr ABPM. No other statistic significant relationship could be identified between office BP and 24hr ABPM values in the KTx group.

Discussion

Arterial hypertension, as the main cause for cardiovascular events, is a strong predictor of graft dysfunction and mortality among patients with renal transplantation [11]. Meanwhile, authors worldwide agree that the prevalence of arterial hypertension in pre-dialysis CKD patients can reach from 43 to95%[12,13] and stands as an important risk factor for progression and cardiovascular disease [14]. Ambulatory blood pressure monitoring stands as an useful tool in arterial hypertension diagnosis protocol [15,16] and seems to be superior to home or office blood pressure monitoring due to a lower risk of masked or white-coat hypertension, although this fact remains controversial.

In our study, both morning and evening office SBP and DBP values were significant higher than overall SBP and DBP values as assessed by 24 hour ABPM in CKD patients, even though we performed 5 morning and 5 evening BP measurements at different time points, which suggests that office BP measurements have an overestimating tendency in our CKD patients. Hence, as we encounter these disagreements, performing both office BP and ABPM is necessary for an accurate diagnosis. Also, we determined that CKD patients had a signifficant higher morning surge and BP variability than KTx recipients, thus being at an increased risk for cardiovascular events, fact that might be explained considering issues of lower adherence to antihypertensive therapy in this category of patients [17]. Significantly increased levels of serum creatinine and higher protein excretion, as found in our CKD group, could also point to poorly controlled BP values in these patients.

In our renal transplant recipients, morning mean office assessed DBPs were higher than overall average DBPs on 24hr ABPM, with no other statistically significant relationship between performed measurements, which suggests that in this particular group, office BP and 24 hour monitoring might have the same prognostic potential. Nonetheless, 24 hr monitoring should be performed routinely [18,19] in this category of patients, due to involvement of immunosuppressant drugs and pro-inflammatory status in the development of arterial hypertension [20,21,22].

The main limitations of this study is the small population size. Other studies, on larger cohorts, are necessary in order to clarify the advantages and disadvantages of office versus ambulatory blood pressure monitoring in patients with renal impairment.

Conclusions

Although office BP tends to overestimate BP burden, both office BP and 24 hr ABPM are useful tools for the management of hypertension in CKD and KTx patients.

Aknowledgment

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References

- Opelz G, Wujciak T, Ritz E Association of chronic kidney graft failure with recipient blood pressure. Kidney International, 1998; 53:217–222.
- Mange KC, Cizman B, Joffe M, Feldman HI Arterial hypertension and renal allograft survival. Journal of the American Medical Association, 2000;283:633–638.
- Stenehjem AE, Gudmundsdottir H, Os I Office blood pressure measurements overestimate blood pressure control in renal transplant patients. Blood Pressure Monitoring, 2006;11:125–133.
- Iimuro S, Imai E, Watanabe T, et al Chronic Kidney Disease Japan Cohort Study Group. Clinical correlates of ambulatory BP monitoring among patients with CKD, 2013;8:721-730.
- Czyżewski Ł, Wyzgał J, Kołek A Evaluation of selected risk factors of cardiovascular diseases among patients after kidney transplantation, with particular focus on the role of 24-hour automatic blood pressure measurement in the diagnosis of hypertension: an introductory report. Ann Transplant, 2014;19:188-198.
- Sarafidis PA, Rumjon A, Macdougall IC Ambulatory blood pressure monitoring: An Invaluable Tool Comes of Age for Patients with Chronic Kidney Disease? Am J Nephrol, 2012;35:238–241.
- Wang J, Guo R, Liu S, et al Molecular mechanisms of FK506-induced hypertension in solid organ transplantation patients, 2014;127:3645-3650.
- Xue W, Zhang Q, Xu Y, Wang W, Zhang X, Hu X Effects of tacrolimus and cyclosporine treatment on metabolic syndrome and cardiovascular risk factors after renal transplantation: a meta-analysis. Chin Med J (Engl), 2014;127:2376-2381.
- Ponticelli C, Cucchiari D, Graziani G. Hypertension in kidney transplant recipients. Transpl Int, 2011;24:523-533.
- Parati G, Stergiou G, O'Brien E, et al European Society of Hypertension practice guidelines for ambulatory blood pressure monitoring. J Hypertens, 2014;32:1359-1366.
- Paoletti E, Gherzi M, Amidone M, Massarino F, Cannella G Association of arterial hypertension with renal target organ damage in kidney transplant recipients: the predictive role of ambulatory blood pressure monitoring. Transplantation, 2009;87:1864–1869.
- SarafidisPA, Sharpe CC, Wood E, et al Prevalence, patterns of treatment, and control ofhypertensionin predialysis patients with chronic kidney disease. Nephron Clin Pract, 2012;120:c147-155.
- Farag YM, Mittal BV, Keithi-Reddy SR, et al Burden and predictors of hypertension in India: results of SEEK (Screening and Early Evaluation of Kidney Disease) study. BMC Nephrol, 2014;15:42.
- Alani H, Tamimi A, Tamimi N, et al Cardiovascular co-morbidity in chronic kidney disease: Current knowledge and future research needs. World J Nephrol, 2014;3:156-168.
- White WB Ambulatory blood-pressure monitoring in clinical practice. N Engl J Med, 2003;348:2377–2378.
- 16. Mancia G, Fagard R, Narkiewicz K, et al 2013 ESH/ESC Guidelines for the management of arterial hypertension: the Task Force for the management of arterial hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). .J Hypertens, 2013;31:1281-357.
- Burnier M, Pruijm M, Wuerzner G, Santschi V Drug adherence in chronic kidney diseases and dialysis. Nephrol Dial Transplant, 2015;30:39-44.
- David VG, Yadav B, Jeyaseelan L, Deborah MN, et al Prospective blood pressure measurement in renal transplant recipients. Indian J Nephrol, 2014;24:154-160.
- Ahmed J, Ozorio V, Farrant M, Van Der Merwe W Ambulatory vs Office Blood Pressure Monitoring in Renal Transplant Recipients. J Clin Hypertens (Greenwich), 2014; doi: 10.1111/jch.12448.
- Campistol JM, Romero R, Paul J, Gutiérrez-Dalmau A Epidemiology of arterial hypertension in renal transplant patients: changes over the last decade. Nephrol Dial Transplant, 2004;19(suppl 3):iii62-iii66.
- 21. Castillo-Lugo JA, Vergne-Marini P Hypertension in kidney transplantation. Semin Nephrol, 2005;25:252-260.
- Barbari A. Posttransplant hypertension: multipathogenic disease process. Exp Clin Transplant, 2013;11:99-108.