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The Crossroads of Frailty and Heart Failure: What More Can We Learn?

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The interest in frailty in heart failure (HF) has grown considerably in the past decade. We now know that frailty, which is commonly defined as a biologic syndrome of decreased reserve and increased vulnerability to stressors,[1] is highly prevalent among adults with HF.[2] There is also evidence that frailty is a harbinger of worse clinical outcomes,[3] particularly among patients undergoing treatment for advanced HF[4,5] and that frailty in HF is associated with greater healthcare utilization.[6] Hence, the strong association between frailty and adverse outcomes in HF begs the question: What more can we learn about frailty in HF?

In this issue, Tanaka et al.[7] provided evidence that frailty is associated with all-cause mortality in a large sample of elderly patients hospitalized for HF. While the link between frailty and mortality is not surprising, they have taken this line of inquiry a step further by examining the additive value of frailty to the Meta-analysis Global Group in Chronic HF (MAGGIC) risk score. Adding the frailty score, as well as a comorbidity score and B-type natriuretic peptide levels, to the MAGGIC risk score resulted in a significant improvement in risk classification. Thus, an assessment of frailty adds significant value to a traditional toolkit used to predict mortality in HF. Importantly, it was the composite elements of the frailty *phenotype* that had incremental value in predicting mortality, not the individual elements of frailty. Based on these findings, we should consider incorporating an assessment of the frailty phenotype to discharge planning for hospitalized patients with HF. Moving forward, and as articulated previously,[8] there remain unanswered questions that relate to two overarching questions: How should we measure frailty in HF, and what mechanisms underlie frailty in HF?

The measurement of frailty has evolved from primitive assessments such as the foot of the bed assessment[9] to robust and validated approaches such as the Frailty Phenotype Criteria[1] or Frailty Index.[10] Tanaka et al. adapted the former to quantify frailty in their cohort. It is the most commonly used assessment of frailty generally and in HF, primarily used to identify those at increased risk for adverse outcomes. Developed using Cardiovascular Health Study data, the Frailty Phenotype Criteria includes shrinking, weakness, slowness, physical exhaustion, and low physical activity, which are each assessed using various tools and questionnaires.[1] Across studies, adaptations of the Frailty Phenotype Criteria have varied

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from minor (e.g. changing wording) to major (e.g. omitting criteria). As exemplified by Tanaka et al., there is concern about the overlap between frailty criteria and HF-related manifestations, particularly among patients hospitalized with HF. As such, it is often questioned how criteria such as physical activity (which usually is reduced around the time of a HF hospitalization) and shrinking (which typically is measured as weight loss) should be included in the assessment of frailty in a HF cohort. Hence, what we now need is consensus on how to measure frailty in HF that, in the ideal case, would appropriately capture all elements of the frailty phenotype and have added value for patients with HF in both hospitalized and community settings.

There is also much to learn about mechanisms of frailty in HF, which could in turn guide interventions. Tanaka et al. demonstrated that frailty is associated with worse outcomes across multiple subgroups, including across the spectrum of age, body mass index, and ejection fraction, as well as in both women and men. Put simply, we must gain greater insight into why, how, and in whom frailty manifests by dissecting contributing factors to frailty in the context of HF along with comorbidities and advancing age. We can begin by examining two factors: age and gender. First, while frailty is often recognized and diagnosed in older adults with HF, there is a high prevalence of frailty in younger adults with HF as well (i.e. accelerated biological aging). Second, it has been reported that frailty affects women more than men, which corroborates the male-female health-survival paradox.[11] Thus, it would be worthwhile to explore how frailty presents in both younger and older adults with HF, as well as in women and men, to better understand the similarities and differences among these sub-populations. Finally, we need a greater understanding of frailty in HF with preserved vs. reduced ejection fraction independently, and along with natural intersections of age and gender in HF.

The application of geriatric conditions like frailty to HF has expanded our perspective on this burdensome illness. There is, however, much to be learned at the crossroads of frailty and HF. From here, we can begin to develop prevention strategies, as well as interventions targeted to frail patients with HF, which will in turn mitigate the adverse outcomes we are witnessing in this growing population.

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