Original Paper

The Frequency of Elevated Blood Pressure in Obese Minority Youth

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In this study, 167 obese persons were recruited (45 African Americans, 122 Caribbean Hispanic persons), with a mean age of 14.6±2.1 years, a mean body mass index (BMI) of 38±7.5 kg/m^2, and mean BMI Z-score of 2.47±0.36; 31 nonobese youth were recruited as controls (7 African Americans, 24 Caribbean Hispanic persons), with a mean age of 14.6±2.1 years, a mean BMI of 20±2.8 kg/m^2, and a mean BMI Z-score of –0.08±0.87. The objective was to assess the frequency of elevated blood pressure in obese minority youth. Weight, height, blood pressure (BP), and various biochemical markers were measured in each participant. Overall, 31% of the obese patients had elevated BP, compared with 3% of the control participants. Obese persons with elevated BP had significantly higher BMI, BMI Z-scores, and hemoglobin A_{1c} levels. The frequency of elevated BP and the degree of systolic BP elevation increased with increasing BMI Z-score. Elevated BP was 10 times more frequent in obese minority youth, emphasizing the importance of screening for hypertension in this high-risk population. (J Clin Hypertens (Greenwich). 2008;10:119–124) ©2008 Le Jacq

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Hypertension (HTN) remains a major risk factor for the development of many diseases including stroke, myocardial infarction, coronary heart disease, renal insufficiency, and decreased cognitive function.[1,2] Although stroke and myocardial infarction are rare in childhood, the origins of cardiovascular disease and HTN are present early in life; hypertensive end-organ damage may occur at a young age.[3–5] Higher blood pressure (BP) in childhood is also predictive of sustained HTN in adulthood.[6]

The prevalence of childhood obesity is increasing at a rapid rate. Data from 1999 through 2001 demonstrate a continuing increase in prevalence to 15.5% of 12- to 19-year-olds and 15.3% of 6- to 11-year-olds.[7] Diabetes and HTN, obesity-related chronic diseases that are usually associated with adulthood, are appearing more frequently in our obese youth. Both of these conditions are likely to be related to the metabolic syndrome (MS), a triad of insulin resistance, dyslipidemia, and elevated BP, which is common in obese youth. Recent studies indicate that the process of atherosclerosis starts at an early age and is linked to obesity and other components of MS in childhood.[5]

In this study, we assessed the frequency of elevated office BP in minority youth referred for the evaluation of obesity and its related complications, including MS, and correlated the incidence of elevated BP to various biochemical parameters associated with MS.

METHODS

Setting and Study Design

This study was part of a larger study designed to assess the frequency of abnormal glucose tolerance test results in obese minority youth. Patients who fulfilled the following criteria were recruited from the Pediatric Endocrine Clinics at the Children's Hospital at Montefiore in the Bronx, New York:

aged 10 to 18 years or in puberty, body mass index (BMI) >85th percentile, family history of type 2 diabetes mellitus (T2DM), African American or Caribbean Hispanic ethnicity, and signs of insulin resistance such as acanthosis nigricans. Control participants consisted of pubertal African American or Caribbean Hispanic persons aged 10 to 18 years with a BMI <85th percentile for age and sex who were recruited from the General Pediatric and Endocrine Clinics.

Written informed consent was obtained from the parent or guardian of each participant aged 13 to 18 years and also from the participants themselves. Assent was obtained from children aged 8 to 13 years. The protocol was approved by the Montefiore Medical Center Institutional Review Board.

Procedures

A detailed medical history was obtained, paying particular attention to smoking; alcohol consumption; birth weight; family history of diabetes, obesity, dyslipidemia, and HTN; and family history of cardiovascular disease (myocardial infarction, stroke, thromboembolic phenomena) in a first degree relative or grandparent. For female patients, information on menstrual history was also obtained. Each patient received a physical examination that included an assessment of the presence of acanthosis nigricans in the intertriginal areas, assessment of acne and hirsutism, height (using a Harpenden stadiometer) and weight, and pubertal staging using the method of Marshall and Tanner.[8] Waist and hip measurements, BMI (kg/m^2), and waist-to-hip ratio were calculated.

