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Editorial

Renin: Angiotensin System in Chronic Heart Failure

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INTRODUCTION

Renin-Angiotensin System for Chronic Heart Failure The value of multiple randomized clinical trials confirmed in patients with acute myocardial infarction (AMI). Type 1 Angiotensin II Receptor Blockers (ARB) provide an alternative method of blocking the renin-angiotensin system (RAS). ARB may be more effective or tolerated than ACE inhibitors (or both). In short, angiotensin II is produced by non-ACE enzymes, which means that ACE inhibitors may be less effective at blocking this peptide than ARB (Figure 1). Tentensin receptor type 1 (AT1) also allows more angiotensin II to be used to stimulate the unhindered AT2 receptor (and possibly another AT receptor pes) which has purportedly beneficial effects. Reduce the progression of cardiovascular disease.

Unlike ARB, ACE (kinase II) inhibitors inhibit the degradation of bradykinin. Increased bradykinin may have effects including enhancing vasodilation, fibrinolysis, and inhibiting cell growth and division, which may contribute to the benefits of ACE inhibitors. On the contrary, the accumulation of bradykinin can cause some adverse effects of ACE. Inhibitors, namely cough, rash, and angioedema. Therefore, due to the undisputed role of ACE inhibitors in CHF and AMI, it is challenging to evaluate the effects of ARB on CHF and AMI, which raises questions about trial design and dose selection. A special issue is the need for direct comparison, including formal "non-inferiority" testing, which has an impact on patient selection, the choice and dosage of RCT inhibitors, and samples.

The 2 main methods used include direct comparison of 2 treatment types (1 trial for CHF and 2 trials for AMI) or the strategy of adding ARB or placebo to ACE inhibitors (2 trials 1) in CHF and AMI. The pharmacological concepts behind these two methods are also more complicated than at first glance. These alternative approaches view the role of bradykinin in a contradictory way. Due to the lack of bradykinin-mediated adverse reactions, the head-to-head comparison method relies on the better tolerability of ARB and the potential greater ability of ARB to block RAS more completely. In contrast, the complementary strategy assumes that the potential clinical benefits of bradykinin outweigh any adverse effects it may cause, and these may add up to block RAS more completely with ARB. The additional methods also lead to different pharmacological effects compared to when ARB is used alone. 7 combination therapy, negative feedback mediates the increase of angiotensin II, which usually occurs together with ARB (and can activate other AT receptors), that is, the combination therapy of ACE reduction inhibitor

Intravenous treatment for unadmitted heart failure for ≥ 4 hours or cardiac arrest after resuscitation) by 13.2%. This is mainly due to the 27.5% reduction in the risk of CHF hospitalization. However, the analysis of the two subgroups distracted attention from this overall positive effect. One person believes that most of the benefits are concentrated in a small number (7%) of patients who have not taken ACEI. 24 Another believes that after adding valsartan, the condition of patients who initially took both ACE inhibitors and beta blockers (the best treatment) worsened. Therefore, due to this obvious risk, the Food and Drug Administration (FDA) and international guidelines recommend avoiding "triple therapy" (a combination of ACE inhibitors, β-blockers, and ARB). However, the FDA approved valsartan for patients who are intolerant to ACE inhibitors27.

CONCLUSION

The renin angiotensin system (RAS) or renin angiotensin aldosterone system (RAAS) is a hormone system that regulates blood pressure, fluid and electrolyte balance, and systemic vascular resistance.

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None

CONFLICT OF INTEREST

The authors declare that they have no conflict of interests.

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