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Ubiquinol and Autonomic Dysfunction in Hypertension and Heart Failure: A Short Review

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Introduction

Coenzyme Q10 (CoQ10) is one of the most commonly used dietary and nutritional supplements and is known to be a potent antioxidant. There are two forms of CoQ10 including ubiquinone (oxidized form) and ubiquinol (reduced form) that are produced in the mitochondria [1-2]. CoQ10 is an essential component in the mitochondrial electron transport chain that plays a role in synthesis of Adenosine Triphosphate (ATP) and scavenging free radicals. A deficiency in CoQ10 can cause cellular energetic impairment and decreased antioxidant defensive mechanisms. Patients with heart failure with preserved ejection fraction (HFpEF) often have a reduction in CoQ10 and are prone to oxidative stress. In addition, HFpEF patients are often hypertensive and this can be a significant contributing factor for the development of HFpEF [3-5]. Autonomic dysfunction could be the essential link of cellular energetic impairment and hypertension associated HFpEF.

Research has shown that cellular energetic impairment (decreased ATP production) reduces sarcoplasmic reticulum Ca2+-ATPase [6]. This results in decreased actin-myosin cross-bridge detachment, which is reflected in increased diastolic stiffness and decreased ventricular compliance. Any deficiency in ATP in the heart caused either by impaired ATP synthesis or overconsumption of ATP has a significant impact on systole and diastole. The less optimal systolic contraction and diastolic relaxation results in decreased stroke volume from compromised blood filling and pumping and subsequently reduced cardiac output. Physiologically, the autonomic nervous system will compensate for the decreased cardiac output by activating the sympathetic nervous system. With the sympathetic nervous system being dominant, the heart rate is increased, and cardiac contractility is enhanced. However, long-term ATP deficiency may lead to overstimulation of the sympathetic nervous system causing receptors desensitization. The cardiac cycle is a constant active process of contraction and relaxation of cardiac myocytes during systole and diastole involving changes in pressures and volumes in each of the cardiac chambers. Therefore, the demand for ATP by cardiac myocytes is significantly higher than for other organs [6,7]. The uncoupling of actin and myosin during diastole is also an active process that consumes ATP.

Ubiquinol and HFpEF

We conducted a pilot study to examine the effects of ubiquinol on the development of HFpEF associated with hypertension using a Dahl Salt Sensitive (DSS) rat model. All ubiquinol treated rats had lower blood pressures and improved diastolic function than the control rats. Improving diastolic function and filling may secondarily modulate the autonomic tone, resulting in decreased blood pressures. We found that ubiquinol did reduce autonomic dysfunction and hypertension.8 Evidence has shown that the myocardium of HFpEF associated with diastolic dysfunction shares the same underlying structural changes as those observed in catecholamine- induced cardiomyopathies [8,9]. Under normal conditions, the sympathetic nervous system is responsible for up-and down-regulation of a variety of homeostatic mechanisms and is well known for its role in a fight-or-flight response. The sympathetic nervous system increases heart rate, myocardial contractility, and vascular resistance. Consequently, the net results of the activation of the sympathetic nervous system leads to increased cardiac output and increased peripheral vascular resistance, contributing to elevated Mean Arterial Pressure

(MAP). However, excessive activation or overstimulation of the sympathetic nervous system leads to sustained increased workload of the heart due to persistent increased heart rate and blood pressure. Over time, the heart hypertrophies and becomes stiff as a result of compensating for the increased workload and developing fibrosis. This impairs diastolic relaxation which consequently reduces ventricular filling. This phenomenon is termed diastolic dysfunction [9,10]. However, the percentage of the blood ejected from the left ventricle (ejection fraction) remains normal or near normal.

Conclusion

When the cardiac output fails to meet the metabolic demands of the organs in the body, HFpEF develops. In turn, diastolic dysfunction causes increased stimulation of the sympathetic nervous system and reduced capacity of the Nitric Oxide (NO)-dependent vascular relaxation, resulting in persistent hypertension. Thus, long- term overstimulation of the sympathetic nervous system resulting from decreased cardiac output leads to autonomic dysfunction [11]. Research has shown that cellular energetic impairment and increased Reactive Oxygen Species (ROS) associated with mitochondrial dysfunction mediate the relationship between hypertension and autonomic dysfunction. Acting as a mitochondrial electron transporter, ubiquinol (coenzyme Q10) contributes to synthesis of ATP and is a scavenger of ROS. Thus, the underlying mechanisms of energetic impairments, autonomic dysfunction, and cardiac structural and functional abnormalities in HFpEF [12] may be associated with a deficiency in the production of ubiquinol [5].

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