A seated BP measurement was obtained by a nurse once in the left arm of each participant, using the Agilent oscillometric device 3 (Agilent Technologies, Palo Alto, CA). Cuff sizes were chosen in accordance with the recommendations of the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents,[6] delineated by the National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents. Elevated BP was defined as a resting systolic and/or diastolic BP level ≥ the 95th percentile for sex, age, and height.[6]

A laboratory evaluation was performed in each patient, including a fasting lipid profile (total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglyceride levels), liver function profile, and blood urea nitrogen and creatinine assessment. An oral glucose tolerance test was performed in all patients after a 12-hour overnight fast. Blood samples for insulin and plasma glucose levels were obtained at 0 minutes and at 30, 60, 90, and 120 minutes after an oral dose of glucose 1.75 g/kg, with a maximum dose of 75 g. Homeostatic model assessment of insulin resistance (HOMA-IR)[9] and glucose-to-insulin ratios were calculated for all participants from glucose and insulin levels obtained in the fasting state. The diagnosis of diabetes and impaired glucose tolerance (IGT) was based on the criteria of the American Diabetes Association.[10] Normal glucose tolerance was defined as fasting plasma glucose <100 mg/dL and a 2-hour plasma glucose level <140 mg/dL, IGT was defined as a fasting plasma glucose level 100 to 126 mg or a 2-hour plasma glucose level of 140 to 199 mg/dL, and T2DM was defined as a fasting glucose level of ≥126 mg/dL or a 2-hour plasma glucose level of ≥200 mg/dL.[10]

Statistical Analysis

Descriptive statistics for continuous variables are presented as means and standard deviations or as medians and ranges as appropriate, and those for categorical variables as relative frequencies. In addition, the BMI Z-scores (the number of standard deviation units that a person's BMI is from the mean) were categorized into <2, 2 to 2.5, >2.5, and an analysis of variance was performed using a Duncan's multiple range test. Those independent variables with resulting P values <.2 from tests of association were included in the initial logistic regression model. A monitored backward stepwise procedure was used to derive the final model that included only variables significant at P<.05. Further, correlations among continuous variables were derived using Spearman's rank correlation coefficients. Analyses were performed using SAS version 9.1.2 (SAS Institute, Cary, NC).

RESULTS

167 obese patients and 31 nonobese controls were recruited. Demographic, anthropometric, and biochemical characteristics of the obese and lean participants are compared in Table I. Obese patients had significantly lower high-density lipoprotein cholesterol levels and significantly higher triglyceride levels. In addition, the obese patients had higher HOMA-IR values, indicating a greater degree of insulin resistance

BP levels of the obese and lean participants are displayed in Table II. Systolic BP and systolic BP index were significantly greater in the obese patients compared with the lean controls. Thirty-one percent of the obese patients (95% confidence interval,

Table I. Participant Characteristics

Parameter	Obese Participants	Control Participants	*P* Value
No.	167	31	
Age, y	14±2.3	14.6±2.1	NS
Sex, male/female	92 (55%)/75 (45%)	14 (45%)/17 (55%)	NS
Ethnic background, AA/CH	45 (27%)/122 (73%)	7 (23%)/24 (77%)	NS
BMI, kg/m^2	38±7.5	20±2.8	<.001
BMI Z-score	2.47±0.36	–0.08±0.87	<.001
Tanner stage, EP/LP	47 (28%)/12 (72%)	6 (19%)/25 (81%)	NS
Total cholesterol, mg/dL	165±32	161±24	NS
HDL-C, mg/dL	48±12	66±16	<.001
LDL-C, mg/dL	94±27	80±21	NS
TG, mg/dL	113±62	78±32	<.001
HbA_{1c}, %	5.5±0.4	5.4±0.3	NS
HOMA	6.4±5.6	2.1±1.2	<.001
IGT	21/167 (12.6%)	0/31 (0%)	.045

Values are presented as mean ± SD. Abbreviations: AA, African American; BMI, body mass index; CH, Caribbean Hispanic; EP, early pubertal (T2–T3); HbA_{1c}, hemoglobin A_{1c}; HDL-C, high-density lipoprotein cholesterol; HOMA, homeostatic model assessment; IGT, impaired glucose tolerance; LDL-C, low-density lipoprotein cholesterol; LP, late pubertal (T4–T5); TG, triglyceride.

0.241–0.382) had elevated BP, compared with only 3% (95% confidence interval, 0.008–0.208) of the lean control participants (*P*=.002).

