Assessing Utility of 24-Hour Ambulatory Blood Pressure Monitoring to Distinguish Pediatric Populations Presenting with Elevated Blood Pressure in Rhode Island

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ABSTRACT

This retrospective study aimed to assess the value of 24-hour ambulatory blood pressure monitoring (ABPM) in distinguishing primary from secondary hypertension in pediatric patients. Our study was conducted on 293 patients referred to a pediatric nephrology clinic over 11 years. Various ABPM parameters were analyzed, including daytime and nighttime systolic and diastolic blood pressures, heart rate, and blood pressure load. Among the participants, 74% were normotensive (white-coat hypertension), 21.5% had primary hypertension, and 4.4% had secondary hypertension. There were no significant differences in the analyzed variables between primary and secondary hypertension groups. Our findings suggest that ABPM might not reliably differentiate between the two in this cohort. As white-coat hypertension becomes more prevalent, ABPM remains a valuable tool in preventing unnecessary workups in children without sustained hypertension. However, our study did not identify specific endpoints for distinguishing primary from secondary hypertension.

KEYWORDS: pediatric hypertension, ambulatory blood pressure monitoring, white-coat hypertension

INTRODUCTION

Elevated blood pressure is a prominent reason why children are referred to a pediatric nephrology clinic. It is the responsibility of the nephrologist to determine whether the patient has sustained hypertension versus white-coat hypertension, and whether the hypertension is essential or secondary in nature. Aside from the importance of diagnosing secondary hypertension and the associated primary etiology, identifying essential hypertension in children is crucial because of its relationship to adult hypertension. Multiple studies have shown that hypertension in childhood predisposes patients to hypertension in adulthood,[1,2] which is increasingly relevant as hypertension in the U.S. pediatric population is increasingly prevalent today than it was two or three decades ago.[3]

Hypertension can be essential in origin or result from secondary etiologies such as renal parenchymal disease, renovascular disease, endocrinopathies, cardiac disease (specifically aortic coarctation), medications or toxins. Secondary causes of hypertension have been shown to be prevalent in the pediatric population. A Polish study of 636 children with sustained hypertension showed that 55% had a secondary etiology.[4] Renal parenchymal disease was responsible for 68% of these cases of secondary hypertension, followed by renovascular and endocrine disorders. A U.S. cohort of 132 children with persistent hypertension from 1987 to 1991 showed a 77% prevalence of secondary causes.[5] Several factors have been identified to help predict which children have secondary hypertension. Secondary etiologies are more likely in children who are prepubertal, of normal weight, present with stage two hypertension, and with a negative family history for hypertension.[6,7] Unfortunately, these predictors are not highly sensitive and seldom preclude the need for an extensive workup to evaluate for secondary causes.

Along with primary and secondary hypertension, patients presenting to pediatric nephrology clinic with elevated blood pressure at their primary care office may be diagnosed as having white-coat hypertension. Identifying white-coat hypertension before an unnecessary workup is initiated is an immensely beneficial role of ambulatory blood pressure monitoring (ABPM). ABPM may serve a role in predicting or excluding patients with secondary hypertension if useful and statistically significant endpoints can be identified. Our study seeks to identify specific endpoints of pediatric ABPM data that can be used to distinguish primary hypertension from secondary hypertension. Moreover, it serves to characterize the evolving demographic and clinical features of a representative cohort referred to pediatric nephrology clinics for evaluation of hypertension.

MATERIALS AND METHODS

Study Population

Patients who underwent ABPM through the Pediatric Nephrology Clinic at Hasbro Children's Hospital between April 1999 and September 2010 were considered for inclusion. ABPM studies were excluded from consideration if any of the following criteria applied: 1) fewer than 50% of attempted readings were valid; 2) no height was available to permit determination of blood pressure percentiles with the exception of patients ≥18 years of age, in which case adult

Figure 1. Required Evaluation for Secondary Hypertension in All Hypertensive Patients

To qualify for inclusion, all hypertensive patients required:
1) At least 3 of 5 of the following (first-line workup as recommended by the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents):
History and physical examination in the Pediatric Nephrology Clinic
BUN, creatinine, and electrolytes
Urinalysis +/- urine culture
CBC
Renal ultrasound
AND/OR
2) At least one of the following (workup for specific causes of secondary hypertension, as clinically indicated):
Urine and/or serum toxicology screen
Polysomnography
Plasma renin and/or aldosterone levels
Plasma and/or urine catecholamines / metanephrines
Plasma and/or urine steroid (including cortisol) levels
Renovascular imaging (including MRA, CTA, renal scintigraphy, and/or conventional arteriography)
Thyroid function panel
Renal biopsy

