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EDITORIAL

Top-notch insights into heart failure

The heart is a vital muscular organ that operates as the central pump of the circulatory system. In heart failure (HF), the heart's ability to pump blood efficiently is compromised, leading to inadequate systemic and pulmonary circulation [1–3]. HF can stem from various aetiologies, including ischaemic cardiomyopathy, hypertensive heart disease, or dilated cardiomyopathy, which result in pathological alterations in myocardial structure and function [4–8].

Chronic obstructive pulmonary disease (COPD) and HF often coexist, with each condition exacerbating the other's symptoms and progression [9]. The systemic inflammation and oxidative stress seen in COPD can lead to myocardial dysfunction, while HF can impair pulmonary function, worsening COPD symptoms [10,11]. This bidirectional relationship increases morbidity and mortality, highlighting the need for integrated management of both conditions. Zhang et al. investigated whether patients with both HF with reduced ejection fraction (HFrEF) and COPD have greater muscle weakness and fatigue compared to those with COPD alone. The study included 25 male patients with HFrEF + COPD and 25 with COPD alone, all with moderate-to-severe COPD. Muscle performance was assessed using isokinetic dynamometry, and functional capacity was measured *via* cardiopulmonary exercise tests, the 6-min walk test (6MWT), and the 4-min step test. Results showed that the COPD-only group had worse lung function but the HFrEF + COPD group had significant knee flexor muscle impairment. Knee flexor power, adjusted for muscle mass, correlated significantly with the 6MWT, step test, and peak work rate. The presence of HFrEF in COPD patients worsens muscle weakness, contributing to increased exercise intolerance and dyspnoea [9].

Cardiovascular involvement in rheumatoid arthritis (RA) increases morbidity and mortality, but early intervention can reduce cardiovascular risks. Gök et al. examined the impact of steroids and methotrexate (Mtx) on heart function in newly diagnosed RA patients. Thirty-six patients were evaluated using echocardiography and Doppler parameters before treatment, after 1 month of steroid treatment, and after 3 months of Mtx treatment. The mean patient age was 52.66 years. Treatment significantly reduced inflammatory markers (i.e. CRP) and disease activity score (DAS28). Improvements were observed in left ventricular (LV) tissue Doppler measurements and tricuspid Doppler values after treatment. The most significant changes were noted after Mtx treatment. Steroids and Mtx significantly improved LV systolic and right ventricular diastolic functions in RA patients [12].

Cardiac amyloidosis, characterised by amyloid protein deposition in myocardial tissue, leads to restrictive cardiomyopathy and impaired ventricular function. This results in progressive HF, manifesting as reduced cardiac output and diastolic dysfunction. Early detection and targeted therapies are essential for managing cardiac amyloidosis and mitigating HF progression [13–15]. In their study, Yu et al. aimed to report genotypes and phenotypes of hereditary transthyretin (TTR) cardiac amyloidosis (hATTR-CA) in a Western Chinese cohort and review genetic profiles in the Chinese population. TTR gene sequencing and endomyocardial biopsy were performed on probands and relatives from West China Hospital between 2018 and 2021. TTR gene alterations were identified in five probands and their two relatives, revealing three variants: Ser23Asn, Glu54Leu, and Thr60Ala. The Glu54Leu variant was newly reported as pathogenic in Chinese hATTR-CA patients. The median diagnosis age was 56 years, with an 8-year delay from onset to diagnosis. Endomyocardial biopsy and TTR immunohistochemistry confirmed diagnoses in all patients. Early TTR genotypic screening and biopsy might be recommended for timely diagnosis [6].

Ischaemic heart disease (IHD) is a leading cause of HF, as reduced blood flow from coronary artery disease can damage myocardial tissue and impair cardiac function. The chronic oxygen deprivation from IHD leads to myocardial infarction and LV dysfunction, progressing to HF. Effective management of IHD is crucial in preventing the onset and progression of HF [5,16,17]. Pan et al. assessed the impact of multivessel revascularisation versus infarct-related artery (IRA)-only revascularisation in 646 AMI patients with multivessel CAD. Multivessel revascularisation significantly reduced cardiovascular deaths over a 60.6-month follow-up. For Killip I–II patients, cardiovascular deaths were 2.6% in the multivessel group versus 9.5% in the IRA-only group. No significant difference was found in Killip III–IV patients. Multivessel revascularisation showed a notable benefit, particularly in Killip I–II patients [7].

