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Diastolic Dysfunction in Coronary Artery Disease

Sir,

Diastolic dysfunction (DD) of the heart refers to an increased stiffness and abnormal relaxation of the left ventricle leading to impaired filling during diastole. Despite this simple definition, truly understanding the cause of DD and its interrelationship with myocardial ischemia and hypertension is extremely complex. DD and coronary artery disease (CAD) are intertwined. Myocardial ischemia plays a role in the pathophysiology of DD,¹ DD has been shown to alter the clinical course in CAD patients, and CAD presents a therapeutic target for DD for which no currently available treatments are known to affect outcome.² It has been recognized, however, that some patients with DD have no symptoms while others with similar abnormalities present with overt heart failure.³ The trigger that tips DD patients into the symptomatic phase is unknown. Since there is a heavy load (significant burden) of patients in the OPDs with DD and coronary artery disease (CAD), we aimed to investigate the status of DD in advanced CAD patients.

We retrospectively studied hospital records of 34 randomly selected patients admitted for CABG from 2013 to 2015 at the Aga Khan University Hospital (AKUH). Patients were divided into two groups, i.e. preserved EF (EF \geq 50%), and reduced EF (EF $<$ 50 %). E/e' ratio was used as one of the measures of diastolic dysfunction.⁴ Heart failure (HF) was defined by the clinical sign and symptoms, and designation of NYHA class II and above by the physician in the medical records.

This study confirms the previous reports that 40-50% of advanced CAD patients requiring revascularization present with preserved EF. The results show that among patients with preserved EF, 33.33% of them presented with \geq NYHA class II symptoms. This shows that advanced CAD patient population has well defined clusters of DD, HF with preserved EF, and HF with

reduced EF. This observation emphasizes the need to document and focus on diastolic function status in coronary artery disease patients, as it is directly related to their prognosis. The results also show that there is an inverse relationship between DD and systolic dysfunction. The trend is towards increased levels of DD with greater degrees of systolic dysfunction.

Previous studies have also reported that CAD patients with HF and preserved EF experienced a 4-fold greater decline in EF over time.⁵ This observation reiterates the importance of looking beyond the EF in CAD patients and targeting these patients with DD for aggressive control of comorbidities until specific treatment for DD becomes available.

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