Journal of the American Society of Hypertension 10(2) (2016) 108–114

Research Article

Relationship of ambulatory blood pressure and body mass index to left ventricular mass index in pediatric patients with casual hypertension

Prema Ramaswamy, MD*, Sathish Chikkabyrappa, MD, Keyur Donda, MD, Marina Osmolovsky, RDCS, Mary Rojas, PhD, and Daniela Rafii, MD

Department of Pediatrics, Maimonides Infants and Children's Hospital of Brooklyn, Brooklyn, NY 11219, USA

Manuscript received August 31, 2015 and accepted November 13, 2015

Abstract

Both obesity and hypertension are associated with left ventricular hypertrophy (LVH) in children. Our objective was to compare the prevalence of LVH in obese and nonobese subjects with casual hypertension who underwent ambulatory blood pressure monitoring (ABPM). Untreated children (aged 6–20 years) underwent 24-hour ABPM, and left ventricular mass index (LVMI) was measured. Subjects were classified into three groups: white coat hypertension (WCH), prehypertension (pre-HT), and hypertension (HT). The prevalence of LVH was compared between obese and nonobese subjects among the groups. Of 69 children who underwent ABPM, thirty-two patients (46%) had WCH, 13 (19%) had pre-HT, and 24 (35%) had HT. Mean age, BMI, and LVMI were similar in the groups (P = not significant [NS]). In all, 22 patients (32%) had LVH, with no difference among WCH versus pre-HT versus HT (37.5% vs. 46% vs. 16.7%, P = NS). Twenty-seven subjects (39%) were obese. The ratio of LVH in obese to nonobese was 55.5% to 16.6% ($P = .001$). In both pre-HT and WCH, patients with LVH had a significantly higher BMI z score ($P = .02$ and $P = .01$, respectively). LVMI correlated strongly with BMI z score ($P = .0001$) but not with any blood pressure parameter. Almost half of children with casual HT have WCH. LVH is prevalent in a third of children with HT, pre-HT, and WCH. In both pre-HT and WCH, patients with LVH were more likely to be obese. More than half of all the obese subjects had LVH. Obese children in all three groups may be at a greater risk for end organ damage. J Am Soc Hypertens 2016;10(2):108–114. © 2016 American Society of Hypertension. All rights reserved.

Keywords: Obesity; left ventricular hypertrophy; hypertension.

Introduction

Casual blood pressure (BP) measurement allows for detection of elevated BP. However, 24-hour ambulatory blood pressure monitoring (ABPM) allows for a more accurate assessment of the physiologic diurnal and nocturnal changes and variability in BP. Its usage in pediatric hypertension and obesity is increasing.[1–3] ABPM is also essential for the detection of white coat hypertension (WCH). This is especially important in children as evidenced by the almost 30%–50% prevalence of WCH in children with persistent mild to moderate casual BP elevation.[4,5]

Both obesity and hypertension have been demonstrated to be associated with left ventricular hypertrophy (LVH) in children.[5–8] LVH in both these conditions may be reversible.[6–8] In children with hypertension, some studies have shown a positive correlation between systolic BP (SBP) parameters as noted by ABPM and left ventricular mass index (LVMI) as measured by echocardiographic left ventricular mass (LVM).[9,10]

The relationship of body mass index (BMI) to LVMI and hypertrophy in the different categories of hypertension as classified by ABPM is not well understood. Our objective was to compare the prevalence of LVH in obese and nonobese subjects with casual hypertension who underwent ABPM.

Methods

Study Population

Medical charts for children ages 6–20 years seen at a pediatric hypertension clinic at Maimonides Medical Center from February 2008 to July 2011 were identified and

Conflict of interest: No conflicts of interest to disclose.

*Corresponding author: Prema Ramaswamy, MD, 4802, 10th Avenue, Maimonides Infants and Children's Hospital, Brooklyn, NY 11219. Tel: (718) 283-7501; Fax: (718) 635-7906.

E-mail: pramaswamy@maimonidesmed.org

1933-1711/$ - see front matter
http://dx.doi.org/10.1016/j.jash.2015.11.009

reviewed retrospectively. Inclusion criteria were: 1, cases where records documented a BP >95th percentile for age, sex, and height by a referring physician on at least three separate occasions and were in turn confirmed in the pediatric hypertension clinic by auscultation; 2, had echocardiography and ABPM for initial diagnostic assessment ≤3 months apart with no known secondary causes of hypertension; and 3, had no antihypertensive medications before or during the ABPM and LVMI measurements. This study was approved by the Institutional Board Review.

