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Handgrip strength in Heart Failure: The stethoscope of the muscle?

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Title: Handgrip strength in Heart Failure: The "stethoscope" of the muscle?**Força de preensão manual na insuficiência cardíaca: O «estetoscópio» do músculo?**

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There have been notable advances in the management of heart failure (HF) over the last decade. With prognosis-modifying therapies and cardiac implantable electronic devices, we have successfully reduced mortality and hospitalizations. However, for many of our patients, living longer does not necessarily mean living well. Exercise intolerance, fatigue, and loss of autonomy continue to dominate the daily lives of those living with HF, raising a crucial question: are we treating the heart but forgetting the muscle?

In this context, the article by Silva et al. is particularly relevant¹. By exploring the relationship between sarcopenia and quality of life (QoL) in patients with HF, the authors highlight a commonly underdiagnosed issue.

Sarcopenia, which involves the loss of skeletal muscle mass and function, is not just an inevitable consequence of aging; it is a comorbidity in HF with an estimated prevalence ranging from 20% to 45%². In the study conducted by Silva et al., which evaluated 114 patients with stable HF, 54.5% of the participants were identified with "probable sarcopenia"³.

The most significant finding of this study is not only the prevalence, but also the direct correlation with what is most important to the patient. The authors demonstrated that handgrip strength (HGS) is an independent predictor of QoL. They specifically noted that a 10 kg increase in HGS was associated with an 8.7-point rise in the Kansas City Cardiomyopathy Questionnaire (KCCQ-23), the most widely used scale for assessing QoL in HF patients. In a clinical setting where we fight for incremental gains, we cannot overlook this level of progress.

A potential critique of the study would be the exclusive use of HGS to define sarcopenia (classifying it as "probable"), without confirmation using muscle mass imaging. It is also important to acknowledge the other limitations mentioned by the authors, such as the observational nature of the study, which prevents us from unequivocally establishing whether sarcopenia worsens QoL or if the disease leads to inactivity. In addition, the study being conducted at a single-center study in a tertiary hospital may limit its generalization to primary care populations.

However, I argue that this apparent limitation of isolated strength assessment is, in fact, the greatest strength of this study's clinical message. In clinical practice, whether in a hospital Internal Medicine consultation or in a Family Physician's office, it is not feasible to perform routine DEXA or CT scans to assess muscle mass in all HF patients. HGS, assessed via a digital dynamometer, is a cheap, fast, and non-invasive tool. As Silva et al. correctly suggest, HGS correlates well with overall strength and can be a valuable prognostic marker⁴.

If we accept HGS as the “stethoscope of the muscle”, we gain a new vital sign. This technical simplicity ensures that the assessment is not limited to physicians alone. It is ideal for integration into the nursing consultation routine, allowing for more frequent monitoring⁵. For the patient, seeing the reading on the dynamometer serves as powerful biofeedback. It helps them understand the physical impact of the disease and visualize the importance of muscle work for their QoL, ultimately motivating them to stick to their exercise routine.

A reduced HGS score should trigger an alert. We do not need to wait for imaging confirmation of muscle atrophy to intervene. The diagnosis of “probable sarcopenia” can be enough to recommend a treatment approach that includes cardiac rehabilitation focused on strength training and nutritional optimization.⁶ It is important to note that strength training is still not fully accessible.

As physicians, we frequently focus on ventricular ejection fraction, natriuretic peptides, and New York Heart Association class. This study invites us to look at our patients' hands. Regular assessment of HGS can help monitor the risk of sarcopenia and improve patient management. More importantly, this simple assessment allows us to conduct dynamic risk stratification. Identifying a patient with low muscle strength means we are able to act before frailty becomes irreversible. Physical exercise prescription must shift from generic advice to a structured intervention.

The work by Silva et al. is a timely reminder that preserving muscle function is inseparable from preserving dignity and autonomy in HF. By demonstrating that strength is a pillar of QoL, the authors challenge us to integrate muscle assessment into our clinical routine.

The heart may be the engine, but the muscle is what enables the patient to move and preserve their autonomy.

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