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Albuminuria and masked hypertension in overweight and obese children and adolescents: A cross-sectional study

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ABSTRACT

Introduction. Childhood overweight and obesity are known risk factors linked to the development of masked hypertension (MH). These factors may have a greater impact on ambulatory blood pressure (BP) than on office BP and be associated with early target organ damage.

Objective. To evaluate the presence of markers of MH in an overweight and obese pediatric population.

Population and methods. An analytical, observational, cross-sectional study was performed. Overweight and obese children aged 5 to 16 years who consulted at two tertiary hospitals in the Autonomous City of Buenos Aires were included. Office blood pressure and ambulatory blood pressure monitoring (ABPM) were recorded. Clinical and anthropometric parameters were evaluated, as were the metabolic profile, renal function, and albuminuria.

Results. A total of 409 children were included, with a median age of 11.3 years; 236 (57.7%) were male. There were 301 (73.6%) normotensive children, 27 (6%) with true hypertension, 42 (10.2%) with white coat hypertension, and 39 (9.5%) with MH. Univariate and multivariate regression models, adjusted for age and sex, showed an association between ambulatory hypertension and office systolic BP (OR 1.1, p <0.001) and albuminuria (OR 3.37, p = 0.03). The presence of albuminuria was the only predictor of MH in obese individuals (OR 3.6, p <0.01).

Conclusion. A significant prevalence of MH was identified in overweight and obese pediatric populations. An association was observed between hypertension, including MH, and the presence of albuminuria in overweight and obese pediatric patients.

Keywords: *pediatrics; albuminuria; obesity; hypertension; undiagnosed diseases.*

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INTRODUCTION

Masked hypertension (MH), defined as normal blood pressure (BP) in the doctor's office but elevated out of office BP,[1-4] has an estimated prevalence of 9% to 16% in the pediatric population.[1-3] It is associated with early organ damage, as indicated by albuminuria.[3,5,6] Childhood overweight and obesity are known modifiable risk factors closely linked to the development of hypertension (HTN), particularly MH.[3] Obesity is associated with activation of the renin-angiotensin-aldosterone system (RAAS), sympathetic hyperactivity, and intravascular volume expansion, mechanisms that contribute to both the development of MH and glomerular damage. This increase in intraglomerular pressure leads to endothelial dysfunction and alterations in the filtration barrier, thereby increasing the risk of albuminuria as an early marker of subclinical renal damage.[2]

One way to diagnose MH is through 24-hour ambulatory BP monitoring (ABPM), which can also provide information on other BP patterns such as nocturnal hypertension.[1,2]

Currently, standardized studies of overweight and obese children and adolescents do not systematically include ABPM or early assessment of hypertension-mediated organ damage (HMOD).

Although there is sufficient evidence to recommend ABPM for detecting MH in overweight and obese patients,[6,7] the availability of ABPM is limited in many health centers. Other early indicators of MH in overweight and obese populations could help screen patients who require ABPM despite being normotensive in the doctor's office.

Identifying early predictors of MH in overweight and obese pediatric populations would allow for the timely detection of patients who need ABPM and optimize antihypertensive treatment, reversing HMOD and reducing future cardiovascular risk.

The objective of this study is to evaluate the presence of markers of MH in overweight and obese pediatric populations.

POPULATION AND METHODS

Analytical, observational, cross-sectional study. Boys and girls aged 5 to 16 years with overweight and obesity who attended the hypertension clinic of two tertiary hospitals in the Autonomous City of Buenos Aires, without associated underlying disease, from February 1, 2018, to December 31, 2023. Children with predisposing perinatal pathology (low birth weight, intrauterine growth restriction, and prematurity), congenital malformations, chronic kidney disease, congenital heart disease, malignant neoplasia or bone marrow transplant, evidence of elevated intracranial pressure, and those taking medications that increase BP and other systemic diseases associated with HTN, were excluded.

The sample size was calculated based on a 25% prevalence of albuminuria in patients with MH and a 5% prevalence in the group without MH, assuming a 10% prevalence of MH in obese children. A power of 80% and an alpha error of 5% were used to detect differences of 20%, resulting in a total of 380 patients, including 38 with MH.[2,3]

Overweight was defined as a body mass index (BMI) Z score between +1 and +2, and obesity as > +2 points according to sex and age, in accordance with the tables of the Sociedad Argentina de Pediatría.[8]

A complete electronic medical record was created for each patient with a detailed medical history. A thorough physical examination was performed, including a general inspection of the patient and the cardiovascular, respiratory, and abdominal systems. Weight was measured using a precision scale, calibrated every 6 months. Height was measured using a correctly positioned wall-mounted pedometer.

BP measurements were taken using the oscillometric method, with validated and calibrated equipment (OMRON MHM-739 and OMRON MHM-7120) and cuffs with bladders appropriate for the patient's build, using the correct technique. The first measurement was taken after 5 minutes of rest, and the next two were taken one minute apart. The first measurement was excluded, and the office BP was taken as the average of the second and third measurements from three consecutive visits.

