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Impact of Obesity on Left Ventricular Thickness in Children with Hypertrophic Cardiomyopathy.

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Introduction: Hypertrophic cardiomyopathy (HCM) is a genetic heart disease with diverse natural history. The hallmark of HCM is severe left ventricular hypertrophy (LVH). In adults with HCM, obesity additionally increases LVH. It is not known whether obesity can lead to further LVH in children with HCM.

Hypothesis: Obesity is associated with additional LVH in children with HCM.

Methods: Echocardiogaphic LV dimensions were determined from 2D and/or M-mode images according to established criteria in 504 children ≥2 and ≤20 years of age with phenotypic sarcomeric HCM. Children with HCM associated with syndromes and storage disorders were excluded. Genotype-positive, phenotype-negative patients were excluded. Echocardiographic measurements of interventricular septal thickness (IVST) and posterior wall thickness (PWT), and patients' weight and height at the time of the echo were collected. Obesity was defined as a body mass index (BMI) ≥95th percentile for age and sex, as defined by CDC guidelines. IVST data was available for 498 and PWT data for 484 patients.

Results: Patient age ranged from 2 to 20 years (mean ± SD, 12.5 ± 3.9) and 340 (68%) were males. Overall, patient BMI ranged from 7-50 (22.7 ± 6.1). Obesity (BMI 18-50, mean 29.1) was present in 140 children whose age ranged from 2-19.6 (11.3 ± 4.1). The overall mean IVST was 20.5 ± 9.6 mm and the overall mean PWT was 11.0 ± 8.4 mm. The mean IVST in the obese patients was 21.6 ± 10.0 mm and mean PWT was 13.3± 14.7 mm. The mean IVST in the non-obese patients was 20.1 ± 9.5 mm and mean PWT was 10.4 ± 4.3 mm. Obesity was not significantly associated with IVST (p=0.12), but was associated with increased PWT (0.0011). The average predicted value of PWT for a non-obese child was 10.4 mm but was 13.3 mm if obese.

Conclusions: Presence of obesity does appear to influence LV thickness in children with HCM. Obesity is associated with increased PWT. However, IVST appears to be independent of obesity. These findings could have implications in risk stratification strategies utilized to identify children at risk for sudden cardiac death. Whether obesity and its impact on LVH influences clinical outcomes in children with HCM needs to be confirmed by further studies.