ORIGINAL ARTICLE

Hypertensive Adolescents: Correlation with Body Mass Index and Lipid and Glucose Profiles

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Abstract

Background: The occurrence of hypertension during adolescence correlates with metabolic changes, obesity, and overweight.

Objective: To correlate the lipid and glucose profiles of hypertensive and nonhypertensive adolescents with age, gender, body mass index (BMI), weight, and height.

Methods: We selected 53 hypertensive adolescents and 182 healthy, nonhypertensive adolescents. The adolescents were divided into three groups: group I (GI; n = 108, 58 males, mean age 15.2 ± 2.2 years), consisted of healthy, nonhypertensive adolescents of healthy parents without a diagnosis of hypertension, dyslipidemia, or diabetes; group II (GII, n = 53, 28 males, mean age 13.9 ± 1.4 years), consisted of adolescents with confirmed hypertension; and group III (GII; n = 74, 31 males, mean age 14.9 ± 2.2 years), consisted of healthy, nonhypertensive adolescents of parents with a diagnosis of hypertension, dyslipidemia, or diabetes.

Results: Gender and weight did not differ significantly among the groups. The subjects in GII were overall younger (around 1 year), shorter, and had a higher BMI compared with those in GI and GIII. After adjustment for age and BMI, GII presented higher glucose and LDL-C levels and lower HDL-C levels compared with GI and GIII. Total cholesterol and triglycerides levels showed no differences between groups. GI and GIII had no significant differences with regard to the analyzed variables.

Conclusion: Hypertensive adolescents showed higher values of BMI, and serum glucose and LDL-C levels, and lower serum HDL-C levels. These findings reveal that the changes in lipid profile and glucose metabolism that occur during adolescence may be influenced by the occurrence of hypertension during this developmental phase. (Int J Cardiovasc Sci. 2017;30(5):401-407)

Keywords: Adolescent; Hypertension; Dyslipidemias; Obesity; Body Mass Index; Overwehight.

Introduction

The continuously growing rates of obesity and overweight in children and adolescents has led to an increased prevalence of metabolic syndrome and hypertension in this population. ¹⁻³ Children with primary hypertension are usually overweight and obese, which complicates the dissociation of the effects of the blood pressure (BP) levels from those of the metabolic abnormalities. ⁴ The prevalence of hypertension increases progressively with increasing values of body

mass index (BMI), and both the aging process (with physiological and biochemical changes) and obesity are known to contribute to BP changes during adolescence. ^{5,6} Furthermore, hypertension in childhood and adolescence can lead to hypertension in adulthood.⁷

Guidelines for the diagnosis of high BP in children and adolescents provide reference BP levels based on gender, age, and height.⁵ Particularly during puberty, accelerated rates of BP changes may be associated with height and Tanner stage of pubertal development.⁸ Height and serum lipid levels are associated with the

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process of sexual maturation, and the levels of total cholesterol (TC) show a negative association with height.^{5,7} According to findings by Kouda et al.,⁷ height velocity is inversely associated with dynamic changes in serum lipids during puberty. These metabolic changes at puberty could reflect a potential cardiovascular risk in adulthood since a shorter stature is associated with a higher risk of development of coronary artery disease (CAD).⁹

Based on these observations, the objective of the present study was to assess and correlate the lipid and glucose profiles of hypertensive and nonhypertensive adolescents with these individuals' age, gender, BMI, weight, and height.

Methods

Patients

We selected 148 consecutive adolescents regularly attending a hypertension ambulatory of a Pediatric Nephrology Clinic at the public health care system due to a diagnosis of hypertension. All hypertensive subjects had an office systolic and/or diastolic BP equal to or above the 95th percentile for gender and height on three or more occasions (office hypertension). The diagnosis of hypertension was confirmed by 24-hour ambulatory blood pressure monitoring (ABPM), in which the presence of hypertension was defined as an average daytime and/or nighttime BP equal to or above the 95th percentile for gender and height according to ABPM pediatric standards.¹⁰ We excluded from the study subjects without confirmed hypertension and included those with essential or secondary hypertension. For the control group, 182 healthy adolescents (90 males, mean age 15.1 ± 2.2 years) were selected from the public healthcare unit.

Each adolescent had their height and weight measured at the time of their appointment. In regard to BP assessment, all measurements were obtained from non-sedated subjects in the supine position and at rest. Systolic and diastolic BP recordings were obtained from the right arm with appropriately sized cuffs, after a minimum of 30 minutes of rest. Three BP measurements were obtained using a standard manual mercury sphygmomanometer, and the average of the three readings was used in the analysis. Levels of BP were classified according to established guidelines:¹¹ normal (systolic < 120 mmHg; diastolic < 80 mmHg),

prehypertension (systolic 120-139 mmHg; diastolic 80-89 mmHg), or hypertension (systolic \geq 140 mmHg; diastolic \geq 90 mmHg). Participants who reported taking BP medications were considered as having hypertension regardless of their BP measurements.

