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Mini Review

Heart failure with preserved ejection fraction: A mini review

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Over the past 20 years, there has been a surge of clinical investigations and useful trials on heart failure (HF) with preserved ejection fraction (HFpEF). HFpEF represents the largest phenotype of HF, a public health concern for its link with high mortality and rates of morbidity. In this mini review, we recap HFpEF details from definition to management strategies through pathophysiological standpoints. HFpEF associates HF symptomatology (dyspnea on exertion, fatigue), biochemistry (raised natriuretic peptide levels) along with echocardiographic evidence (left ventricular diastolic dysfunction or significant structural heart disease, with EF ≥ 50%), and in particular occurrences invasive hemodynamic sizes [1]. HFpEF is characterized by an elevation in left ventricular pressures, the cornerstone of diastolic dysfunction. Notwithstanding apparent preserved systolic function, hidden abnormalities may lead to left atrium dysfunction, pulmonary hypertension and alteration in right ventricular function.

The prevalence of HFpEF has been increasing with age and its comorbidities. Several similarities (risk factors) and differences (comorbidities) have been reported in HFpEF versus HF with midrange EF (HFmrEF) and HF with reduced EF (HFrEF) with regards to their epidemiology (percentages of each subgroup), clinical indicators (left ventricular measurements), lab findings (NT-proBNP concentrations) and underlying mechanisms (inflammation, endothelial dysfunction, diastolic dysfunction linked with myocardial fibrosis and calcium homeostasis). The HFpEF patients are more likely to be older and of female gender while the HFrEF ones are mostly of male gender [2]. Production of natriuretic peptides and their circulating levels are lower in HFpEF than in HFrEF as hypertrophic hearts with small left ventricular cavity in HFpEF patients do not substantially elevate end-diastolic wall stress. Patients with HFmrEF had larger left ventricular and atrial dimensions but lower left ventricular ejection fraction when compared with the HFpEF.

The pathophysiology of HFpEF, largely undetermined, lies in coronary microvascular, large vessels dysfunction; and extracardiac pathologies such as obesity, diabetes mellitus, metabolic syndrome, and kidney disease which frequently cohabit, initiating intramyocardial inflammation. Neuro-adrenergic overactivity and baroreflex dysfunction common to all forms of HF appear directly proportional to the alteration of the left ventricular ejection [1,3]. Recent data have pointed out the link between HFpEF and comorbidities such as arterial hypertension, atrial fibrillation (AF)/atrial flutter, anemia, hemochromatosis, and chronic obstructive pulmonary disease (COPD). Moreover, HFpEF patients exhibit systemic vascular abnormalities (abnormal peripheral vessel compliance), endothelial and adipocyte modifications, and changes in skeletal muscle (sarcopenia and decreased oxygen utilization). In postmenopausal women, studies have identified estrogen (E2) deficits and estrogen receptor (ERs) lethargy to induce left ventricular diastolic dysfunction and progress in HFpEF. Infiltrative cardiomyopathies; such as amyloidosis, have been pointed out as seldom etiology of HFpEF [4].

Despite its increasing prevalence, the understanding and treatment of HFpEF remain limited. Based on the results of referenced studies, the American College of Cardiology (ACC) and the American Heart Association (AHA) have made some recommendations for the management of HFpEF. The SPRINT study mentioned the role of intensive blood pressure control in preventing HF and improving cardiovascular outcomes. This study highlighted the benefit of effective control in hypertensive patients with HFpEF (Class 1c). In non-symptomatic HF patients with high levels of natriuretic peptides, the EMPEROR-Preserved study underlined the significant benefit of SGLT2i such as dapagliflozin

and empagliflozin in decreasing HF hospitalizations and cardiovascular lethality (Class 2a) [5]. Due to the absence of sizable randomized studies, the management of HFpEF patients with AF can be modeled on the clinical recommendations for AF (Class 2a) [6]. Regarding the use of MRA in patients with HFpEF, the TOPCAT study showed its beneficial effect in increasing diastolic function and reducing HF hospitalizations (Class 2b) with an additional risk of hyperkalemia and renal failure. Angiotensin type 1 receptor blockade (ARB) and angiotensin receptor-neprilysin inhibition (ARNi) have been found to decrease HF hospitalizations in patients with HFpEF (Class 2b) [7].

In addition, hypertension treatment, use of SGLT2i, and lifestyle changes such as weight loss, dietary approach, physical activity, and cardiorespiratory fitness may reduce the incidence of HFpEF [8]. On the other hand, the use of left atrial assist device (LAAD) realized by transcatheter implantation of an interatrial shunt device (IASD) to increase aortic pression and decrease LA pressure in keeping arterial pulsatility, although promising, needs further studies [9].

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