We grouped all obese participants with increased systolic BP (n=46) or increased diastolic BP (n=7) and compared them with obese participants with normal BP (n=115). Obese patients with elevated BP were significantly heavier than those with normal BP, as indicated by higher BMI and BMI Z-scores, and had higher hemoglobin A_{1c} levels than those with normal BP levels (Table III). A progressive increase in the number of participants with elevated BP was noted with increasing BMI Z-scores. An increase in absolute systolic BP and systolic BP index was also noted with increasing BMI Z-scores (Table IV). When stratifying BP by BMI Z-score categories (mild obesity BMI Z-score, <2; moderate obesity BMI Z-score, 2–2.5; and severe obesity BMI Z-score, >2.5), there was a significant increase in BP with increasing levels of obesity (Table IV).

No significant differences were noted between obese subjects with elevated BP and those with normal BP with respect to age, family history of HTN, lipids, HOMA-IR, or rates of IGT (Table III). No significant differences in frequency of elevated BP was present with respect to sex or ethnicity; 37.8% of the African American participant had elevated BP, compared with 28.7% of the Caribbean Hispanic participants (*P*=NS). A similar frequency of elevated BP was noted between the early and late pubertal obese participants (31.9% of Tanner stage 2–3 vs 30.8% of Tanner stage 4–5; *P*=NS).

Table II. Blood Pressure in Obese and Control Participants

	Obese Participants (n=167)	Control Participants (n=31)	*P* Value
SBP, mm Hg	121±12.4	105±10	<.001
DBP, mm Hg	66±10	64±8	.33
SBP index[a]	0.95±0.10	0.83±0.09	<.001
DBP index[a]	0.80±0.12	0.80±0.10	.54
Elevated SBP	46 (28%)	1 (3%)	.002
Elevated DBP	7 (4%)	0 (0%)	NS

Values are presented as mean ± SD. [a]Blood pressure index = participant's blood pressure level divided by 95th percentile blood pressure level for participant's age, sex, and height. Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure.

DISCUSSION

In this cross-sectional study of BP in obese minority youth referred to a pediatric endocrine clinic for evaluation of obesity, we found that obese children had a significantly greater frequency of elevated BP than lean controls of similar ethnic backgrounds. We also observed that the magnitude of BP elevation rose as the severity of obesity increased. This finding is consistent with other studies that have demonstrated that increases in BMI in childhood have been correlated with increases in systolic BP in US children.[11,12]

Of particular concern is that the young early pubertal participants had a frequency of elevated BP similar to that of the late pubertal participants. In the Muscatine study,[13] BP measurements were obtained in 2445 participants aged 7 to 18 years

Table III. Characteristics of Obese Participants With and Without Elevated BP

	Elevated BP	Normal BP	*P* Value
No.	52	115	
Age, y	14±2.4	14±2.3	NS
Birth weight, kg	3.2±0.90	3.1±0.92	NS
Gestational age, wk	39±2.3	39±2.2	NS
BMI, kg/m^2	40±7.8	37±7.3	.04
BMI Z-score	2.57±0.33	2.43±0.03	.01
SBP index[a]	1.05±0.06	0.91±0.07	<.001
DBP index[a]	0.85±0.12	0.80±0.11	.002
Waist-to-hip ratio	0.85±0.26	0.85±0.23	NS
Total cholesterol, mg/dL	160±37	167±29	NS
HDL-C, mg/dL	46±11	49±12	.10
LDL-C mg/dL	92±31	96±24	.44
TG, mg/dL	117±64	111±61	.56
HbA_{1c}	5.62±0.49	5.42±0.39	.010
HOMA	6.3±4.3	6.7±6.1	NS
IGT	6/52 (12%)	15/115 (13%)	NS

Values are presented as mean ± SD. [a]Blood pressure (BP) index = participant's blood pressure level divided by 95th percentile blood pressure level for participant's age, sex, and height. Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; HbA_{1c}, hemoglobin A_{1c}; HDL-C, high-density lipoprotein cholesterol; HOMA, homeostatic model assessment; IGT, impaired glucose tolerance; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TG, triglyceride.

Table IV. Blood Pressure Levels of Obese Participants According to BMI Z-Score

	Z-Score <2	Z-Score 2–2.5	Z-Score >2.5	*P* Value
No.	56	61	101	–
SBP	108±12	119±10	124±14	<.001
DBP	65±10	66±9.0	67±10	.40
SBP index[a]	0.87±0.10	0.95±0.08	0.97±0.10	<.001
DBP index[a]	0.79±0.12	0.81±0.11	0.81±0.12	.63
No. with elevated BP	5/56 (9%)	18/61 (30%)	40/101 (40%)	<.001

Values represent mean ± SD. [a]Blood pressure index = participant's blood pressure level divided by 95th percentile blood pressure level for participants' age, sex, and height. Abbreviations: DBP, diastolic blood pressure; SBP, systolic blood pressure.

