ARTERIAL HYPERTENSION IN CHRONIC KIDNEY DISEASE

Emazarova Mokhitabon Mamatkulovna
Assistant of the Department of Nephrology and Hemodialysis,
Department of internal medicine
1 course (PhD) student of Doctoral studies at TashPMI
https://doi.org/10.5281/zenodo.7569681

Abstract. *Chronic kidney disease (CKD) is becoming an increasingly common disease worldwide, and is closely associated with cardiovascular disease (CVD). Arterial hypertension is both a cause and a consequence of CKD and affects the vast majority of patients with CKD. Control of arterial hypertension is important in patients with CKD because it slows the progression of the disease as well as reduces the risk of cardiovascular disease. Non-pharmacological interventions are useful in lowering BP in CKD, but they are rarely sufficient to adequately control BP. Patients with CKD and arterial hypertension are often required to have a combination of antihypertensive drugs to achieve BP goals. Certain pharmacological treatments provide additional renoprotective and/or cardioprotective effects independent of BP, and this should be taken into account when prescribing therapy.*

Keywords: *chronic kidney disease, drug treatment, cardiovascular disease, control of arterial hypertension, eGFR.*

Introduction

Chronic kidney disease (CKD) affects 10-15% of the population worldwide and its prevalence is increasing. CKD is defined as the presence of reduced kidney function, or kidney damage (often indicated by the presence of proteinuria) for ≥ 3 months.

Arterial hypertension is both a cause and a consequence of CKD and contributes to its progression. Arterial hypertension and CKD are independent risk factors for cardiovascular disease (CVD). When both exist together, the risks of morbidity and mortality from CVD increase substantially. It is important to note that from a therapeutic point of view, lowering BP can slow down the decline, thereby preventing progression to end-stage renal disease and reducing the incidence of cardiovascular disease in this group of patients..

The pathogenesis of arterial hypertension (CKD)

A number of mechanisms contribute to the development of arterial hypertension in CKD. An increase in sympathetic tone caused by afferent signals generated by functionally dying kidneys contributes to the development of arterial hypertension in CKD. As eGFR decreases, the renin-angiotensin-aldosterone system (RAAS) is activated, which promotes salt and water retention. This is aggravated by hypersensitivity of BP to salt. Endothelial dysfunction is characteristic of late stages of CKD and its association with arterial hypertension is well known. Increased arterial stiffness is also observed in all forms of CKD, it is associated with the development of arterial hypertension, and is an independent risk factor for cardiovascular events. Once hypertension has developed, several factors, including increased oxidative metabolism followed by relative renal hypoxia, may contribute to the further progression of BP and CKD. Normally, BP shows a nocturnal drop of ~10 to 20%. This is controlled by several factors, including diurnal fluctuations in autonomic function, salt excretion, and the RAAS. Dysregulation of these systems in CKD leads to an increase

in nocturnal BP, which leads to increased morbidity and mortality from CVD and the risk of CKD progression.

Treatment of BP in CKD

The REIN-2 study (Efficacy of Ramipril in Nephropathy-2) examined whether intensive BP control with the addition of a dihydropyridine calcium channel antagonist (blocker) (CCB) to an established angiotensin-converting enzyme (ACE) inhibitor was superior to standard BP control with an ACE inhibitor alone. The study included patients with CKD and initial proteinuria > 1 g/day. The addition of CCB did indeed lower BP; however, this did not lead to an improvement in renoprotection. Recommendations published after these landmark studies reflected these results by proposing lower targets only for patients with significant proteinuria. However, these studies did not take into account the potential benefits of intensive BP control in relation to cardiovascular endpoints.

Conclusion

Lowering BP in CKD slows the progression of the disease and reduces the incidence of cardiovascular disease. Understanding the pathophysiological mechanisms leading to the development of arterial hypertension in this group of patients is useful for effectively addressing both endpoints. Existing recommendations do not offer solutions for achieving optimal BP targets. Pharmacological therapies designed to achieve these goals offer varying degrees of risk reduction depending on patient characteristics. Thus, one drug does not fit all, and understanding what really lowers BP in CKD is key to making informed, individualized decisions.

REFERENCES

1. Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, et al. Prevalence of chronic kidney disease in the United States. *JAMA.* 2007;298:2038–2047.
2. Mills KT, Xu Y, Zhang W, Bundy JD, Chen CS, Kelly TN, et al. A systematic analysis of worldwide population-based data on the global burden of chronic kidney disease in 2010. *Kidney Int.* 2015;88:950–957.
3. Baekken M, Os I, Sandvik L, Oektedalen O. Microalbuminuria associated with indicators of inflammatory activity in an HIV-positive population. *Nephrol Dial Transplant.* 2008;23:3130–3137