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Effect of Inhibition of the Renin Angiotensin System on Cardiovascular Disease

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DESCRIPTION

The updated guidelines emphasize targeted reduction of overall cardiovascular risk. Hypercholesterolemia and hypertension have synergistic adverse effects on insulin resistance and endothelial dysfunction. Statins are of paramount importance in patients with hypercholesterolemia to prevent cardiovascular disease by lowering low-density lipoprotein cholesterol, ameliorating endothelial dysfunction, and providing other anti-atherosclerotic effects. Unfortunately, statin therapy induces insulin resistance in a dose-dependent manner and increases the risk of type two diabetes. In addition to lowering blood pressure, inhibitors of the renin-angiotensin system ameliorate both endothelial dysfunction and insulin resistance. Furthermore, hypercholesterolemia interacts with the renin-angiotensin system at several stages of insulin resistance and endothelial dysfunction. In this regard, combination therapy with a statin and an inhibitor of the renin-angiotensin system, in addition to lowering both cholesterol levels and blood pressure and reducing cardiovascular events, compared to monotherapy in both patients, show additive or synergistic beneficial effects on endothelial dysfunction and insulin resistance. This is mediated through both separate and interconnected mechanisms. The combination of statins and inhibitors of the renin-angiotensin system in developing optimal therapeutic strategies for the prevention or treatment of cardiovascular disease in patients with hypertension, hypercholesterolemia, diabetes, metabolic syndrome, obesity may be important.

Statins effectively reduce morbidity and mortality from cardiovascular disease. However, even after reaching targets for low-density lipoprotein cholesterol, the risk of cardiovascular disease remains. Combinations of statins and agents that affect the renin-angiotensin system have been studied to reduce this risk. Greek Atorvastatin and Coronary Heart Disease Evaluation, Japanese Coronary Artery Disease, Anglo-Scandinavian Cardiac Outcomes Trial, Evaluation of Treatment Efficacy in Metabolic Syndrome without Perceivable Diabetes It has been suggested to reduce the incidence of disease. Less than statins alone and significantly less than inhibition of the renin-angiotensin system alone. These benefits appear to be related to endothelial function, vascular inflammation, and effects on atherosclerotic plaque initiation, progression, and rupture. These effects are driven, at least in part, by micrornas, mediators involved in the pathogenesis and clinical manifestations of atherosclerosis (e.g., restoration of endothelial function and reduction of vascular inflammation). Some micrornas are favorably affected by statins and others are favorably affected by inhibition of the renin-angiotensin system. There is a family of mirs that are associated with destabilization of coronary artery plaque and are favorably affected by both statins and inhibition of the renin-angiotensin system. Patients at high risk for cardiovascular disease, hypertension, obesity, metabolic syndrome, and/or diabetes should be prescribed a combination of statins and her renin-angiotensin system inhibitors on a regular basis to maximize clinical benefit. The renin-angiotensin system is a key regulator of blood pressure and vascular response to injury. There is increasing evidence that inhibition of the renin-angiotensin system may provide end-organ protection independent of blood pressure reduction. The two classes of drugs directly target angiotensin through complementary mechanisms. Angiotensin-converting enzyme inhibitors block the conversion of angiotensin to the active peptide angiotensin II, increasing the availability of bradykinin. Angiotensin receptor blockers selectively antagonize angiotensin at angiotensin receptors, increase angiotensin receptor activation, and may also modulate the effects of angiotensin II breakdown products.

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CONFLICT OF INTEREST

The author's declared that they have no conflict of interest.

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