All patients underwent ABPM with validated equipment (Spacelabs 90207 and 90217) using the appropriate cuff. The device was programmed to record BP every 20 minutes during waking hours and every 30 minutes during sleep.

Ambulatory hypertension (AH) was diagnosed when the average systolic and/or diastolic BP by ABPM was ≥ 95th percentile (Pc) for height and sex according to the corresponding tables.[9] White coat hypertension (WCH) was defined as office BP ≥ Pc 95 with normal ABPM, while MH was defined as normal office BP with AH. Finally, true HTN included those with HTN in the office and by ABPM.[10,11]

Routine clinical laboratory tests were performed to assess renal function and the associated metabolic profile, including dyslipidemia and insulin resistance. A 24-hour urine sample was collected and tested for albuminuria, defined as moderate (30-300 mg/24 h).[12] This was determined using the immunoturbidimetry analytical method.

Categorical variables were described as counts and percentages; continuous variables were expressed as median and interquartile range (IQR) or mean and standard deviation (SD), depending on whether they were normally or abnormally distributed, respectively. Group means were compared using the *t-test* or Mann-Whitney test, depending on the data distribution. The chi-square test was used to analyze qualitative data. A *p-value* <0.05 was considered statistically significant. Multiple imputations for the cholesterol variable with missing values were performed in SAS PROC MI, generating 20 imputed datasets using linear regression. Subsequently, the true HBP and MH groups were compared for continuous variables using linear models in each imputed set, and the results were combined using PROC MIANALYZE to obtain final estimates, standard errors, 95% confidence intervals, and *p-values*. Univariate and multivariate logistic regression models were performed. All variables associated with MH (p <0.10) and those reported in the literature were included in the model. We configured the models, including sociodemographic and biological variables, using stepwise manner to evaluate the contribution and changes associated with each one. Interaction was assessed using a likelihood ratio test associated with the model with and without interaction terms. Multicollinearity among variables was assessed using variance inflation factors (VIFs). The best model was selected, adjusted for age and sex.

Statistical models to predict ambulatory hypertension and MH were developed using SAS 9.4. The STROBE guidelines were used to report the findings of this study.[13]

All participants were asked for informed consent and/or assent, as appropriate. The study was approved by the Research Ethics Committees of the participating centers.

RESULTS

Initially, 430 patients were included. Those with abnormal renal Doppler (n = 3), hypothyroidism (n = 3), abnormal renal scintigraphy (n = 3), and OSAS (n = 12) were excluded. A total of 409 patients were included in the final analysis, with a median age of 11.36 years (IQR 9.2-14.1), 236 males (57.7%).

Table 1 presents the characteristics of the entire population and its stratification by MH and true HTN.

When combining office and ambulatory BP values, we found 301 (73.6%) normotensive (NT) individuals, 27 (6%) with true HTN, 42 (10.2%) with white coat hypertension, and 39 (9.5%) with MH. Therefore, the normotensive group according to ABPM includes those with normotension and WCH (n = 343); 16.1% (n = 66) had AH, including those with true HTN and MH.

Of the total number of patients, 6 (1.5%) had isolated nocturnal hypertension and were included in the analysis in the MH group.

The median BMI was 28.36, and 162 (39.6%) patients had a Z score of +3.

The metabolic panel showed a mean ± SD of total cholesterol 221.6 ± 16.7 mg/mL,

Table 1. Characteristics of the sample

Variable	Total sample n = 409	NT by ABPM n = 343	MH n = 39	*p* NT vs. MH
Age (years, IQR)	11.3 (9.2-14.1)	11.6 (9.3-14.1)	11.1 (8.9-13.4)	0.37
Male gender n (%)	236 (57.7)	200 (58.3)	20 (51.3)	0.4
Z score +3 n (%)	162 (39.6)	138 (40.2)	17 (43.6)	0.9
SBP (mmHg) (mean, SD)	111.6 ± 14.1	110.2 ± 13.6	110.7 ± 1.7	0.82
DBP (mmHg) (mean, SD)	65.9 ± 8.5	65.4 ± 8.4	65.5 ± 7.7	0.97
Urinary Na >8 g/d n (%)*	42 (17.1)	30 (15.4)	6 (22.2)	0.51
Microalbuminuria n (%)*	17 (6.9)	12 (6.1)	5 (18.5)	0.02

**Urinary Na and microalbuminuria: n = 246.*
ABPM: ambulatory blood pressure monitoring; NT: normotensive; MH: masked hypertension; SBP: systolic blood pressure; DBP: diastolic blood pressure; IQR: interquartile range.

HDL 32.3 ± 4.8 mg/mL, and triglycerides 198 ± 69.3 mg/dL. The homeostasis model assessment (HOMA) value, as an estimator of insulin resistance, was 6.05 ± 3.2.

Among children with MH, 5 were identified with albuminuria, representing a prevalence of 12.8% (95% CI 4.8-38.6).

Univariate and multivariate regression models, adjusted for age and sex, showed that office systolic BP (OR 1.1, $p < 0.001$) and albuminuria (OR 3.37, $p = 0.03$) were independent predictors of AH (*Table 2*). HOMA, total cholesterol, and BMI were evaluated as potential confounding variables, with no significant associations observed.

