THE PARTICULARITIES OF ABPM PARAMETERS IN HYPERTENSIVE PATIENTS WITH NON-DIALYSIS CKD

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REZUMAT

Chronic kidney disease (CKD) affects approximately two million people (in a population of 20 million) in Romania. Hypertension is often associated with CKD and both (hypertension and CKD) are risk factors for cardiovascular (CV) events. Ambulatory blood pressure monitoring (ABPM) is increasingly used all around the world for the diagnosis and monitoring of BP (blood pressure) because it is proven that the ABPM is superior to office BP measurements in evaluating patients with hypertension, with or without CKD. Reduced nocturnal BP fall (non-dipping or reverse-dipping patterns) is associated with target organ damage, especially kidney disease and the proportion of non-dippers and reverse-dippers patients increases progressively with the reduction of glomerular filtration rate (GFR). Another ABPM parameter, ambulatory arterial stiffness index (AASI), is an index which was recently proposed for the evaluation of arterial stiffness (a better tool than PP). It has prognostic value for cardiac death and stroke and several studies have showed that is negatively related to eGFR and is positively related to albuminuria. Hyperbaric area index (HBI) might be considered a novel sensitive marker [independent of patterns of NBPC (nocturnal BP change)] for the reduction of kidney function. These facts suggest that ABPM offers multiple useful data with impact, not only in future CV and renal outcomes assessment, but also in the treatment and management of hypertensive patients with CKD.

Keywords: hypertension, ambulatory blood pressure monitoring, chronic kidney disease, cardiovascular risk, ambulatory arterial stiffness index, hyperbaric area index, pulse pressure

INTRODUCTION

It is known that kidney disease is the 9th leading cause of death in the United States (1). The prevalence of CKD in Romania is 9-12%, similar to other European countries (2). Kidney Disease Improving Global Outcomes (KDIGO) has defined CKD as abnormalities of kidney structure or function, present for >3 months with major implications for health. The abnormalities include one or more markers of kidney damage (eg, albuminuria >30 mg/g of creatinine, urine electrolyte or sediment abnormalities and other abnormalities due to tubular disorders, abnormalities detected by histology, history of kidney transplantation, or structural abnormalities detected by imaging) or a glomerular filtration rate (GFR) <60 mL/min/1.73 m^2 (GFR categories 3a to 5) (3).

Approximately 80-85% of patients with CKD have hypertension and the proportion increases further as the GFR falls (4). Cardiovascular disease is the leading cause of death in patients with CKD (5).

The CKD patients are at increased risk of cardiovascular events. On the other hand, hypertension is often associated with CKD, – it either causes CKD, is a result of CKD, or is associated with CKD without evidence of a causal relationship. ABPM

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has become indispensable for the diagnosis and monitoring of hypertension in renal patients, because many studies have proven the superiority of ABPM to random office BP measurements, in patients with hypertension with and without CKD (6). Although ABPM is increasingly used all around the world for the diagnosis and monitoring of BP, in Romania it is only in its infancy.

This article aims to highlight the particularities of ABPM parameters in hypertensive patients with CKD.

Both hypertension and low GFR, are risk factors for CV events. Hypertension is considered the most important factor for CKD progression. For the diagnosis and monitoring of hypertension both office and home measurement may be used. ABPM requires the employment of a digital device and has become a powerful tool for the appraisal of hypertension since the late 80s. However, ABPM is not yet standardized, nor is its equivalence to office BP measurements established, as most validated studies on CV disease and renal disease have used office BP assessment.

ABPM parameters and CKD

Compared to office blood pressure (BP), the ABPM can offer more useful parameters for the management of CKD and for CV risk assessment: morning BP; BP variability; ambulatory arterial stiffness index (AASI); nocturnal BP drop (diurnal index); pulse pressure, systolic/diastolic/mean BP, minimum/maximum; hyperbaric index (HBI).

Several recent international hypertension management guidelines (7-9) grant increasing trust to methods of measuring BP outside the medical environment, self-measurements at home (HBP), 24-hour ambulatory BP (ABP) measurements, or both. Indeed, mean BP over 24 hours is significantly better associated with morbidity and mortality (with respect to cerebrovascular or coronary events) than BP obtained in the office (10-12).

Normal values for ABPM are a daytime average of less than 135/85 mmHg and a night-time average of less than 120/70 mmHg, but even lower thresholds are advocated, particularly in high-risk groups, such as diabetic patients (13).