Table 1. Demographic and Baseline Clinical Data on Patients Meeting Inclusion Criteria

	Normotensive Group	Primary (1°) Hypertensive Group	Secondary (2°) Hypertensive Group	Overall	p-value (1° vs 2°)
Number of patients	217 (74%)	63 (22%)	13 (4%)	293 (100%)	
Gender (# of males)	155 (71%)	34 (54%)	9 (69%)	198 (68%)	
Age (years)	13.8 ± 3.5	14.2 ± 3.3	12.2 ± 4.6	13.8 ± 3.5	0.17
Race (Caucasian)	127 (59%)	45 (71%)	12 (92%)	184 (63%)	
Race (Hispanic)	60 (28%)	13 (21%)	1 (8%)	74 (25%)	
Race (Black)	22 (10%)	5 (8%)	0 (0%)	27 (9%)	
Race (Asian)	3 (1%)	0 (0%)	0 (0%)	3 (1%)	
Race (Indian)	2 (1%)	0 (0%)	0 (0%)	2 (1%)	
Race (Unknown)	3 (1%)	0 (0%)	0 (0%)	3 (1%)	
Height (cm)	162.4 ± 17.9	160.3 ± 14.8	153.4 ± 20.7	161.6 ± 17.5	0.16
Weight (kg)	73.1 ± 26.2	80.8 ± 32.6	64.3 ± 31.6	74.4 ± 28.1	0.07
BMI (kg/m²)	26.8 ± 6.7	30.3 ± 8.9	25.8 ± 9.0	27.5 ± 7.5	
Normal weight (%)	66 (30%)	15 (24%)	6 (46%)	87 (30%)	
Overweight (%)	44 (20%)	9 (14%)	0 (0%)	53 (18%)	
Obese (%)	107 (49%)	39 (62%)	7 (54%)	153 (52%)	
Urine sodium (mEq/L)	162.8 ± 60.9	178.8 ± 67.8	108.5 ± 81.3	166.4 ± 63.3	0.17
Urine creatinine (mg/dL)	166.6 ± 82.5	156 ± 83.4	125.2 ± 85.7	162 ± 83.1	0.18
Urine microalbumin (mg/g Cr)	18.7 ± 45.6	19.6 ± 28.9	77.0 ± 176.3	21.2 ± 53.9	0.18

interpretive criteria were used; 3) the patient was receiving one or more antihypertensive medications at the time of ABPM; 4) the patient had a previous, valid ABPM study during the trial period.

Measures were taken to ensure that all hypertensive patients had undergone clinically appropriate workup to identify potential causes of secondary hypertension. This workup, as directed by the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents,[7] is delineated in **Figure 1**.

Baseline Data

Demographic and clinical parameters were recorded on all patients (**Table 1**). These included age, gender, ethnicity, height, weight, body mass index (BMI), date of ABPM, and, when available, urine sodium, creatinine and microalbumin.

Performance and Interpretation of ABPM

ABPM was performed using a validated oscillometric cuff, with reports generated by Rozinn Electronics, Model RZ250. The monitor was calibrated to record daytime readings (from 6:00 AM to 10:00 PM) every 20–30 minutes, and nighttime readings (from 10:00 PM to 6:00 AM) every 30–60 minutes. An automated report was generated for each ABPM study, with summarized data including daytime, nighttime, and overall systolic and diastolic BP (average + SD), daytime, nighttime, and overall pulse (average + SD), and the total number of readings attempted and obtained. For all patients between 1–17 years of age, 90th, 95th, and 99th percentiles for SBP and DBP were obtained using standardized tables from the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents.[7] Hypertension was defined as a mean ambulatory SBP and/or DBP at or above the 95th percentile. For patients who were ≥18 years of age, ambulatory hypertension was defined in accordance with JNC-7 criteria:[8] overall ambulatory BP ≥130/80, daytime ambulatory BP ≥135/85, and/or nighttime ambulatory BP ≥120/75. All ABPM studies were reviewed by a pediatric nephrologist to calculate the percentage of systolic and diastolic BP readings at or above the 90th, 95th, and 99th percentiles (BP load).

