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High Dose Vitamin C in Patients with Coronary Microvascular Dysfunction in Heart Failure with Preserved Ejection Fraction

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Introduction

- Heart failure with a preserved ejection fraction (HFrEF) has normal ejection fraction, however, it presents with a similar clinical syndrome as heart failure with reduced ejection fraction. Despite the significant prevalence, there is no proven therapy to improve outcomes in patients with HFrEF other than managing coexisting conditions.

- The new paradigm presumes that comorbidities cause a systemic proinflammatory state resulting in coronary microvascular dysfunction (CMD).

- Vitamin C as an antioxidant may play a role in preventing the endothelial dysfunction by increasing scavenging radical species and sparing endothelial cell-derived nitric oxide to help modulate blood flow.

Hypothesis

- We hypothesize that high dose Vitamin C will improve CMD in HFrEF.

Specific Aims

1. To measure the effects on the myocardial perfusion reserve index (MPRI) of CMD in HFrEF by a stress cardiac MRI (CMR) before and after 12 weeks of vitamin C intake (Figure 1).

2. To measure the effects on peak oxygen consumption (VO2) with CMD in HFrEF before and after 12 weeks of vitamin C intake.

3. To measure biomolecular effects of vitamin C on cardiac and inflammatory biomarkers before and after 12 weeks of vitamin C intake.

Methods

- This is a single center, pilot study with blinded interpretation of test results. 15 subjects will be enrolled during the course of their routine care with their cardiologist. The study will measure the effects of high dose vitamin C on CMD by performing a stress CMR before and after 12 weeks of treatment. A Paired t-test will be used to compare the quantity of CMD for the statistical analysis.

Study Design

Future Implications

- The data collected from this study will be used for measurement of variance and power calculation in vitamin C treatment for future studies.

- Exploratory outcomes such as biomarkers would provide ideas about pathophysiology, future risk marker and therapeutic target.

Reference


