Kidney diseases caused by hypertension.

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Abstract

Uncontrolled high blood pressure over time can lead to the arteries surrounding the kidneys becoming more constricted, frail, or rigid. The renal tissue cannot receive adequate blood from these damaged arteries. Kidney arteries with damage do not filter blood well. Your blood is filtered by tiny, finger-like kidney structures called nephrons. Damage to the blood arteries in the kidneys may cause them to stop functioning properly. When this occurs, the kidneys are unable to completely cleanse your body of wastes and extra fluid. Extra fluid in the blood arteries can create a deadly cycle that raises blood pressure even higher and damages the kidneys more.

Keywords: Nephrology, Hypertension, Kidney disease.

Introduction

People with diabetes are far more likely to have hypertension, and having diabetes increases the chance of developing cardiovascular disease. The pathophysiological relationships between diabetes and hypertension are the focus of this review. Diabetes causes an increase of sodium transporters in the kidneys, which results in different salt processing in the kidneys. Diabetes may cause angiotensin II to act directly on the renin-angiotensin-aldosterone system, increasing blood pressure, as well as indirectly by increasing sympathetic activity. Blocking the renin-angiotensin-aldosterone pathway is a common treatment for hypertension, and data indicates that it may also lower the prevalence of diabetes. Diabetes patients typically have autonomic dysfunction, which may raise sympathetic tone and stimulate the generation of renin in the juxtaglomerular apparatus, resulting in hypertension. Additionally, the circadian blood pressure pattern is usually irregular in persons with diabetes. The onset and progression of diabetic kidney disease, whose pathophysiology is mediated by a number of routes including endothelial dysfunction and advanced glycation end products, is another significant association between hypertension and diabetes. Last but not least, obesity and the metabolic syndrome may also play a role in the aetiology of hypertension and diabetes through their impact on a number of hormones and inflammation [1].

Globally, hypertension is a major contributor to the risk of cardiovascular disease and all-cause mortality. Chronic kidney disease and hypertension are two illnesses that are intimately related to one another because chronic kidney disease can aggravate hypertension and cause renal function to decline. A reduced number of functional nephrons, sodium retention and volume expansion, upregulation of the sympathetic nervous system, hormonal factors like upregulation of the renin-angiotensin-aldosterone system, and endothelial dysfunction are just a few of the factors that interact to cause hypertension in the context of chronic kidney disease. The progression to end-stage renal disease can be accelerated by poorly managed hypertension. This review examines the sympathetic nervous system, the renin-angiotensin-aldosterone system, and the function of sodium as pathophysiological factors that contribute to hypertension. The link between hypertension and renovascular illness in the context of chronic renal disease is briefly discussed as a potential cause and target for therapeutic intervention. The benefits of long-term ideal blood pressure control on the cardiovascular system are also emphasised, along with treatment methods and goals [2].

Due to its high frequency, correlations with cardiovascular illness, and progression of chronic kidney disease, managing arterial hypertension in patients with chronic renal disease continues to be a significant problem. However, the effects of blood pressure lowering in terms of renal protection or harm remain debatable. Several clinical trials and meta-analyses have shown that aggressive treatment of hypertension in patients with and without chronic kidney disease lowers the risk of chronic kidney disease and all-cause mortality. Patients with chronic renal disease have atypical BP patterns outside of the doctor's office, according to both home and ambulatory BP measurement, and more research is needed. so that treatment plans can be modified based on how ambulatory versus office BP measures relate to certain outcomes [3,4].

Despite the fact that renin-angiotensin system blockade seems to be helpful in patients with advanced chronic kidney disease, particularly in the presence of proteinuria, discontinuing renin-angiotensin system inhibition should be taken into

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consideration in cases of frequent episodes of acute kidney injury or hypotension while awaiting the results of ongoing trials. Future research must clarify the indications and advantages of renal denervation's use in people with chronic kidney disease in light of the new evidence supporting its use in arterial hypertension. In addition, new participants in the pathophysiology of arterial hypertension and CKD, such as microRNAs and the gut microbiota, have clinical applications [5].

Conclusion

The cornerstone of treatment for CKD patients is still strict blood pressure management. The CKD patient with hypertension typically needs a combination of medications to reach their target blood pressure. The numerous antihypertensive medications' varying pharmacokinetics and pharmacodynamics should be taken into consideration when treating these patients. By doing this, the likelihood of achieving objective BP will increase while the possibility of negative effects will be reduced.

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