The adolescents were considered overweight or obese when presenting BMI values equal to or above the 85th and 95th percentiles for age and gender, respectively. ¹²⁻¹⁴ The BMI was calculated using the standard formula. ^{12,15} Both parents and adolescents were asked to collect blood samples between 1 week before and 1 week after the appointment, for assessment of fasting serum glucose, TC, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), triglycerides (TG), and creatinine. The parents' data were only used to establish diagnoses of diabetes and dyslipidemia and were not further analyzed in the study.

In the hypertensive and control groups, the exclusion criteria were subjects without blood samples and adolescents with a diagnosis of diabetes, dyslipidemia, and chronic kidney disease. The presence of obesity or overweight was not considered an exclusion criterion. The following tests were additionally obtained from the subjects in the hypertensive group: microalbuminuria (abnormal values between 30 to 300 mg/g creatinine), 16 glomerular filtration rate (GFR; abnormal values below 75 mL/min/1.73 m²), 17 and basal insulin (normal values between 5 and 25 μ U/mL). 6

The adolescents were divided into three groups: group I (GI; n = 108, 58 males, mean age 15.2 \pm 2.2 years), comprising healthy, nonhypertensive adolescents born to healthy parents without a diagnosis of or taking any medication for hypertension, dyslipidemia, or diabetes; group II (GII; n = 53, 28 males, mean age 13.9 \pm 1.4 years), comprising adolescents with confirmed hypertension; and group III (GIII; n = 74, 31 males, mean age 14.9 \pm 2.2 years), comprising healthy, nonhypertensive adolescents whose parents a) were being treated for dyslipidemia or presented baseline levels of TC \geq 200 mg/dL, LDL-C > 130 mg/dL, HDL-C < 40 mg/dL, or TG \geq 150 mg/dL; b) had diabetes mellitus or a fasting plasma glucose \geq 100 mg/dL; and c) were taking medications for hypertension or presented a systolic BP \geq 140 mmHg or diastolic BP \geq 90 mmHg. ^{18,19}

The study was approved by the institution's Ethics Committee. The legal representative of each adolescent signed an informed consent form before the examination. All adolescents also signed a consent form.

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Blood sample analysis

Levels of fasting serum glucose, TC, HDL-C, LDL-C, TG, creatinine, GFR, and microalbuminuria were determined using standard techniques and assays at a central laboratory. Briefly, for measurements of TC, LDL-C, HDL-C, TG, and glucose values, approximately 5 mL of venipuncture blood were collected from each subject and placed in Vacutainer tubes after a 12- to 14-hour fast. The serum was separated from the red cells by centrifugation at 3,000 rpm for 10 minutes at 4°C up to 2 hours after the venipuncture procedure. The serum was then placed in microtubes and stored at -20°C for subsequent determination of lipid fractions and glucose levels. Serum levels of TC, HDL-C, TG, and glucose were determined by enzymatic methods (Roche Diagnostics), while LDL-C values were estimated by the Friedewald formula: LDL-C = TC - (HDL-C + TG / 5).¹³

Statistical analysis

The results of the quantitative variables are described as means and standard deviations. Frequencies and percentages for gender as a variable are presented, and the chi-square test was used to compare the groups. For age, weight, height, and BMI, the groups were compared using one-way analysis of variance (ANOVA). Lipid variables were compared with analysis of covariance (ANCOVA) including age, height, and BMI as covariables. The least significant difference (LSD) test was used for *post hoc* multiple comparisons. Values of p < 0.05 were considered statistically significant. The data were analyzed with the program IBM SPSS Statistics, v.20.

Results

A total of 95 hypertensive adolescents from the GII, who were younger than 12 years of age and lacked laboratory tests, were excluded from the study. Since the information regarding the Tanner pubertal stage was unavailable, the age of 11 years was used as a surrogate marker for the onset of puberty. A total of 53 hypertensive adolescents (28 males, mean age 13.9 ± 1.4 years) remained in the study. All these subjects were receiving antihypertensive therapy comprising angiotensin-converting enzyme inhibitors (ACEIs; n = 19), amlodipine (n = 5), propranolol (n = 1), or combinations of ACEI and amlodipine (n = 11), amlodipine and propranolol (n = 2), ACEI and propranolol (n = 1). None of the patients was using diuretics. In 19 adolescents

(35%), we identified potential causes of secondary hypertension including coarctation of the aorta, reflux nephropathy, ectopic kidney, polycystic kidney disease, chronic pyelonephritis, renal artery stenosis, solitary kidney, and renal atrophy.

