

LEFT ATRIAL STRAIN IS IMPAIRED IN CHILDREN WITH HYPERTROPHIC CARDIOMYOPATHY

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Background: Children with hypertrophic cardiomyopathy (HCM) that develop restrictive physiology or fibrosis have worse outcomes. Left atrial strain (LAS) is a non-invasive surrogate of left ventricular filling pressure in adults with hypertrophic cardiomyopathy. The utility of LAS in children with HCM is unclear. We hypothesized that LAS is impaired in children with HCM compared to normal controls and that LAS correlates with risk factors for HCM including septal thickness, left ventricular (LV) mass, and fibrosis.

Materials/Methods: Prospective echocardiography was performed in patients with HCM <21 years of age and healthy controls. Septal wall z-score, LV mass, and mitral inflow Doppler were obtained. LAS and strain rates (SR) were averaged from 2- and 4-chamber views with P-wave as reference. Strain values and SR were recorded at 3 phases: atrial contraction (ϵ_{ac}), atrial filling (ϵ_{fill}), and passive conduit (ϵ_{con}). If available, LV mass by cardiac MRI was noted and retrospective quantification of delayed enhancement (DE) was performed using the AHA 17-segment model.

Results: Thirty children with HCM (median age 14, IQR 6-17, 69% male) were compared to 43 controls (median age 14, IQR 9-16, 58% male). Children with HCM have significantly worse ϵ_{fill} , ϵ_{con} , SR_{ac}, SR_{fill}, and SR_{con} compared to controls. Sub-analysis in HCM patients found that LAS and SR measurements did not correlate with septal thickness or LV mass by echo. CMR was available in 16 patients, (+DE in 11, # segments range 0-12). Increasing LV mass correlated with worse ϵ_{con} ($r = -0.5$, $p = 0.02$), ϵ_{fill} ($r = -0.5$, $p = 0.02$), SR_{fill} ($r = -0.5$, $p = 0.04$), and SR_{con} ($r = 0.5$, $p = 0.04$). Increasing number of DE segments correlated with worse ϵ_{con} ($r = -0.5$, $p = 0.04$), ϵ_{fill} ($r = -0.5$, $p = 0.03$), and SR_{con} ($r = 0.6$, $p = 0.01$). Conventional mitral inflow did not correlate with DE or LV mass by CMR.

Conclusions: LAS is impaired in children with HCM compared to controls. LAS is worse with increasing LV mass and fibrosis by CMR. Further study of LAS as a prognostic tool in pediatric HCM is warranted. Additional study of LAS as a potential echocardiographic marker to differentiate between HCM and hypertrophic adaptation ("athlete's heart") is also warranted.