

Particularity of Patients with Recovered Ejection Fraction; Experience of Heart Failure Unity of Cardiology Department - Ibn Rochd University Hospital

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Abstract

To check if the HF-Recovered population has a distinct clinical phenotype, biology, and prognosis by analyzing baseline clinical data, biomarker data representing several key biological pathways (neurohormonal activation, myocyte stress and injury, oxidative stress and inflammation), and long-term clinical outcomes.

Keywords: heart failure; improved ejection fraction; reduced ejection fraction

Introduction

Heart failure with recovered or improved ejection fraction (HFIEF) has been proposed as a new category of heart failure HF (1) representing patients with reduced left ventricular ejection fraction (LVEF) who have shown an improvement in ejection fraction, spontaneously or in response to a therapeutic intervention.

Patients and methods:

We retrospectively reviewed the database of 170 patients with reduced fraction ejection followed in the heart failure unity of cardiovascular department of Ibn Rochd Hospital University from January 2022 to January 2023.

For this analysis, participants were classified into 2 categories of HF: Heart failure with reduced ejection fraction (HF-REF) and Heart failure with improved ejection fraction (HFIEF). Patients were classified as having HF-REF if the enrollment echocardiogram showed an EF <50% and those with a documented history of EF <50% who have demonstrated an improvement in ejection fraction (EF) were considered to have HF-Recovered. Patients with preserved ejection fraction EF ≥50% and incomplete baseline information were excluded.

Clinical and biological parameters (Brain natriuretic peptide (BNP), troponin I, C-reactive protein, uric acid, and creatinine) were compared across the 2 categories of HF.

Results:

Of the 235 patients in our cohort, 65 patients had insufficient baseline data to include in this analysis. In the final 170 patients were included in our study cohort. The time period between enrolment and the control transthoracic

echography was on average 7 months. Patients who had shown an improvement of LVEF were 11.1% (19 patients). However, 88.8% (151 patients) had a persistent reduced LVEF (HFREF). The mean age was 62.3 years and the gender ratio was 1.8. Diabetes was the most common comorbidity (42.7%, n=73) followed by Hypertension (HTA) (39.3%, n=67). Patients with HFIEF were younger (mean age: 55.5 vs 64.7; p=0.04), had more recent onset of heart failure (39.4% vs 22.7%; p=0.01) and less prevalence of diabetes (19% vs 50%; p=0.04). HF-Recovered patients had less severe symptoms, with a greater prevalence of patients in New York Heart Association class I or II than the HF-REF populations. Also, they tended to have lower systolic blood pressure, better renal function, and larger left ventricular diameter at end diastole. Prevalence of atrial fibrillation (AF) were similar (16.7% vs 28%; p=0.4). As for the ethology of heart failure, ischemic cardiomyopathy was the most frequent for both groups followed by hypertensive cardiomyopathy in HFIEF and valvular cardiomyopathy for patient with persistent HFREF.

Demographics and Medical History

The mean age was 62.3 years and the gender ratio was 1.8. Diabetes was the most common comorbidity (42.7%, n=73) followed by Hypertension (HTA) (39.3%, n=67). Patients with HFIEF were younger (mean age: 55.5 vs 64.7; p=0.04), had more recent onset of heart failure (39.4% vs 22.7%; p=0.01) and less prevalence of diabetes (19% vs 50%; p=0.04).

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Clinical Measures

Systolic blood pressure in the HF-Recovered group was lower than in HF-REF. There were no significant differences between mean serum sodium levels across the groups.

Biochemical profiles

Serum creatinine, BNP, troponin I were lowest in the HF-Recovered group and highest in the HF-REF group. Uric acid was also lowest in the HF-Recovered group. There was no C-reactive protein across HF groups. However, nearly a third (30%) of HF-Recovered patients still had a BNP level above the 95th percentile (135 pg/mL), suggesting persistent neurohormonal activation (2). Nearly half of the HF-Recovered group had evidence of oxidative stress, with 47% of patients having uric acid levels above the 95th percentile (2.6–6 mg/dL for women and 3.5–7.2 mg/dL for men) (3). Detectable troponin I levels were seen in 44% of the HF-Recovered patients.

Adverse cardiovascular outcomes

The HF-Recovered group had a substantial number of hospitalizations that was lower than the incidence in the HF-reduced group.

Discussion

The HF-Recovered patient, we also believe, represents a third, distinct category along the continuum of HF. Punnoose et al [4] reported that nearly 70% of patients with normal EF on echocardiography in their tertiary care referral center registry had had a reduced EF previously and suggested that this group of patients represents a separate clinical entity.

Our data suggest that the HF-Recovered population has different demographics, comorbidities, and symptom severity compared with the HF-REF populations. In this study, we compared key biological pathways across these populations by using traditional biomarkers (BNP, uric acid, high-sensitivity C-reactive protein, and troponin).

These findings suggest that there is persistent neurohormonal activation, increased oxidative stress, and cardiomyocyte injury and stress despite apparent recovery of EF. Although HF-Recovered patients had the best prognosis in terms of readmission for acute heart failure. We note that the HF-Recovered patients did not, by any means, have a normal prognosis. Despite normalization of EF, these patients continued to experience substantial HF symptoms and clinical events.

From a patient care perspective, these findings provide a rationale to continue background medical or device therapy for HF-Recovered patients. This recommendation is consistent with prior reports suggesting that the

cessation of medical therapy was associated with a recurrence of LV dysfunction in patients who had previously improved or “recovered” their EF. [5,6] In fact, we note that the majority of patients in our HF-Recovered group remained on medical therapy despite normalization of LV function. This is consistent with the idea that a recovery of EF does not necessarily constitute recovery from HF.

Conclusion

We suggest that the HF-Recovered EF population represents a distinct HF phenotype with biochemical properties and natural history that differ from the traditional HF-REF populations. Furthermore, these patients continue to experience HF events, suggesting that this is not true myocardial recovery. Our study underscores the need to further investigate pathophysiological differences in these patient populations in an effort to better tailor therapy. It also highlights the need to identify characteristics and predictors of both reverse remodeling and myocardial recovery in the pre-LV assist device cardiomyopathy population.

Conflict of interest

None declared

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