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Heart Failure and Cardiomyopathies

SERUM BIOMARKERS OF EARLY STAGES OF HYPERTROPHIC CARDIOMYOPATHY IN A YOUNG POPULATION

Moderated Poster Contributions
Heart Failure and Cardiomyopathies Moderated Poster Theater, Poster Hall B1
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Session Title: New Insights in Hypertrophic Cardiomyopathy
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Background: Hypertrophic cardiomyopathy (HCM) is the most common monogenic cardiac disorder and the leading cause of sudden cardiac death in the young. Although in a majority of HCM cases there are gene mutations coding for sarcomere proteins, the onset for the clinical consequences of these mutations are difficult to predict, as these mutations do not show any clear relationship to the degree of myocardial hypertrophy. Hence identification of early markers for this disease is important. The aim of this study was to investigate novel serum biomarkers reflecting myocardial remodeling, microfibrosis and coronary endotheliopathy in young presymtomatic HCM patients and in individuals at risk for developing HCM.

Methods: Eighty-nine participants (18 HCM patients, 14 HCM-risk individuals, and 57 healthy controls) with median age of 15 (range 0-30) years underwent assessment with echocardiography and serum analysis for myostatin, cathepsin S, endostatin, type I collagen degradation marker (ICTP), matrix metalloproteinase (MMP) 9, vascular (VCAM) and intercellular adhesion molecules (ICAM). In some individuals, myocardial perfusion was measured both at rest and after adenosine via magnetic resonance.

Results: Both cathepsin S and endostatin were increased in the HCM group ($p<0.05$) vs HCM-risk and control groups, and showed weak correlation to left ventricular mass (LVM; $p<0.001$; $r>0.3$) and diastolic function, expressed as E/e´ ($p<0.01$; $r>0.3$). In the HCM-risk group, myostatin was decreased ($p<0.05$), whereas ICAM was increased compared to the other groups ($p<0.05$). ICAM correlated with adenosine-induced myocardial perfusion ($p=0.04$, $r=0.4$). No differences in ICTP, MMP9 and VCAM were observed between the groups ($p>0.1$).

Conclusion: To the best of our knowledge, this is the first study to suggest early onset changes in biomarkers of myoblast regulation, endothelial function and matrix remodeling in young presymptomatic HCM patients and in HCM-risk individuals.