LVM was calculated from two-dimensional–guided M-mode echocardiographic measurements of the left ventricle. Measurements of the left ventricle internal dimension, interventricular septal thickness, and posterior wall thickness were made during diastole according to methods established by the American Society of Echocardiography.[11] Parameters were measured three times for each patient and the results averaged. All echocardiographic measurements were performed by one observer (MO), an echotechnologist with over 15 years of experience, who was blinded to the BP values and demographic information including the BMI of the patients. LVM was calculated using the Devereux equation.[12] LVMI was calculated by dividing LVM by height$^{2.7}$ which has been shown to minimize effects of age, gender, ethnicity, and overweight status.[13,14] LVH was defined as elevated LVMI for age and sex as defined by published norms in children.[15] The relative wall thickness was also calculated to define patients with concentric hypertrophy.[16]

ABPM was performed, and the data were analyzed by calculating average BP, BP load, and BP index for the entire 24-hour period, wake period, and sleep period as described in the AHA statements on ABP.[17,18] In addition, BP dipping was calculated by subtracting the average sleep BP from the average wake and dividing the sum by the average wake BP. Based on the recommendations of the AHA statement,[17,18] patients were classified into three groups: WCH, prehypertension (pre-HT), and hypertension (HT). Obesity was defined as a BMI of greater than 95% for age.

Data Analyses

Descriptive statistics are presented as percentages, means, and standard deviations (SDs). Bivariate analyses for differences between groups with continuous variables used *t* tests and analysis of variances for independent groups. Fisher's exact test was used for comparison of nominal data such as LVH between groups. Correlations between LVMI and continuous demographic and clinical variables were determined using Pearson correlation. Multiple regression analysis determined the strength of association between LVMI and multiple independent variables. IBM SPSS version 19 was used for analyses.

Results

In 69 children and adolescents, the average age was 14.8 (SD 3.3) years and they (75%) were male. Forty-six percent had WCH, 19% had pre-HT, and 35% had HT. Forty-five percent were Hispanic and 33% Caucasian. The average BMI z score was 1.6 (SD 0.96). There were no differences in mean age, gender, BMI, LVMI, or percentage of children with LVH between the groups (P = NS), although the BMI z score was higher in the WCH and pre-HT groups. Table 1 displays the patient demographics and clinical characteristics across hypertensive groups. The percentage of Hispanic patients was highest in the WCH group, but this failed to reach statistical significance; it is possible this was precluded by the small number of patients in each subgroup.

Comparisons of demographic and clinical data between patients with and without LVH are shown in Table 2. Patients with and without LVH did not differ in gender distribution or height. They also did not differ in clinic SBP or in any systolic parameter by ABPM. Clinic diastolic pressure, 24-hour diastolic pressure, and awake diastolic pressure were higher in the group with no LVH ($P = .003$, $<.03$, and $<.02$, respectively).

However, patients with LVH differed from those with no LVH by having significantly higher weight, BMI, and BMI z scores ($P < .0001$).

Over a third of all the subjects, 27 (39%) were obese, and of these, 15 (55.5%) had LVH compared with 42 nonobese subjects of whom only 7 (16.6%) had LVH ($P = .001$). Comparison of clinical information between obese and nonobese patients revealed a statistically significant increase in mean 24-hour diastolic pressure (71 vs. 67 mm Hg, $P = .01$) and mean diastolic load (25 vs. 17%, $P = .02$) in the nonobese group, but nondipping ($P = .01$) and LVMI ($P = .0001$) were higher in the obese.

We compared clinical parameters between patients with and without LVH within each group (Table 3). In both the pre-HT and the WCH, the only significant difference between patients with and without LVH was a significantly higher BMI in those with LVH ($P = .02$ and $P = .01$, respectively). The BMI in the HT group was higher in those who had LVH compared with those who did not, but this did not reach statistical significance, likely due to the small number of patients. There was no significant difference in the LVMI of the patients who had LVH among the three groups ($P = .42$). Within the subgroups, concentric hypertrophy was noted in 0/4 HT, 2/6 in pre-HT, and in 5/12 WCH (Table 4). The difference was not statistically significant, but this is possibly related to the small number of patients in the groups.

The association between echocardiographic and ABPM variables was determined by univariate and multivariate analyses. LVMI demonstrated the strongest bivariate correlation with BMI ($r = 0.563$, $P = .0001$) (Figure 1) among all demographic and clinical variables analyzed. LVMI did not