Determination of Hypertension Class

All subjects were referred to the Pediatric Nephrology Clinic due to elevated office or hospital BP readings. Those who did not meet criteria for hypertension by ABPM were classified as having white-coat hypertension. In patients confirmed to have hypertension by ABPM, comprehensive chart review was performed to determine adequacy of workup for secondary hypertension (**Figure 1**). Etiology of ABPM-confirmed hypertension was determined to be primary or secondary based on objective laboratory and radiologic data, and clinical judgment of the pediatric nephrologist.

Statistical Methods

All demographic data were collected when ABPM was performed. Height percentile for age was determined using CDC clinical growth charts (published in 2000). Continuous variables are presented as mean ± SD; categorical variables are presented as numbers and percentages. Statistical analyses were performed via SPSS. P values of <0.05 were used to determine statistical significance.

RESULTS

Over an 11-year period, 345 patients were considered for inclusion; 52 were excluded from analysis. Of the 293 remaining patients, 217 (74%) were found to be normotensive by ABPM (conferring white-coat hypertension diagnosis). Among the 76 patients (26%) who were hypertensive by ABPM, 63 had primary hypertension and 13 had secondary hypertension. Children with secondary hypertension appeared to be younger with lower height, weight, and BMI, compared to their counterparts with primary hypertension (see **Table 1**); however, none of these differences met the threshold for statistical significance.

ABPM procedures yielded an average of 45 valid readings per patient, with 88% of all attempted readings being valid. Mean BP readings by ABPM were 117/65 in the normotensive group, 135/75 in the primary hypertension group, and 136/79 in the secondary hypertension group. Peak BP readings averaged 151/91, 172/103, and 180/102, respectively. Peak SBP and DBP readings were significantly higher in both hypertensive groups compared to the normotensive group but were not significantly different between the primary and secondary hypertension groups (**Table 2**).

All categories of BP load exhibited a similar pattern, with significantly higher BP load in the hypertensive groups compared to the normotensive group. The average percentage of BP readings at or above the 95th percentile was 29% in the normotensive group, 74% in the primary hypertension group, and 78% in the secondary hypertension group. No significant differences in any of the BP load categories were identified between the primary and secondary hypertension group (**Table 3**).

The study population included a substantial proportion of non-dippers, with a mean SBP dip of 9.3% across the cohort. The average magnitude of nocturnal dipping in SBP was 9.5% in the normotensive group, 8.9% in the primary hypertension group, and 7.7% in the secondary hypertension group. Nocturnal dipping in DBP averaged 14.7%, 12.3%, and 11.4%, respectively. These figures were not statistically significant between the groups (**Table 4**).

Review of the heart rate data from the ABPM readouts revealed significant trends between groups of hypertension class and weight class. Mean heart rate was higher in both hypertensive groups (86 for primary hypertension, 89 for secondary hypertension) compared to the normotensive group (77), though not significantly different between both hypertensive groups. There was no difference in nocturnal HR dipping when comparing the primary and secondary hypertensive groups. Of the 293 children included in the analysis, 87 (30%) were of normal weight, 53 (18%) were overweight, and 153 (52%) were obese. Average BMI was 27.5 kg/m2. BMI exceeded 40 kg/m2 in 18 subjects. The standard deviation in heart rate during the 24-hour ABPM was significantly lower in overweight ($p = 0.007$) and obese ($p < 0.0001$) children than in their normal-weight counterparts.

Table 2. Comparing Peak Systolic and Diastolic BP in Primary versus Secondary Hypertensive Patients

Variables	Normotensive Group	Primary (1˚) Hypertensive Group	Secondary (2˚) Hypertensive Group	p-value (1˚ vs 2˚)
Peak SBP	151 ± 14	172 ± 17	180 ± 33	0.93
Peak DBP	91 ± 10	103 ± 16	102 ± 16	0.68

Table 3. Comparing BP Load in Primary versus Secondary Hypertensive Patients

Variables	Normotensive Group	Primary (1˚) Hypertensive Group	Secondary (2˚) Hypertensive Group	p-value (1˚ vs 2˚)
BP Load at 90th %ile	40 ± 19%	81 ± 12%	84 ± 15%	0.77
BP Load at 95th %ile	29 ± 17%	74 ± 13%	78 ± 20%	0.78
BP Load at 99th %ile	13 ± 10%	56 ± 19%	64 ± 24%	0.62
Systolic BP Load at 95th %ile	26 ± 16%	72 ± 13%	77 ± 21%	0.72
Diastolic BP Load at 95th %ile	9 ± 10%	32 ± 28%	42 ± 28%	0.56