Levels of creatinine (0.6 \pm 0.14 mg/dL), microalbuminuria (12.1 \pm 16.7 mg/g creatinine), and GFR (161 \pm 47 mL/min/1.73 m²) were normal in the hypertensive adolescents. There were no significant differences in gender and weight among the three groups. The subjects in GII were younger (maximum 1 year), shorter, and had higher BMI values compared with those in GI and GIII (Table 1). After adjustment for age and BMI values, the subjects in GII presented higher glucose and LDL-C levels and lower HDL-C levels when compared with those in GI and GIII (Table 2). Levels of TC and TG did not differ between groups. In regard to height, 25 adolescents (23%) in GI, 12 (22%) in GII, and 12 (16%) in GIII were considered short or very short for age. In regard to weight, 25 adolescents (22%) in GI, 28 (52%) in GII, and 16 (21%) in GIII were considered obese or overweight for age. When only the GII was analyzed, there was no significant difference in relation to age, height (p = 0.8), weight (p = 0.1), or levels of TC, LDL-C, HDL-C, TG, and glucose in adolescents with essential or secondary hypertension (data not shown). In both GI and GIII, there were no significant differences in regard to the analyzed variables.

Discussion

The present study revealed that hypertensive adolescents, regardless of the cause of hypertension and after adjustments for age and BMI, presented higher glucose and LDL-C values and lower HDL-C levels when compared with nonhypertensive adolescents. Additionally, among the nonhypertensive patients, even those born to parents with diagnosed dyslipidemia, hypertension, and diabetes, the values of glucose and lipid variables did not differ significantly. These findings suggest a correlation between hypertension and metabolic abnormalities during adolescence. The increased BMI in the group of hypertensive adolescents was mainly associated with height since weight did not differ significantly among the groups. Although the hypertensive group was somewhat younger (about 1 year in relation to the other groups), the values of height related to age in both males and females in this group were considered short or very short in only 22% of the cases. A similar finding was identified in GI and GIII,

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Table 1 - Basal characteristics of each group Variable N Mean Standard Deviation Group p value Age (years) Ι 108 15.2 П 53 13.9 1.4 *0.001 Ш 74 14.9 2.2 GI x GII **< 0.001 GI x GIII **0.392 GII x GIII **0.005 Weight (kg) Ι 108 58.3 15.7 ΙΙ 53 62.8 20.8 *0.093 74 Ш 56.3 15.0 Height (cm) Ι 108 1.65 0.11 ΙΙ 53 1.60 0.12 *0.024 III74 1.62 0.11 GI x GII **0.007 GI x GIII **0.147 GII x GIII **0.195 Ι 108 Body Mass Index (kg/m²) 21.2 4.3 ΙΙ *< 0.001 53 24.5 6.7 Ш 74 21.1 4.1 GI x GII **< 0.001 GI x GIII **0.844 GII x GIII **< 0.001

*One-way analysis of variance (ANOVA), p < 0.05. **Least significant difference (LSD), p < 0.05.

which comprised nonhypertensive adolescents. We were unable to rule out the possibility that this age difference of 1 year could have influenced the final height of the subjects in this group when compared with those in GI and GIII. Moreover, these findings corroborate those from other studies that have revealed an inverse relationship between height and lipid profile. ^{20,21}

Adolescents with a significant increase in height are likely to present decreased serum lipid levels, whereas those with a lower increase in height are likely to present increased serum lipid levels.²² A short height is also associated with other CAD risk factors such as hypertension, high LDL-C levels, and diabetes. A prospective study involving these adolescents would

be required to analyze their final height and correlate it to their lipid profile.

As far as diabetes is concerned, the present study also identified higher glucose levels in hypertensive adolescents. However, this association between metabolic and lipid profiles, which was not the objective of the present study, may also be genetically determined, as indicated by Nelson et al.²² Several changes occur during puberty, the developmental period of the population analyzed in this study. These changes include an increase in height, development of secondary sexual characteristics, and modifications in the cardiovascular system, which may lead to electrocardiographic and BP abnormalities.²³ It is known that BP and arterial

