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EDITOR'S PAGE

Depression in Heart Failure

Beyond a SADHART

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This month's issue of *JACC: Heart Failure* includes a focus on comorbidities in heart failure. As a clinician and investigator, I have always been fascinated with the mind-heart interaction. Early in my career, I became particularly interested in mood disorders and mental stress and how they interact with cardiac function. Through personal experiences of friends and family members experiencing depression and my lifelong collaboration with many world-class psychiatrists and psychologists who were interested in cardiology, we were able to confirm the relationship between mood disorders, particularly depression, and heart failure.

One of the important findings, which was uncovered many years ago and has stood the test of time, is that depression, like many other conventional risk factors such as hypertension, hypercholesterolemia, and smoking, poses a 2- to 3-fold increased risk of major morbidity and mortality in cardiac patients, particularly heart failure patients (1). Although nonpharmacological strategies such as cognitive behavioral therapy are effective in reducing depressive symptoms, these strategies are labor intensive and often not practical in the broad patient population of heart failure. Pharmacologic therapy using selective serotonin reuptake inhibitors, as shown in the SADHART-CHF and MOOD-HF studies, has not borne out the results and does not show any significant improvement in clinical events, although the depressive symptoms were reduced but not greater than the placebo (2,3). Screening for depression in cardiac patients who are hospitalized is a recommendation in our guidelines because we understand the importance of depression as a cardiovascular risk factor. Indeed, we understand that patients who do not improve are at risk for major morbidity, mortality, and other complications that may require intensive therapies specifically targeted toward the depression.

In this issue of *JACC: Heart Failure*, Jiang et al. (4) present results of the OCEAN-HF trial, a trial that investigated omega-3 supplementation to improve depressive symptoms in heart failure patients. In the previously published SADHART study, low levels of eicosapentaenoic acid and docosahexaenoic acid were associated with worse outcomes and, thus, the premise for this clinical trial. Although the study was essentially a pilot study and the findings are suggestive and not definitive, omega-3 supplementation was associated with a reduction in cognitive depression and improved social function. In a trial of only 108 patients, supplementation was associated with improved red blood cell omega-3 fatty acid levels versus placebo. The findings are encouraging but not enough to stand alone. Thus, larger scale studies are needed to evaluate strategies of supplementation, which in previous analyses have been shown to improve depressive symptoms and may be particularly effective in the heart failure population, which appears to be resistant to many conventional therapies for depression.

As a community of caregivers of patients with heart failure, we have made significant strides in understanding how to treat comorbidities with great progress in the areas of hypertension, ischemic heart disease, and diabetes. However, a comorbidity that affects not only clinical outcomes but also quality of life and patient-reported outcomes continues to be a challenging one with few gains, other than the importance of heightened awareness. Let us hope that the OCEAN trial becomes a forceful river that stimulates further investigation and guides our interventions so that this resistant morbidity can be tackled during our lifetime.

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