Table 4. Comparing Nocturnal Systolic and Diastolic BP Dips in Primary vs Secondary Hypertensive Patients

Variables	Normotensive Group	Primary (1˚) Hypertensive Group	Secondary (2˚) Hypertensive Group	p-value (1˚ vs 2˚)
Nocturnal Systolic BP Dip (%)	9.5 ± 6.8%	8.9 ± 6.9%	7.7 ± 6.2%	1.00
Nocturnal Diastolic BP Dip (%)	14.7 ± 9.1%	12.3 ± 7.9%	11.4 ± 8.5%	1.00

DISCUSSION

This retrospective cohort study examined a population referred to an urban pediatric nephrology clinic for evaluation of hypertension and sought to identify categories of ABPM data that could discern causes of primary hypertension from secondary hypertension. Determining such ABPM criteria might prove beneficial in selecting which children warrant an extensive workup for secondary hypertension. As hypertension in U.S. children is more prevalent today than 20–30 years ago this has become increasingly important. The National Health and Nutrition Examination Survey (NHANES) provided information about the changing prevalence of hypertension in the pediatric population. A study that compared NHANES data from the years 1988–2004 to the years 1999–2008, the prevalence of elevated blood pressure (pre-hypertension or hypertension) increased from 15.8% to 19.2% in males and from 8.2% to 12.6% in females.[3] This increase in population-based pediatric blood pressures is closely related to the rising prevalence of obesity in the U.S. population. Most recent data, from 2011–2012, show that in children 2–19, 14.9% are overweight and 16.9% are obese.[9] A study of 1665 children 8–17 showed that the incidence of high or borderline high BP in normal weight children was 8.4%, whereas for overweight and obese children was 12.8% and 18.0%, respectively.[10] Another study of 5102 children showed that being overweight conferred a relative risk of 3.26 (95% CI 2.50–4.24) for hypertension.[11] Other pediatric studies[12,13,14] have documented significant correlations between BMI and risk of prehypertension or hypertension. In our study, the incidence of primary hypertension was as follows: 17% of patients with normal BMI, 17% of patients with overweight BMI, and 36% of patients with obese BMI. An obese BMI conferred a significant increased risk of primary hypertension.

Prior pediatric ABPM studies[15,16,17] identified significant (but disparate) findings in children with secondary compared to primary hypertension. One retrospective analysis examined ABPM data in 145 children with untreated hypertension (69% of which had secondary hypertension).[15] Results showed that in children with secondary hypertension, overnight non-dipping status (defined as 10% decrease in blood pressure at night) was found in 65% for systolic BP and 21% for diastolic BP, compared to 11% and 0%, respectively, for children with primary hypertension. This study also showed a greater likelihood of sustained nocturnal hypertension in the secondary hypertension group. Another series analyzed 97 ABPM reports obtained from 85 patients seen at the pediatric hypertension clinic. The presence of a daytime diastolic BP load ≥ 25% and/or a nighttime systolic BP load of ≥ 50% on ABPM were determined to be highly specific for secondary hypertension.[16] Lastly, a group reviewed ABPM reports from 76 children, of which 16 were subsequently diagnosed with white-coat hypertension, 50 with primary hypertension and 10 with secondary hypertension.[17] Daytime and nocturnal systolic and diastolic BP values were greater among children with secondary hypertension than in those with primary hypertension.

Compared to prior analyses regarding markers for secondary hypertension in ABPM, our cohort data did not corroborate these findings, and found no categories of ABPM data that were significantly different between the primary hypertension and the secondary hypertension group. Our study was limited by a small sample size of hypertensive patients, as our analysis of 293 ABPM procedures yielded 63 children with primary hypertension and 13 with secondary hypertension. This could explain our inability to find statistically significant differences in ABPM parameters between groups, especially if the differences sought were relatively subtle. Secondary hypertension is a heterogeneous diagnosis with a variety of cardiac, renal, endocrine, and iatrogenic causes and, as such, a central unifying diurnal blood pressure pattern for "secondary hypertension" may not exist but may instead depend upon the specific mechanism which is responsible. Many different causes of secondary hypertension were represented in our study population (**Table 5**). Furthermore, there was significant overlap between the primary hypertension group and secondary hypertension group in all categories of absolute blood pressure values, BP load, and nocturnal dipping.