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| Table 2 – Lipids and glucose levels | | | | | |
|-------------------------------------|-------|-----|-------|--------------------|-----------|
| Variable | Group | N | Mean | Standard Deviation | p value |
| Glucose | I | 108 | 77.5 | 11.3 | |
| | II | 53 | 88.7 | 9.8 | *< 0.001 |
| | III | 74 | 78.8 | 9.8 | |
| GI x GII | | | | | **< 0.001 |
| GI x GIII | | | | | **0.409 |
| GII x GIII | | | | | **< 0.001 |
| LDL-C | I | 108 | 68.2 | 26.4 | |
| | II | 53 | 82.4 | 20.5 | *0.018 |
| | III | 74 | 72.9 | 28.3 | |
| GI x GII | | | | | **0.001 |
| GI x GIII | | | | | **0.240 |
| GII x GIII | | | | | **0.042 |
| HDL-C | I | 108 | 62.7 | 13.9 | |
| | II | 53 | 46.3 | 14.2 | *< 0.001 |
| | III | 74 | 61.3 | 13.5 | |
| GI x GII | | | | | **< 0.001 |
| GI x GIII | | | | | **0.507 |
| GII x GIII | | | | | **< 0.001 |
| Triglycerides | I | 108 | 79.6 | 37.8 | |
| | II | 52 | 99.7 | 63.2 | *0.814 |
| | III | 74 | 82.3 | 42.2 | |
| Total cholesterol | I | 108 | 146.8 | 26.2 | |
| | II | 53 | 148.0 | 23.9 | *0.643 |
| | III | 74 | 150.1 | 27.8 | |

*One-way analysis of variance (ANOVA), p < 0.05. **Least significant difference (LSD), p < 0.05. LDL-C: low-density lipoprotein cholesterol; HDL-C: high-density lipoprotein cholesterol.

stiffness increase with age and that an early hormonal control (hormone levels and expression of receptors) may simultaneously influence the growth of long bones and arteries, leading to further developmental changes in vascular structure and function, as well as changes in the susceptibility to artery stiffness and hypertension.^{24,25} These alterations may also influence the thickness of the media layer which, when increased, is considered a marker of atherosclerotic disease.²⁶⁻²⁷

Height, as a means to calculate BMI, is widely used to quantify the risk of CAD. Recent studies have questioned the direct or inverse association of BMI values with CAD severity, which would explain the obesity paradox,²⁸ considering that most studies consider only the final BMI value, instead of weight or height values alone. Additionally, results from meta-analysis have suggested that height may be regarded as an independent factor in the calculation

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of coronary risk.²⁸ Unfortunately, few studies have correlated the progression of height with the lipid profile, glucose levels, and hypertension in children and adolescents, as most seek to associate these variables with childhood and juvenile obesity.29-32 A study by Batide-Alanore et al.³³ including 865 families has confirmed that short height is associated with an adverse cardiovascular profile. Despite the small number of participants, the findings of this study, showing that hypertensive adolescents (with essential or secondary hypertension) were shorter and presented higher glucose and LDL-C serum levels and lower HDL-C serum levels, may contribute to the development of further prospective studies assessing the behavior of these variables at adult age and their correlation with the incidence of CAD. These findings show the correlation and complexity of the mechanisms involving hypertension in growing adolescents, who are constantly affected by metabolic changes.

Study limitations

The present study has some important limitations, including a small number of assessed individuals, lack of fasting insulin levels⁶ in all groups, younger age of subjects in GII (which may have influenced their shorter height in relation to the other groups), the inclusion of adolescents with primary and secondary hypertension, and the absence of Tanner stage information to determine the sexual development of the participants (considering that early puberty is associated with short height, lipid profile alterations, and increased cardiovascular risk).²⁰ In regard to essential and secondary hypertension, we only included in the present study those adolescents without chronic kidney disease, confirmed by levels of GFR, creatinine, and microalbuminuria, and chose adolescents with similar weight and height in both

groups. Also, we lacked data on the birth weight and abdominal circumference of the subjects, which could have added more information about these individuals.

Conclusion

Hypertensive adolescents had higher BMI values and increased serum glucose and LDL-C levels, and lower serum HDL-C levels. These findings reveal that the changes in lipid profile and glucose metabolism that occur during adolescence might be influenced by the presence of hypertension at this developmental phase.

Author contributions

Conception and design of the research: Baroncini LAV, Sylvestre LC, Giroldo ML, Précoma DB, Pecoits Filho R. Acquisition of data: Baroncini LAV, Sylvestre LC, Baroncini CV, Giroldo ML. Analysis and interpretation of the data: Baroncini LAV, Sylvestre LC, Baroncini CV, Giroldo ML, Précoma DB, Pecoits Filho R. Statistical analysis: Baroncini LAV, Sylvestre LC, Giroldo ML, Précoma DB, Pecoits Filho R. Writing of the manuscript: Baroncini LAV. Critical revision of the manuscript for intellectual content: Baroncini LAV, Pecoits Filho R.

Potential Conflict of Interest

No potential conflict of interest relevant to this article was reported.

Sources of Funding

There were no external funding sources for this study.

Study Association

This article is part of the thesis of PhD submitted by Liz Andréa Villela Baroncini, from PUC-Paraná.

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