Abstract: P5316
Birthweight, childhood growth patterns and left ventricular structure at midlife: Northern Finland 1966 Birth Cohort Study

Authors:
N. Korpela1, K. Kaikkonen1, J. Auvinen2, M.P. Tulppo1, J. Junnila1, J. Perkiomaki1, M.-R. Jarvelin3, H.V. Huikuri1, A.M. Kiviniemi1, 1Medical Research Center Oulu, Research Unit of Internal Medicine - Oulu - Finland , 2Center for Life Course Health Research, University of Oulu - Oulu - Finland , 3Imperial College London, Department of Epidemiology and Biostatistics, MRC-PHE Centre for Environment &x0026; Health - London - United Kingdom ,

Topic(s):
Risk Factors and Prevention – Epidemiology

Citation:
European Heart Journal (2019) 40 (Supplement), 3214

Background: Higher cardiac left ventricular (LV) mass (LVM) has independently been associated with cardiovascular risk and mortality. Changes in LV structure have been observed in children with obesity. However, information about the long-term influence of early growth on LV structure is limited.

Purpose: To explore the associations between early growth patterns, specifically birth weight, age and body mass index (BMI) at infant BMI peak and at childhood BMI rebound, and LV structure at midlife.

Methods: At the age of 46, a sample of the participants of the Northern Finland Birth Cohort 1966 took part in follow-up examinations. A randomly selected subpopulation was enrolled to echocardiographic examinations (n=1124). BMI at various ages in childhood was calculated from frequent anthropometric measurements collected from child welfare clinical records. Age and BMI at BMI peak (n=541, mean age 9.0 months) and at BMI rebound (n=657, mean age 5.8 years) were derived from random effect models.

Results: Birth weight (n=1124), BMI at BMI peak and at BMI rebound were directly associated with LVM (beta [standardized]: 0.171, 0.186 and 0.223, respectively, p<0.001 for all) and LVM index (LVMI) (beta: 0.146, 0.120 and 0.120, respectively, p<0.01 for all) at midlife. All the associations, except that between BMI at BMI peak and LVMI, remained significant after adjustments for gender, adult blood pressure, heart rate, antihypertensive medications, heart diseases and diabetes (p<0.05 for all). Significant interaction between gender and age at BMI rebound was observed in LVMI. Age at BMI rebound was inversely associated with LVMI in both genders, the association being stronger in males (beta: −0.212, p<0.001) compared to females (beta: −0.117, p=0.025) and independent of potential confounders. There were direct univariate associations of birth weight and BMI at BMI peak and rebound with LV relative wall thickness (p=0.009–0.019), which were largely explained by potential confounders. However, the association of birth weight with LV relative wall thickness retained its significance even after relevant adjustments (beta: 0.097, p=0.001). After additional adjustments for adult BMI, only associations of birth weight and BMI at BMI peak with LVM remained significant, which, however, did not remain significant after additional adjustment for adult height.

Conclusions: This study shows that birth weight and early growth patterns are associated with LV structure at midlife, which is largely explained by adult BMI. Therefore, the relationship between early growth patterns to LV structure in the adulthood may be largely explained by their strong tracking to obesity and related cardiovascular risk factors at midlife and to some extent by normal somatic growth.