Hypertrophic cardiomyopathy (HCM) is a genetic disorder often leading to LV outflow tract obstruction and HF symptoms [18]. In their study, Akhan et al. aimed to identify differences between obstructive (Obs-HCM) and nonobstructive HCM (Nonobs-HCM) through electrocardiographic (ECG) and echocardiographic (ECHO) evaluations. The subgroup analysis included 60 HCM patients, 23 obstructive and 37 nonobstructive, from the 'LVH-TR study.' Obs-HCM patients had significantly higher body surface area, ST-segment depression, QT durations, LV mass index, and systolic anterior motion (SAM) rates compared to Nonobs-HCM patients. ST-segment depression, QT prolongation, and SAM were significant predictors of obstruction [8].

The goal of therapy for chronic HF is to improve symptoms management and quality of life, decrease

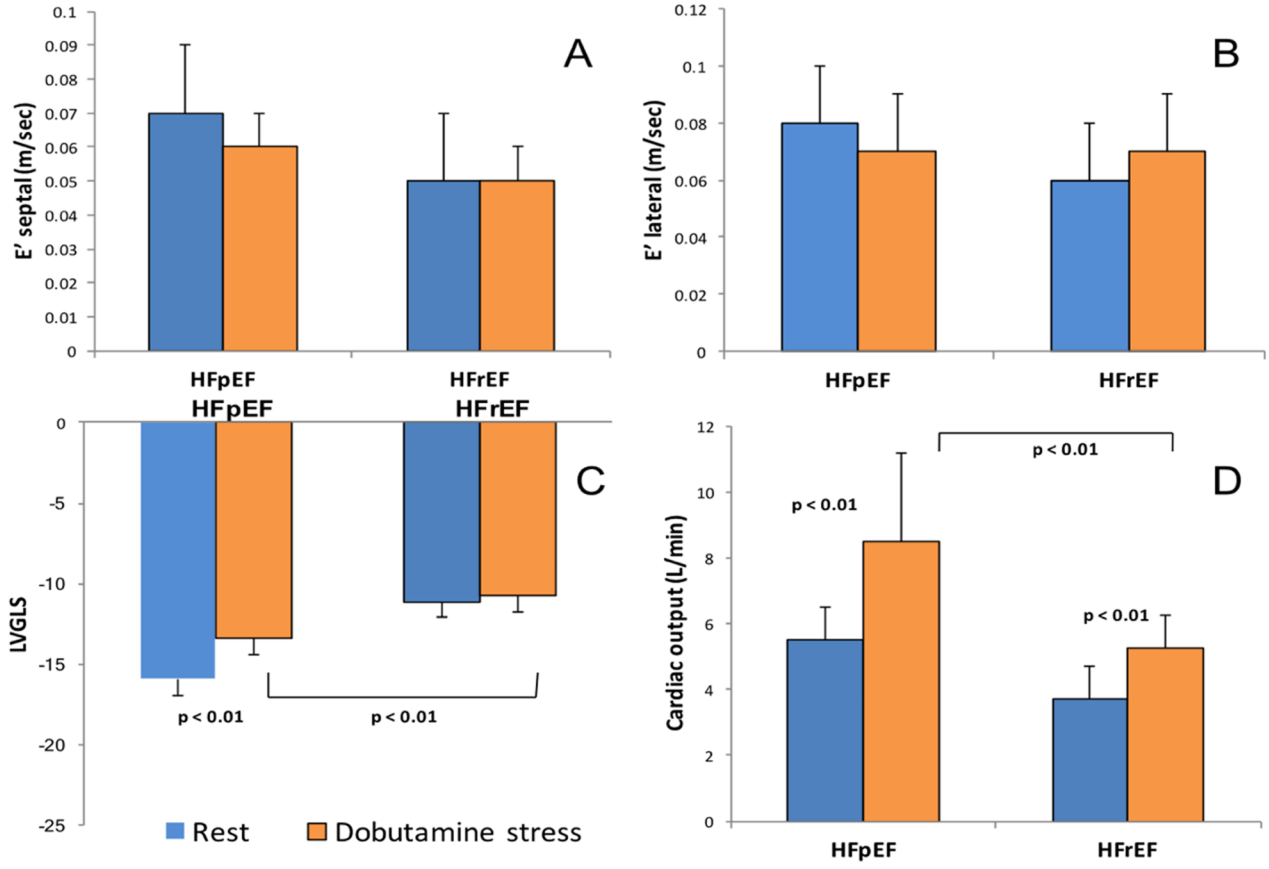

Figure 1. Bar diagram showing changes in early (E') tissue Doppler velocity at septal mitral annulus (a), at lateral mitral annulus (B), left ventricular global longitudinal strain (C) and cardiac output (D) between rest and dobutamine stress echo in the cohort (from reference [24].

