CE

Treatment of Resistant Hypertension in the Patient With Chronic Kidney Disease

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ABSTRACT

The burden of risk and the impact of disease associated with poorly controlled hypertension in the United States population presents significant challenges to primary care providers, including nurse practitioners. The lack of blood pressure control leads to a staggering number of premature deaths annually. To reduce risk for major cardiovascular events, aggressive approaches to treatment of resistant hypertension must be considered. Hypertension plays a key role in the progression of chronic kidney disease. The goal of treating resistant hypertension must be directed at reducing cardiovascular risk while preserving renal function.

Keywords: chronic kidney disease, clinical inertia, hypertension, resistant hypertension

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Treatment of resistant hypertension (RHTN) in a patient with chronic kidney disease (CKD) presents nurse practitioners (NPs) with unique challenges as they attempt to effectively treat RHTN while preserving kidney function. Notwithstanding appropriate treatment, the "uncontrollable" part of hypertension, RHTN, is a significant threat, because long-standing high levels of blood pressure along with concomitant debilitating entities, such as CKD, create a prominent high-cardiovascular-risk milieu. Effective individualized treatment for RHTN requires NPs to recognize that RHTN and CKD are entangled in a complex and vicious interaction. The purpose of this study is to

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At the conclusion of this activity, the participant will be able to:

A. Identify the evidence-based medical treatments for resistant HBP
B. Describe the lifestyle modifications effective in managing resistant HBP in patents with CKD
C. Explain diagnostic studies for several causes of secondary HBP

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enhance and expand NPs' understanding of RHTN with CKD through discussion of evidence-based best practice. The discussion includes a review of pathophysiologic mechanisms related to RHTN associated with CKD as well as both pharmacologic and nonpharmacologic treatment. A real-life case study is presented to demonstrate the use of treatment principles.

PROBLEM IDENTIFICATION

Essential hypertension (HTN) is the most prevalent controllable disease in developed countries, affecting > 25% of the adult population. The global disease burden is immense, with 62% of strokes, 49% of heart disease, and 7.5 million deaths per year attributed to HTN, which is a major modifiable risk factor affecting nearly 1 in 3 adults in the United States. Thus, HTN, as the foremost risk factor for coronary artery disease and stroke, is responsible for the majority of cardiovascular morbidity and mortality in the US. Furthermore, HTN is also the leading cause of congestive heart failure, either directly causing hypertensive heart disease or indirectly contributing to ischemic cardiomyopathy.[1] Lack of blood pressure control presents as RHTN, a common clinic problem encountered by NPs, primary care clinicians, and specialists. Although the exact prevalence of RHTN is unknown, clinical trials suggested that it is not rare and involves perhaps 20%-30% of patients with HTN.[2] RHTN has been defined as blood pressure that remains above goal despite concurrent use of 3 antihypertensive agents of different classes, ideally including a diuretic, and all at optimal doses.[1] An increased interest in the independent role of RHTN has evolved from the common observation that many patients with essential HTN fail to meet their treatment goal despite polytherapy.

Recent studies have struggled to approximate the prevalence of RHTN, which may occur in up to 30% of all patients with HTN.[3] Other studies focused on documenting the strength of an association with chronic diseases such as CKD. In some cases, CKD was found to be almost 4 times more common in patients with RHTN.[1] It is of paramount importance that NPs recognize that the increased prevalence of kidney failure associated with high costs and poor outcomes of treatment constitute a worldwide public health threat.[4] Aggressive treatment of RHTN by providers can lead to prevention of renal disease and decrease risk of cardiovascular disease.

Numerous risk factors contribute to the development of hypertension. Figure 1 includes a summary of the social determinants as well as the main behavioral and metabolic risk factors that may contribute to the development of HTN and complications.

OVERVIEW OF RESISTANT HTN IN CKD

RHTN is both a cause and a consequence of CKD. Failure to effectively treat RHTN increases the risk of important adverse outcomes, including kidney failure, early development and accelerated progression of cardiovascular disease, and premature death.[5]

To achieve effective treatment, the NP utilizes an understanding of pathophysiology and the importance of blood pressure control in patients with RHTN and CKD.

Pathophysiology

RHTN plays a key role in progression of CKD.[5] A strong association exists between the 2 conditions, with a complex reciprocal interaction.[1] Renal dysfunction causes increased sodium and water retention. Patients with water retention may develop fluid overload, which in turn makes blood pressure control difficult. Persistently elevated blood pressure causes nephrosclerosis, leading to further kidney damage and perpetuation of the cycle. Adequate blood pressure control slows the progression of glomerular filtration rate (GFR) decline in patients with proteinuria and serves to preserve current renal function.[5]

The sympathetic nervous system is known to be a major contributor to the pathophysiology of RHTN. Increased renal sympathetic activity leads to a cascade of actions including: (a) decreased renal blood flow and decreased glomerular filtration rate by renal vasoconstriction; and (b), through stimulation of the release of renin by the juxtaglomerular cells, angiotensin II is produced. The cascade of action is further amplified by direct activation of renin resulting from kidney injury secondary to poorly controlled RHTN. Increases in renal sympathetic nerve activity also directly increase renal tubular sodium reabsorption.[6] The complex cycle that includes activation of