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A History of Asthma From Childhood and Left Ventricular Mass in Asymptomatic Young Adults

The Bogalusa Heart Study

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ABSTRACT

OBJECTIVES This study aimed to examine whether a history of asthma from childhood is associated with left ventricular (LV) mass in adulthood.

BACKGROUND Asthma has been related to various cardiovascular risk factors affecting LV hypertrophy. The authors saw a need for a prospective study to analyze the relationship between a history of asthma from childhood and markers of LV mass among asymptomatic young adults.

METHODS Prospective analyses were performed among 1,118 Bogalusa Heart Study participants (average age at follow-up 36.7 ± 5.1 years), with a baseline history of self-reported asthma collected since childhood (average age at baseline 26.8 ± 10.1 years). LV mass (g) was assessed using 2-dimensional guided M-mode echocardiography and was indexed for body height ($m^{2.7}$) as LV mass index (LVMI; $g/m^{2.7}$). A multivariate linear mixed model was fitted for the repeated measures.

RESULTS After an average of 10.4 ± 7.5 years of follow-up, participants with a history of asthma from childhood had a greater LV mass (167.6 vs. 156.9; $p = 0.01$) and LVMI (40.7 vs. 37.7; $p < 0.01$) with adjustment for age, sex, race, smoking status, antihypertensive medication, heart rate, and systolic blood pressure (SBP). The difference of LVMI between group with asthma and the group without asthma remained significant after additional adjustment for body mass index (39.0 vs. 37.1; $p = 0.03$) and high-sensitivity C-reactive protein (38.4 vs. 36.6; $p = 0.04$). In addition, the authors found significant interactions between SBP and asthma on LV mass and LVMI (p for interaction <0.01, respectively). The associations between asthma and LV measures appeared to be stronger among pre-hypertensive and hypertensive participants (SBP ≥130 mm Hg) compared with participants with normal SBP (<130 mm Hg) (regression coefficient: 39.5 vs. 2.3 for LV mass and 9.0 vs. 0.9 for LVMI).

CONCLUSIONS The findings of this study indicate that a history of asthma is associated with higher LVMI, and this association is stronger among participants with pre-hypertension and hypertension. (J Am Coll Cardiol HF 2017;5:497–504)

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ABBREVIATIONS AND ACRONYMS

BMI = body mass index

CV = cardiovascular

CVD = cardiovascular disease

hsCRP = high-sensitivity C-reactive protein

LV = left ventricular

LVMI = left ventricular mass index

SBP = systolic blood pressure

Left ventricular (LV) hypertrophy is recognized as target-organ damage resulting from a chronic increase in pressure and volume overload, with an estimated prevalence of 14.9% for men and 9.1% for women in the general population, and 36% to 41% among hypertensive patients (1,2). Left ventricular mass indexed to height$^{2.7}$ (LVMI) predicts incident events, progression, and severity stages of heart failure (3). Increased LVMI, a cardiac subclinical measure indicating the extent of LV hypertrophy, is an independent risk factor for death and major cardiovascular (CV) outcomes (4,5). Besides traditional CV risk factors, emerging evidence from both experimental and observational studies has linked chronic inflammation to increased LV mass (6,7).

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Asthma is a chronic inflammatory disorder in which the airway becomes inflamed, narrow, and swollen, thus causing coughing, wheezing, shortness of breath, and chest tightness. The prevalence of asthma has been growing during the past decade with an estimated current prevalence of 8.6% in children (8) and 7.4% in adults (9). Emerging evidence from epidemiological studies has shown that asthma in adulthood is associated with an increased risk of premature death (10), coronary heart disease (11), and stroke (12). However, there is only 1 study analyzing the relationship between adult asthma and heart failure (13) and 1 cross-sectional study assessing the association between asthma and LV hypertrophy in older adults (14). We saw a need for a prospective study to assess the relationship between a history of asthma from childhood and LV mass. We hypothesized that having a history of asthma from childhood could be related to increased LV mass in later life.

In this study, we performed prospective association analyses between a history of asthma from childhood and LV mass in adulthood in the Bogalusa Heart Study, a large and long ongoing, community-based, biracial, children's cohort study of the natural history of CV risk factors and their impact on vascular and metabolic changes throughout the life span since 1973. In addition, we were particularly interested in testing the interactions between asthma and all other covariates on LV mass, such as demographics and major cardiovascular disease (CVD) risk factors.

METHODS

STUDY COHORT. The Bogalusa Heart Study is a biracial (65% white and 35% black), community-based, long-term investigation of risk factors and natural history of CVD (15) that was founded by Dr. Gerald S. Berenson in 1973. Twenty-three repeated cross-sectional surveys have been conducted since 1973, with serial observations every 2 to 3 years from childhood through adulthood. Baseline information on asthma was collected from 8,289 individuals during 1983 to 2007. Follow-up outcomes of CV subclinical markers were measured from 1995 to the present. After excluding subjects who had only baseline data (n = 6,464) or only follow-up data (n = 161) and whose CV markers were measured before self-reported asthma (n = 20), the remaining subjects formed a longitudinal study cohort, with 1 to 4 follow-up appointments. In addition, to minimize the potential bias caused by abnormal CV conditions, we excluded 52 participants (139 records of echocardiography) with self-reported or physician-diagnosed heart disease across all surveys during follow-up (including heart attack, stroke, angina pectoris, ventricular arrhythmia, mitral valve prolapse, valve replacement, enlarged left ventricle, congestive cardiac failure, blockage in a ventricle, tachyarrhythmia, mitral valve stenosis, heart murmur, unspecified mitral disease, congenital heart disease, arteritis, bypass surgery, and angioplasty). All subjects in this study (N = 1,118) gave informed written consent at each examination, and for those participants who were younger than 18 years of age, the written consent of a parent or guardian was obtained. Study protocols were approved by the Biomedical Committee of the Tulane University (New Orleans, Louisiana) Institutional Review Board.

HISTORY OF ASTHMA. Information on asthma history was obtained by questionnaire. During 1983 to 1994, asthma prevalence was measured by the child's parent or guardian's responding affirmatively to the question, "Does your child now have asthma or has your child had asthma in the past?" For children, the age of asthma diagnosis was approximated as the time when the questionnaire was completed. After 1994, young adults 18 to 50 years of age were asked, "Do you now have asthma or have you had asthma in the past?" For those young adults who reported a history of asthma, the age of asthma diagnosis was determined by answering the question, "When were you first diagnosed with asthma?" If this information was missing, the time when the questionnaire was completed became the proxy time. Among all 8,289 individual subjects (12,647 records), 3,189 participants provided a self-reported history of asthma (7,547 records) in 2 to 6 survey cycles. For participants with multiple asthma episodes, the first report of