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Short Communication OMICS International

Association between Sarcopenia and Endothelial Dysfunction in Patients with Heart Failure: Time for Action

Marcelo Rodrigues dos Santos*

Georg-August-University Göttingen, Göttingen, Germany

***Corresponding author**: Dr. Marcelo Rodrigues dos Santos, Postdoctoral Georg-August-University Göttingen, Cardiology Robert-Koch-Straße 40 Göttingen, Germany, Tel: +49 01628100919; E-mail: celopersonal@hotmail.com

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Sarcopenia is well recognized as a comorbidity in elderly population. This entity was recently accepted as one of the most important factor leading to mortality in patients with heart failure (HF). In a cohort composed of patients with preserved and reduced ejection fraction, Fülster and collaborators [1] showed that 19.5% of patients with HF presented sarcopenia. In this study, sarcopenia was defined by appendicular skeletal muscle index (calculated as the lean muscle mass of both arms and legs divided by the height squared) 2 standard deviation below the mean of a healthy young reference group aged 18-40 years [1]. They also showed that those patients with sarcopenia had worse exercise capacity and muscle strength when compared with no sarcopenic patients [1].

More recently, we showed that the presence of sarcopenia in patients with HF is associated with endothelial dysfunction evaluated by venous occlusion plethysmography [2]. Sarcopenic patients showed lower baseline forearm and leg blood flow when compared with controls and those patients without sarcopenia. More interestingly, when we performed reactive hyperemia (~5 minutes of total ischemia) in the limbs, we observed lower peak flow in both forearm and leg in those patients with sarcopenia. Reactive hyperemia is a noninvasive maneuver to test endothelial function. After a period of ischemia, the increased shear stress on the vessel stimulates nitric oxide release.

We know that patients with HF present endothelial dysfunction when compared with health subjects. The new finding of this study was that sarcopenic patients showed more pronounced endothelial dysfunction when compared with controls and those patients without sarcopenia. In a logistic regression model analysis, we observed that peak flow in the leg was independently associated with the 6-min walk distance adjusted for age and others cofactors such as coronary artery disease (hazard ratio, 0.903; 95% confidence interval, 0.835-0.976; P=0.01). In other words, lower blood flow supply to the legs is a factor contributing with exercise intolerance in patients with HF and sarcopenia.

This result leads us to think about possible treatments in these patients, specially focused on skeletal muscle mass and endothelial function. Exercise training program and pharmacological treatment should be tested in these patients. Several studies have shown that exercise training ameliorates skeletal muscle vasodilation in patients with HF. Similar approach has been observed with pharmacological treatment. For instance, renin-angiotensin inhibitors showed improvement in vasodilation in patients with HF. However, none of these studies were tested in sarcopenic patients with HF. Therefore, it is time for action! We should develop randomized controlled trials to test possible treatments on endothelial function in patients with HF and sarcopenia.

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