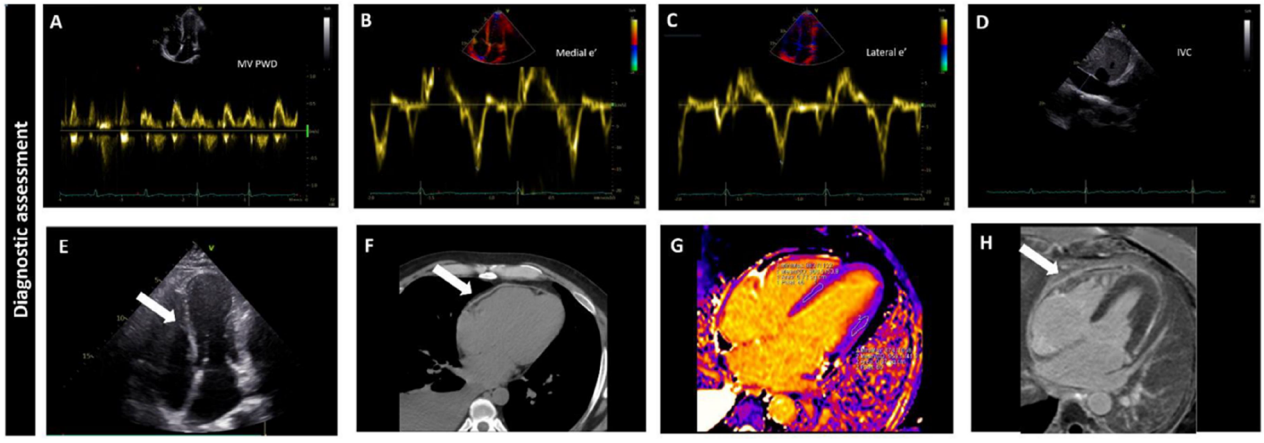

Figure 2. The echocardiogram showed apparently normal left ventricular (LV) systolic and diastolic function (Panel A) with mitral medial e' velocity (Panel B) higher than the lateral one (Panel C), the so called annulus reversus. IVC was markedly dilated without any variation with inspiration (Panel D). During inspiration the interventricular septum showed a left-sided shift (white arrow on Panel E). A thoracic computed tomography was performed and showed increased pericardial thickness without calcifications (white arrow on Panel F); and a cardiac magnetic resonance showed normal myocardial T1 mapping (Panel G) and diffused pericardial late gadolinium enhancement (white arrow on Panel H) (From reference [26].

hospitalisations, and decrease overall mortality associated with this disease. The core foundational medication classes for HFrEF includes a renin-angiotensin system inhibitor (such as an angiotensin receptor neprilysin inhibitor (ARNI), angiotensin-converting enzyme inhibitor (ACEi), or angiotensin II receptor blockers (ARB)), a beta-blocker, a mineralcorticoid receptor antagonist (MRA) and a sodium-glucose co-transporter 2 Inhibitor (SGLT2i)

[2,19–22]. Afshani et al. investigated the impact of empagliflozin, an SGLT2i, on cardiac structure and function in patients with type 2 diabetes or prediabetes and HFrEF [23]. Conducted as a randomised, double-blind trial with 104 participants, empagliflozin (10 mg daily) alongside standard HFrEF treatments significantly reduced LV end-diastolic volume index and end-systolic volume index by 10.0 and 8.0 mL/m^2, respectively ($p < 0.0001$). The empagliflozin group also showed a substantial increase in LVEF ($p < 0.0001$) and a lower hospitalisation rate for HF compared to the control group (3.8% *vs.* 23.1%; $p = 0.008$). These findings underscored empagliflozin's potential as a beneficial therapy for improving cardiac outcomes in patients with HFrEF and diabetes or prediabetes.

Pharmacological stress test results in haemodynamic changes in HF patients. In their study, Sengupta et al. aimed to compare the hemodynamic responses to dobutamine stress between HFrEF and HFpEF patients [24]. Forty patients with each condition underwent dobutamine stress echocardiography. The duration of the stress test was similar between HFrEF and HFpEF groups. Dobutamine infusion did not significantly change LVEF or stroke volume (SV) in either group, but HFpEF patients exhibited a higher E/e' ratio at peak stress. HFpEF patients showed a significant increase in LV global longitudinal strain (LVGLS) and a decrease in left atrial and LV stiffness indices post-dobutamine, whereas HFrEF patients did not exhibit these changes (Figure 1). Additionally, peak longitudinal strain indices (PALS and PACS) were lower in HFrEF both at rest and after stress compared to HFpEF. These findings highlight distinct hemodynamic responses to pharmacological stress in HFrEF and HFpEF, contributing to a better understanding of their pathophysiological differences.

In this issue of Acta Cardiologica, alongside the original article mentioned, several focus images have also been featured (Figure 2) [25–32].

Disclosure statement

Nothing to disclose.

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