The presence of albuminuria was the only predictor of MH in obese patients, increasing the risk more than threefold (OR 3.6, $p < 0.01$).

DISCUSSION

In this study, a prevalence of MH of 9.5% was observed in overweight and obese children and adolescents. The observed prevalence of MH showed values comparable to those reported worldwide, which range between 9% and 16% in overweight and obese children.[14,15] Locally, one study reported a prevalence of MH of 9.1% in children with risk factors, of whom 9 out of 10 were obese.[14]

Lurbe et al. demonstrated significantly higher systolic blood pressure (SBP) and diastolic blood pressure (DBP) values in the office in patients with MH.[14] Lurbe et al., and So H. et al. also showed that a higher level of obesity is associated with higher BP during the day and night over 24 hours, and a higher prevalence of AH.[16,17]

In our study, the average office SBP values were higher and were associated with AH in overweight and obese children. Thus, for every 1 mmHg increase above the expected SBP for height and sex, patients have an approximately 10% higher risk of developing AH. Therefore, in the presence of elevated SBP values in the office, the use of ABPM is recommended for the diagnosis of AH.[3,4,15]

Albuminuria is used to identify early stages of kidney damage. A prevalence of microalbuminuria of 10% is reported in overweight and obese children, strongly related to insulin resistance.[18]

Khalili *et al.* compared the prevalence of microalbuminuria in 100 obese children with 100 children of normal weight. They found a prevalence of 8.5% in the former group, with no significant differences between those aged 12 years or older and those aged 12 years or younger, or between the sexes.[19] In our study, we found a prevalence of albuminuria of 6.9% in the population studied. About renal consequences in pediatric patients with MH, there are virtually no data on early hypertensive renal damage, specifically with albuminuria.[3]

Various studies have identified possible pathways of kidney damage.[20,21] Obesity is a state of chronic inflammation that causes the release of proinflammatory cytokines. These activate the sympathetic nervous system and the RAAS, promoting water and sodium retention, with elevated blood pressure and glomerular damage. In turn, angiotensin II produces direct toxicity on podocytes. Additionally, excess adipose tissue stimulates the sympathetic nervous system through the direct effects of glucose and insulin on the central nervous system. In addition to the central effects of insulin and leptin, persistent sympathetic activation leads to a chronic increase in peripheral vascular resistance, mediated by alpha receptors, which also affects the renal system. Added to all of the above is the mechanism of hyperinsulinemia and insulin resistance that generate glomerular hyperfiltration (GHF), thereby increasing the risk of renal injury with increased albumin excretion.[20-22]

The presence of GHF could alter the interpretation of Pc in ABPM. GHF, which is

Table 2. Predictors of ambulatory hypertension

Variable	Univariate OR (95% CI)	Multivariate OR (95% CI)	*P*
Microalbuminuria	3.26 (1.2-8.9)	3.37 (1.13-10.1)	0.03
SBP in the office	1.06 (1.04-1.08)	1.1 (1.05-1.14)	<0.001
DBP in the office	1.06 (1.03–1.09)	0.97 (0.92-1.03)	0.36

Model adjusted for age and sex.
SBP: systolic blood pressure; DBP: diastolic blood pressure; OR: odds ratio; CI: confidence interval.

common in obese children, is associated with increased cardiac output and intravascular volume, which can raise BP regardless of true vascular dysfunction. Given that ABPM reference Pc values are based on children without renal impairment or obesity, the presence of GHF could bias BP classification, especially in the diagnosis of masked hypertension.[23]

The study suggests the need for systematic screening for MH in patients with albuminuria using ABPM. It emphasizes the importance of thorough evaluation of target organ damage in overweight/obese pediatric patients, especially when ABPM is not routinely available, to ensure timely referral.

A limitation of our study is its cross-sectional design, which prevents us from establishing direct causality. Thus, it is likely that MH contributes to glomerular barotrauma, which is already initiated by obesity, and is a precipitating factor for microalbuminuria. However, the converse cannot be ruled out, as albuminuria may be a marker of endothelial dysfunction and precipitate the onset of MH, as proposed.[20]

It is also important to clarify that, although they incorporated a significant number of patients, the analytical subgroups are limited. This limitation reduces the statistical power to infer robust associations. Therefore, there is a clear need for future research with larger samples to validate the findings and explore the relationship between albuminuria and MH in greater depth. It should also be noted that albuminuria was assessed by a single measurement, without confirmation in repeated samples, which could introduce bias and limit the accuracy of patient classification.

The fact that patients were recruited from tertiary care centers is a limitation that prevents generalization to the entire pediatric population and may represent selection bias; therefore, these data should be corroborated in primary care centers.

Nevertheless, our work represents a notable contribution to the study of MH in pediatrics, due to the large number of patients evaluated and the use of ABPM in a pediatric population, with systematic evaluation of microalbuminuria and other metabolic markers.

CONCLUSION

The study identified a prevalence of 9.5% of MH in the overweight and obese pediatric population. Predictors of HTN were office SBP and albuminuria. The presence of albuminuria was associated with MH in overweight and obese children. ■

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