and again when the participants were aged between 20 and 30 years. Adult BP was shown to correlate with childhood BP and body size. It was shown in the Framingham Heart Study that both in the short and long term, patients with borderline isolated systolic HTN are at increased risk for progression to definite HTN and the development of cardiovascular disease.[14] A recent study by Sun and colleagues[15] involved longitudinal modeling of long-term serial data collected from childhood into adulthood in the Fels Longitudinal Study. This was used to link BP levels in childhood to HTN and MS in adulthood. They found that the relative risk of adult HTN for children with repeated measurements of elevated systolic BP was greater for 5- to 7-year-old children than for older children and adolescents.[15] Because BP in early childhood and adolescence usually tracks into adult life, it is necessary to target these high-risk individuals early in life.[16–18]

This ethnic population is at particular risk for the development of MS and therefore is an important cohort to study. The National Longitudinal Survey of Youth, conducted from 1986 to 1998 among children aged 4 to 12 years, showed the prevalence of overweight to be 21.5% among African Americans, 21.8% among Hispanic persons, and 12.3% among non-Hispanic white children.[19] In addition, several studies have shown increased insulin resistance in African American and Hispanic compared with Caucasian children,[20–28] and this difference was independent of adiposity.[28] Ethnic differences in lipids, lipoproteins, and BP are well documented in adults and begin early in childhood.[29–31] While HTN affects persons of all ethnic backgrounds, the rates have been found to be particularly high among African Americans.[32] We, however, found no difference in the frequency of elevated BP between the African

American and Caribbean Hispanic participants.

The link between obesity and elevated BP in children and adolescents has received increasing attention in the literature.[33–35] Results of the Bogalusa Heart Study demonstrated that overweight schoolchildren were at increased risk for adverse levels of several cardiovascular disease risk factors. These individuals were 4.5 times more likely to have elevated systolic BP and 2.4 times more likely to have elevated diastolic BP and had raised low-density lipoprotein cholesterol, low high-density lipoprotein cholesterol, raised triglyceride, and high fasting insulin concentrations.[33] In a school-based HTN and obesity screening study, Sorof and associates[34] reported a 3-times-greater prevalence of HTN in obese adolescents compared with nonobese adolescents. Primary HTN, once considered a rare diagnosis in pediatrics, has become more common.[35] Insulin levels have been shown to correlate with HTN at all body weights, but the association is stronger in obese hypertensive patients.[36] The degree of insulin resistance in adults has been demonstrated to correlate with the severity of HTN.[37] In the Insulin Resistance Atherosclerosis Study,[38] adult participants who were insulin sensitive had a reduced risk of HTN.

Our lean participants had significantly lower HOMA-IR levels and lower rates of IGT, indicating a greater degree of insulin sensitivity than in our obese patients. When examining obese patients with elevated BP and those with normal BP, however, there were no significant differences in HOMA-IR, indicating similar degrees of insulin resistance in these 2 groups.

Atherosclerosis develops silently during childhood, before complications such as myocardial infarction and stroke occur. Overweight and obesity are related to vascular thickening.[39] Increased carotid intimal thickening has been used as a marker of generalized atherosclerosis in adults that precedes clinical cardiovascular events. The Muscatine study demonstrated that total cholesterol in childhood was a significant independent risk factor for carotid intima-media thickening in both men and women.[33] The Bogalusa Heart Study revealed that an elevated low-density lipoprotein cholesterol level and elevated BMI during childhood were independent risk factors for increased carotid intima-media thickness in young adulthood.[40] Increased carotid intima-media thickness was not present among obese adults who had been normal weight as children, emphasizing the cumulative effects of childhood obesity persisting into adulthood.[41] A clear relationship exists between obesity and a number of risk factors including elevated BP, lipid and lipoprotein abnormalities, increasing prevalence of type II diabetes, and increasing left ventricular mass.[42]

The findings of this study are limited by the single automated BP reading, which was not validated with a mercury sphygmomanometer, and by the ethnic makeup of the study participants. Since only 1 BP assessment was obtained, we cannot say that these patients have HTN. Since even a single elevated BP reading in childhood is predictive of the development of HTN later in life,[16] however, these children are clearly at increased risk for the development of HTN. The population studied was of African American and Caribbean Hispanic descent, and thus no comments about other ethnic backgrounds can be made. These data emphasize the importance of careful evaluation and follow-up of BP in obese minority youth, a population at high risk for the development of cardiovascular disease.

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