Combined cardiopulmonary-stress echocardiography testing in discovering latent HFpEF

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Heart failure with preserved ejection fraction (HFpEF) has overtaken heart failure in the setting of reduced ejection fraction (HFREF; also known as systolic heart failure) as the most common form of HF and now comprises more than 50% of all patients with HF¹². Still, the recognition of HFpEF can be difficult due to the multiple confounding co-morbidities that can impair exercise capacity and mimic the signs and symptoms of HF³. Thus, the clinician’s ability to make early diagnoses and timely initiate therapeutic interventions is often limited.

Here, we present a case in whom we through the combined cardiopulmonary and exercise stress-echo-cardiography testing (CPET-ESE) discovered masked/latent HFpEF.

A 50 year old man came to our lab for CPET testing due to the exercise-induced chest pain and dyspnea, which began to be occurring during his regular work in the last few months. Symptoms usually stoped after rest. He has been taking medications for hypertension (HTA) and hyperlipoproteinaemia (HLP) and had chronic left bundle branch block (LBBB). His physical exam was unremarkable. Echocardiogram at rest revealed signs of left ventricular (LV) hypertrophy with normal regional contractility and normal systolic function. Mitral inflow pattern showed impaired relaxation of LV, but with normal LV filling pressures; E/E’ at rest was 6,5 (figure 1). Based on symptoms patient reported and echocardiography results at rest, we decided to check the possible existence of ischemic heart disease, as well as to check the systolic/diastolic function of the heart. Thus, we have performed CPET-ESE testing on semi-supine ergobicycle, using Ramp 15 protocol. We used the tissue Doppler imaging measurements (E’) and mitral inflow early wave measurement (E) both at rest and at maximal effort during the combined ESE-CPX test for identification of the diastolic dysfunction and increased LV filling pressure.

Patient performed test for 12 minutes and achieved workload of 114 watts when he stopped due to the dyspnea. He had not reported any chest pain during or after the test. The echocardiography analysis during and after the test showed no changes in regional LV contractility or wall motion abnormalities. However, his peak oxygen consumption (VO2 peak) was lower than expected for his age and sex, and his ventilatory efficiency (VE/VCO2 slope) was mildly impaired (class II), figure 2. Importantly, stress-echocardiography revealed significant increase in LV filling pressure during exercise (E/E’ was 16), while mitral inflow pattern pointed to restrictive LV filling; figure 3. Results of the combined CPET-ESE test pointed to the existence of HFpEF with no signs of ischemic heart disease.

Discussion

The value of ESE in identifying ischemic heart disease in patients with chest pain during exercise is well known...
(4). On the other hand, it has been only recently shown that combined CPET-ESE test is a feasible and reliable test can identify patients with masked HFpEF (5). Traditional echocardiographic parameters have been insensitive for the HFpEF diagnosis because they have tried to identify patients with dyspnea during exercise using only resting diagnostic criteria. So, ESE can mimic physiological condition to monitor at the same time diastolic function and LV filling pressure with Doppler echocardiography and symptoms by CPET6-7. The only prespecified criteria for the HFpEF diagnosis was a ratio of a Doppler early mitral flow (E wave) and tissue Doppler imaging of early mitral annulus movement (E') at peak exercise greater than 158. However, concomitant analysis of cardiopulmonary functional capacity can improve the specificity of changes in E/E' and can help in avoiding the overreliance on a single echocardiographic measurement during ESE.

Therefore, our decision to perform CPET-ESE test seemed reasonable having in mind patient’ symptoms. And the results of the test confirmed that our decision was the right one. Both, suboptimal increase in VO2 peak and significant increase in E/E' demonstrated that patient has HFpEF. In addition, it has been shown that HF-pEF patients have the lower peak VO2, the lower peak PetCO2 values, and the higher VE/VCO2 slope, which was also the case with our patient9-10. And the normal electrocardiogram and wall motion during and after exercise, together with the absence of chest pain, showed us that ischemic heart disease was not the cause for a increase in LV filling pressure and subsequent HFpEF. Many other mechanism, including cardiac, vascular and metabolic factors (4 u guazzi), may contribute to observed reduction in exercise capacity in our patient. Nevertheless, their net result has been demonstrated during CPET with lower VO2 peak and increase in VE/VCO2 slope.

Conclusion

In conclusion, to date, there is no approved therapy for HFpEF patients that reduces their high risk for serious adverse events. To improve their outcome, a deeper understanding of the subpopulations that fit under the HFpEF syndrome, and more specific test that can reliably identify these patients are needed. The combined ESE-CPET test has shown to be just that; a feasible and reliable test that can identify patients with masked HFpEF. The opportunity for early recognition could open a new window for future research to find a successful therapy for these patients.

References: