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The Renin–angiotensin System and its Role in Cardiovascular–kidney–metabolic syndrome

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Introduction

The Renin-Angiotensin System (RAS) plays a pivotal role in maintaining physiological homeostasis by regulating blood pressure, electrolyte balance, and vascular tone. It comprises a cascade of enzymatic reactions that ultimately lead to the production of angiotensin II (Ang II), a potent vasoconstrictor and key mediator of RAS effects. While the RAS is crucial for normal physiological function, dysregulation of this system is associated with the development and progression of cardiovascular diseases, kidney disorders, and metabolic syndromes [1].

Description

Cardiovascular-kidney-metabolic syndrome represents a cluster of interrelated disorders characterized by hypertension, insulin resistance, dyslipidemia, obesity, and renal dysfunction. The underlying pathophysiology involves intricate interactions between cardiovascular, renal, and metabolic pathways, with the RAS emerging as a central player in this complex syndrome. The dysregulation of the RAS contributes significantly to the pathogenesis of cardiovascular-kidney-metabolic syndrome through several mechanisms: Ang II exerts potent vasoconstrictive effects, leading to increased peripheral resistance and elevated blood pressure. Moreover, Ang II promotes sodium and water retention, further exacerbating hypertension [2].

Insulin Resistance: Ang II interferes with insulin signaling pathways, impairing glucose uptake and promoting insulin resistance. This dysregulation contributes to the development of type 2 diabetes mellitus, a common component of the metabolic syndrome. Ang II influences lipid metabolism by promoting the expression of pro-inflammatory cytokines and inhibiting lipoprotein lipase activity, leading to dyslipidemia characterized by elevated levels of triglycerides and Low-Density Lipoproteins (LDL). Ang II induces adipocyte hypertrophy and promotes the differentiation of preadipocytes into mature adipocytes, contributing to the expansion of adipose tissue mass observed in obesity. Chronic activation of the RAS leads to renal structural alterations, glomerular hyperfiltration, and proteinuria, contributing to the progression of Chronic Kidney Disease (CKD) in patients with cardiovascular-kidney-metabolic syndrome [3].

Given the central role of the RAS in the pathogenesis of cardiovascular-kidney-metabolic syndrome, targeting this system has emerged as a promising therapeutic strategy. Pharmacological interventions aimed at modulating RAS activity include: Angiotensin-Converting Enzyme (ACE) Inhibitors: ACE inhibitors block the conversion of angiotensin I to Ang II, thereby attenuating vasoconstriction and reducing blood pressure. Additionally, ACE inhibitors exert renoprotective effects by dilating efferent arterioles and reducing intraglomerular pressure [4].

ARBs selectively antagonize the binding of Ang II to its receptors, thus preventing its downstream effects on vascular tone, sodium retention, and aldosterone release. ARBs are particularly beneficial in patients intolerant to ACE inhibitors due to cough or angioedema. Direct Renin Inhibitors (DRIs): DRIs inhibit the enzymatic activity of renin, the rate-limiting step in the RAS cascade, thereby reducing the production of Ang II. By targeting renin directly, DRIs offer an alternative approach for RAS blockade. Mineralocorticoid Receptor Antagonists (MRAs): MRAs block the action of aldosterone, a key hormone involved in sodium retention and potassium excretion. By inhibiting aldosterone effects, MRAs mitigate fluid retention and reduce blood pressure [5].

Conclusion

The Renin-Angiotensin System plays a pivotal role in the pathogenesis of cardiovascular-kidney-metabolic syndrome, contributing to hypertension, insulin resistance, dyslipidemia, obesity, and renal dysfunction. Targeting the RAS with pharmacological interventions such as ACE inhibitors, ARBs, DRIs, and MRAs offers promising therapeutic options for managing this complex syndrome. Further research is warranted to elucidate the underlying mechanisms and optimize treatment strategies to improve clinical outcomes in patients with cardiovascular-kidney-metabolic syndrome.

Acknowledgement

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Conflict of Interest

None.

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