DETERMINANTS OF CARDIOPULMONARY EXERCISE TESTING PERFORMANCE IN SEVERELY OBSTRUCTIVE HYPERTROPHIC CARDIOMYOPATHY

Poster Contributions
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Background: Patients with hypertrophic obstructive cardiomyopathy (HOCM) suffer functional limitation demonstrated by cardiopulmonary study (CPX). It is unclear whether left ventricular outflow tract (LVOT) obstruction is the primary determinant of exercise capacity. We aimed to establish the determinants of peak oxygen consumption (pVO2) and ventilatory efficiency (VE/VCO2) in patients with severe HOCM referred for septal ethanol ablation.

Methods: Symptomatic patients with HOCM underwent echo, CPX using upright bicycle ergometry, spirometry, and cardiac catheterization prior to septal ethanol ablation.

Results: We studied 65 patients (age 59 ± 13 yrs, 59% male, class III/IV =80%) with severe HOCM (septum = 20 ± 3 mm, LVOT gradient =75 ± 41(rest) and 158 ± 48mmHg (provoked). Estimates of left ventricular filling pressures and RVSP were $E'/E$ ratio = 14 ± 7 and RVSP = 39 ± 10mmHg, respectively. Exercise duration was 9 ± 3 min. There was poor functional capacity with pVO2= 15.6 ± 4.4 mL/kg/min (64 ± 16 % of predicted) and high VE/VCO2 = 38 ± 8 (normal <35). Neither LVOT gradients nor septal thickness was correlated to pVO2 or VE/VCO2. The echo parameters with the strongest correlation to pVO2 and VE/VCO2 were $E'/E$ (r =-0.41, P = 0.002; r =-0.51, P<0.001) and RVSP (r = -0.41, P = 0.001; r =-0.47, P <0.001), respectively. Multivariate linear regression revealed independent predictors of pVO2 were $E'/E$ (B coefficient -0.19, P=0.005), % predicted forced vital capacity (B coefficient 0.09, P=0.002) and heart rate reserve (B coefficient -0.06, P=0.06) with R2=0.48 (adjusted for age). The independent predictors of VE/VCO2, on multivariate linear regression were $E'/E$ (B coefficient 0.28, P=0.03), %predicted forced vital capacity (B coefficient -0.13, P=0.01) and left ventricular end systolic diameter (B coefficient -0.55, P=0.01) with R2=0.54 (adjusted for age).

Conclusion: Patients with severe HOCM have significantly impaired exercise capacity. Elevated filling pressures, and not septal hypertrophy and LVOT obstruction, appear to contribute predominantly to reduced aerobic capacity and poor ventilatory efficiency in this cohort.