The existence of „white-coat" normo-tension is well-known. The ABPM has identified patients in whom out-of-office BP is clearly much higher than office BP, but the prevalence of this phenomenon is less well-known. Therefore, certain patients remain under-treated and are at higher risk for adverse CV outcomes. Elevated BP at night may be observed in some patients, also (14).

Multiple studies on CKD patients have demonstrated that ABPM and home BP measurement are better correlated with end-organ damage than office BP measurements. This is true for markers of kidney damage, particularily for proteinuria: a cross-sectional study of 232 CKD patients pointed out that ABPM is more strongly associated with proteinuria than office BP (15).

Nighttime BP and CKD

The only non-invasive technique which allows BP to be monitored during sleep is ABPM. There is increasing evidence that nighttime BP assessment can provide important information: diminished decrease of BP during night is associated with target organ damage, especially with CKD, and this may be a useful, albeit nonspecific, clue for secondary hypertension (renal hypertension). In addition, The Ohasama study demonstrated that the lack of nocturnal decline in BP is a risk factor for CV death (16).

Patients whose BP decreases at night are sometimes called "dippers" and those whose BP does not drop are also known as "non-dippers" (17).

The circadian BP profile is different in patients with CKD than in other patients, being marked by a preponderance of non-dippers and reverse-dippers (which is true not only for patients with renal hypertension, but also for those without hypertension). The lower the eGFR, higher this preponderance among renal patients. Several studies have shown that ABPM is more advantageous than office BP in evaluating the risk for both CV events and for CKD progression to ESRD and death. It seems that collecting data by ABPM can predict subsequent renal evolution and occurrence of CV events in patients with CKD (18).

There is an association between elevated nighttime BP, left ventricular (LV) hypertrophy, albuminuria and CKD (19) The riser BP pattern, or reverse-dipper, means that nocturnal BP exceeds daytime BP. A cross-sectional study that involved 10271 hypertensive patients found a large difference between the patients with and without CKD in the prevalence of the riser BP pattern 17.6% vs. 7.1%, which means that CKD is associated with a 2.5 times higher prevalence of the BP pattern associated with the highest CVD risk. Moreover, among patients with CKD, the lower the GFR, the higher the prevalence of this high-risk BP pattern: 8.1% in stage 1 CKD vs. 34.9% in stage 5 CKD (20).

Because of higher non-dipper BP profile in CKD patients, researchers tried to apply this find-

ing in treatment management. Many trials have reported that adjusting the administration time for the blood pressure lowering medication significantly reduces CV events, bedtime administration being preferred to early morning (21). It is recomended to use ABPM for diagnosis and management of hypertension in all patients with CKD (22).

Pulse pressure

The pulse pressure (PP) is deffined as the difference between the systolic and the diastolic pressures. This value seems to be superior in predictive value to the systolic or diastolic values taken separately.

The higher PP increases the stress on arteries. This stress produces fatigue and an increased rupture rate in the elastic components of the arterial wall, increasing the risk of atherosclerosis and thrombosis (23).

In the Framingham Heart Study it was observed that an increment of 10 mmHg in the PP was associated with a 23% increase in the risk of developing coronary heart disease (CHD) (24). This clear association of CHD with PP was visible in patients over the age of 50 years, and most evident in patients over the age of 60 years (25) and the adverse outcomes's association with higher PP observed in patients over the age of 50 years, appear to apply to hypertensive, as well as to normotensive patients, including those with normal systolic but low diastolic blood pressure (26).

Several researchers studied the relationship between PP and decreased GFR. It was observed that PP is an independent risk factor for progression of CKD (20,27-30,). In a post-hoc analysis of the RENAAL trial, 1513 patients with diabetic nephropathy were studied for the effects of BP level on the progression of diabetic nephropathy (29). After controlling for multiple potential confounders, a 17% higher relative risk of developing ESRD was associated with a 10 mmHg increment in PP. A similar association was not found for diastolic pressure.In a study on 329 patients with CKD it was observed that a 10 mmHg higher PP was significantly associated (after approximately 6 months of follow-up) with a 10% increase in relative risk of kidney function decline. This study observed that PP is a better predictor for progression of CKD (even for mild to moderate reductions in GFR) than DBP or even SBP (28).In a cross-sectional cohort of 2144 patients with CKD from the Chronic Renal Insufficiency Cohort (CRIC) study, Weir et al. (2011) found that PP, a correlate of the pulsatile hemodynamic load and conduit vessel stiffness, as well as an important CV risk factor, is also independently associated with urine protein excretion (31,32). This association may explain why microalbuminuria predicts cardiovascular events in nondiabetic subjects. PP is not only a simple marker for atherosclerotic disease, but the independence from concomitant vascular disease of its association with proteinuria suggests that PP influences albuminuria directly (33).

The ambulatory arterial stiffness index

The AASI, derived from ABPM, is an index which was recently proposed for the evaluation of arterial stiffness (34).

On repeated measurements, the values of SBP and DBP follow a similar pattern, being linearly correlated to one another. However, for subjects with arterial stiffness, a given increase in DBP is associated with a greater increases in SBP, as a consequence of declining arterial compliance. This relationship can be assessed by scatter-plotting DBP against SBP (i.e. individual measurements obtained by means of ABPM) and calculating the linear regression slope of DBP on SBP, which is considered an overall measure of arterial capacitance while its complement (1 minus the regression slope) is used to assess arterial stiffness. The stiffer the arteries the closer is AASI to 1 (35). It is known that aging is an important determinant for arterial stiffness, therefore it is probable to exist a relationship between age and AASI.

The fact that AASI is an indirect arterial stiffeness marker is supported by one study published by Li et al. They found that AASI closely correlated not only with the peripheral and central systolic augmentation indices, but also with aortic pulse wave velocity (36).

In a cohort of 11,291 patients, Dolan et al. found that AASI has prognostic value for cardiac death and stroke (37).

One study on european subjects found that elevated AASI is a stronger predictor of stroke, beyond traditional CV risk factors, including PP and the mean arterial pressure (38). Presently, a great number of specialists propose that the software for ABPM analysis should include in the near future AASI as a marker in cardiovascular risk assessment.

Several studies have showed a correlation between AASI and renal function among hypertensive patients: hypertensive patients with CKD have a higher AASI than hypertensive patients without

CKD (39,40). Ratto et al. demonstrated that AASI is negatively related to eGFR and positively related to albuminuria (41).

CKD patients have stiffer arterial tree when compared to normal-creatinine-patients of similar age and BP (42,43). Beyond hypertension and dyslipidemia, uremia itself plays a role in the stiffening process, maybe by mineral metabolism alterations and arterial calcification (44).

Hyperbaric area index

Hyperbaric area is defined as the area encircled by the line of ABPM and two boundary lines of hypertension limits: 135/85 mmHg (during the day) and 120/70 mmHg (during the night). The HBI is described as 24-h adjusted hyperbaric area, an index of BP load (obtained by ABPM) on different organs. Since diastolic HBI is more affected by arteriosclerosis, it is not used frequently.

The CKD-JAC study, with a cohort of 2,977 japanese patients, founds that HBI might be considered a novel sensitive marker [independent of patterns of NBPC (nocturnal BP blood pressure change)] for the reduction of kidney function (45).

Blood pressure variability

Daily BP variability (BPV), obtained by a ABPM device, is considered more and more as an important risk factor for CV events occurence (with prognostic value for CV mobidity and mortality) and end-organ damage. It was shown that an enhanced variability in nocturnal SBP in initially untreated hypertensive subjects is an independent predictor of cardiac events.

In the past the CV consequences of hypertension were considered to be primarily determined by the average BP values. Nowadays however, multiple trials and observational studies have highlighted a possible important role of enhanced BPV in this regard. Measures of BPV can be obtained through different methods. In the Jackson heart study on African Americans, CKD was associated with higher 24-h BPV (using 24-h ABPM); however, after adjustment for mean 24-h BP, this association was no longer present. Several studies focusing on untreated essential hypertensive patients have shown that an increased short-term BPV is inversely correlated with GFR and is directly correlated with urinary albumin excretion.

CONCLUSIONS

The ABPM can help evaluate the risk of CKD progression to ESRD and death when added to office BP monitoring. The ABPM is mandatory for the adequate diagnosis and CVD risk assessment in hypertensive patients with CKD and may bring a contribution to establish the proper therapeutic scheme in order to diminish the long term CVD risk, although presently most treatment decisions use clinic BP values obtained by standard measurement methods.

In conclusion, it is strongly recomended to use ABPM for diagnosis and management of hypertension in all patients with CKD.

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