Heart Failure with Preserved Ejection Fraction

(Is This Diastolic Heart Failure ?)

Is it Failure to Preserve, Failure to Reserve, and Failure on the compliance Curve

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Epidemiological studies have established that approximatey half of all patients with CHF have a preserved LV ejection fraction. (HFpEF). This is labelled by some as Diastolic Heart Failure (DHF). This syndrome is predominantly seen in older HT individuals. The prevalence of HFpEF is progressively increasing (1), generally seen in the population towards old age. Although the HFpEF was previously thought to have more favourable course than HF with reduced ejection fraction (HFrEF) , recent studies have shown that the mortality rate (1), Hospital readmission rate (2) and the economic cost (3) of both is almost same.

Several features of the pathophysiology of HFpEF have been well characterized, including structural and functional alterations in the heart, such as myocardial hypertrophy, changes in composition of extracellular matrix and abnormalities in intracellular calcium handling (4). These cellular and biochemical alterations may be responsible for impaired LV diastolic relaxation and decreased LV compliance that is observed in HFpEF (5,6). However these abnormalities are not specific for HFpEF, as they can be found with HFrEF and HT individuals without HF (7). Thus, some investigators (8) have proposed that the pathophysiology of HFpEF may involve additional CV alterations beyond diastolic dysfunction, such as impairment of systolic function. It is impotant to note that EF is a crude measure of LV systolic function.and that is influenced by many factors beyond contractility per se, including loading conditions and chamber geometry.

Recent studies (9,10,11) of systolic function in HFpEF have focused on indices other than LVEF. However , these studies have yielded conflicting results, with some reporting abnormalities (9,10) in systolic functions and other observing no abnormalities in systolic function.

In one of the studies by Borlaug et al (12) provides important insights into the systolic function of the patients with HFpEF. By using landmark Rochestor Epidemiology Project, 3 groups of subjects were examined: Healthy control patients without CV disease (n=617), HT control patients without HF (n= 719), and patients with HFpEF (n= 244). The auther noninvasively assessed load – dependent indexes of chamber-level contractility (pre-load recruitable stroke work and wall stress-corrected endocardial fractional shorterning) and myocardial contractility (stress-corrected midwall fractional shortening).

These indexes were greater in HT than in Normotensive control groups. In contrast, these were lower in patients with HFpEF than both HT and normotensive control groups. These findings indicate that despite the apparently normal "preserved" EF, the patients with HFpEF exibit evidence of impaired contractile function.

Importantly, because the impairment in the contractility in HFpEF is mild, it is unlikely to be the culprit mechanism that underlies the pathogensis of HFpEF. Instead, the impaired contractility in HFpEF is due to other alterations in myocardial structure and function. And that these altarations are the ones that are responsible for the transition of a HT heart to failing heart.

In the study by Borlaug et al. (12), all of the cardiovascular measures were assessed in the resting state. From a clinical perspective, one of the hallmarks of HFpEF is that symptoms are usually not reported at rest, but may become clinically manifest during low levels of exertion and may impose marked limitations in exercise tolerance. Only small number of studies have investigated the alterations in CV response to exercise that charactersize patients with HFpEF (13-

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16). In the study by Phan et al. (17) 20 healthy control and 37 patients with HFpEF were examined at rest and during submaximal aerobic exercise. Consistant with findings from previous studies (15,16,18), patients with HFpEF had evidence of chronotropic incompetence during exercise, which manifested as a deficit in their heart rate reserve. During exercise, patients with HFpEF also exhibited significant deficits in their ability to augment several indices of systolic function.

As reviewed by Dr Jagtap, there may be significant LV systolic dysfunction in patients with HFNEF which is not apparent on routine echocardiographic examination. Hence LV function and LVEF are not interchangeable. The predicted reduction in stroke volume in precompensated state rather than LVEF may explain symptoms of heart failure. Diuretics, ACE Inhibitors, ARBs and Aldosterone antagonist are used in the management, however betablockers are used in patients with LV systolic dysfunction and may not be useful in HFNEF as observed in SENIORS study.

In the present study on Assessment of Diastolic dysfunction in Obesity, the diastolic dysfunction was more with increasing grades of obesity. In most of the studies Systolic dysfunction was associated with diastolic dysfunction and was more with grades of obesity. It was not seen in the present study as only 11.6 % of these patients were having severe obesity. 2D Echo and Doppler study is simple bedside investigation to assess LV systolic and diastolic function in obese patients. To begin with, diastolic dysfunction is predominant and then systolic dysfunction starts as the grade of obesity increases.

Some studies(12,17) provide valuable insights to our understanding of the pathophysiology of HFpEF. However, additional studies are needed for complete understanding. Some should be directed towards the pathophysiologic mechanisms and others on developing interventions that target the specific mechanisms that have been identified. These patients are burdened by abnormalities in diastolic function, deficits in exercise reserve, and some compromise in their systolic function. Similarly there is failure on the part of medical experts to develop efficacious interventions that improve their morbidity and mortality.

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