Our study highlighted several characteristics of a pediatric hypertension cohort that were markedly disparate from figures previously published in the medical literature. The prevalence of white-coat hypertension was 74%. Only 26% of children referred from their primary care providers for suspected hypertension had confirmed hypertension based on ABPM results. The reported frequency of white-coat hypertension from prior studies in a pediatric population

Table 5. Etiology of Secondary Hypertension

Etiology of Secondary Hypertension	Number of Patients, n
Total Patients with Secondary Hypertension	13
Renal Parenchymal Disease	6
Chronic kidney disease (obstructive uropathy)	1
Wegener's granulomatosis	1
Post-infectious glomerulonephritis	1
Dystrophic right kidney	1
Dysplastic right kidney	1
Renal scarring of unilateral kidney	1
Renovascular Disease	1
Thyroid Disturbances	2
Subclinical hypothyroidism	1
Hyperthyroidism	1
Hyperaldosteronism	2
Obstructive Sleep Apnea	1

has ranged from 35–45%.[17,18,19] A study done between 2005–2006 showed that 46% patients that initially presented with elevated blood pressure had white-coat hypertension based on ABPM.[18] This same study showed the cost-effectiveness of using ABPM; it estimated that for every 1000 patients $2.4M can be saved by using ABPM to identify patients with white-coat hypertension who need not further hypertensive workup. If the increased prevalence of white-coat hypertension seen in our study is applicable on a broader scale, it implies substantial utility of ABPM in avoiding unnecessary treatment in children without sustained hypertension.

Conversely, the prevalence of secondary hypertension in our cohort was much lower than anticipated. Only 13 (4%) of all children evaluated, and 13 children from the 76 (17%) with ABPM-confirmed hypertension, had an identifiable secondary etiology. As our study was retrospective, there was no protocol in place guaranteeing an exhaustive workup for secondary causes; instead, a workup for pertinent secondary causes was undertaken at the discretion of the pediatric nephrologist. Criteria were established (as detailed in **Figure 1**) to identify a minimally appropriate workup for secondary hypertension, with 15 hypertensive children excluded from analysis due to insufficient workup. This may have led to cases of secondary hypertension having been overlooked, leading to a falsely low prevalence in the study population. Despite this, it remains difficult to reconcile the low frequency of secondary hypertension seen in our study with the figures of 55% and 77% obtained in pediatric cohorts from the 1980s and early 1990s.[4,5]

Our results regarding the low prevalence of secondary hypertension in our study, may, in fact, highlight that essential hypertension is an increasingly common phenomenon, due to the upsurge in obesity and metabolic syndrome in the pediatric population. NHANES data from 2007–2008[20] yielded a prevalence of overweight BMI or obesity of 35.5% in school-aged children (6–11 years), and 34.2% in adolescents (12–19 years). In contrast, children being evaluated in our office had a strikingly high prevalence of overweight (18%) and obesity (52%), with only 30% of all subjects being of normal weight. As previously mentioned, several large pediatric studies have documented strong associations between overweight/obesity and the risk of prehypertension or frank hypertension.[11,12,13,14] Sedentary lifestyles have also been frequently implicated in childhood obesity.[21] This may provide an explanation for the differential ABPM mean heart rate data that we observed between classes of weight and hypertension. Mean heart rate (by ABPM) was significantly higher in both hypertensive groups compared to their normotensive counterparts, which could reflect deconditioning or increased sympathetic activity. It was also interesting to find decreased heart rate variability in the overweight and obese groups, which we postulate is due to a lower level of physical activity over the 24-hour measurement period.

Importantly, in 2017 the American Academy of Pediatrics published Clinical Practice Guideline for Screening and Management of High Blood Pressure in Children and Adolescents,[22] as an update to the 2004 Fourth Report on the Diagnosis, Evaluation and Treatment of High Blood Pressure in Children and Adolescents. Significant changes in the updated guidelines included new normative pediatric BP tables based on normal weight-weight children, a simplified screening table for identifying BPs needing further evaluation, a simplified BP classification in adolescents ≥ 13 years of age, more limited recommendation to perform screening BP measurements only at preventative care visits, and an expanded role for ABPM in the diagnosis and management of pediatric hypertension. For children ages 1–13 yr. normal BP is <90th percentile, elevated BP ≥ 90th percentile to <95th percentile or 120/80 mm Hg to <95th percentile (whichever is lower), stage 1 hypertension is ≥ 95th percentile to < 95th percentile + 12 mm Hg, or 130/80 to 139/89 mm Hg (whichever is lower), and stage 2 hypertension is ≥ 95th percentile + 12 mm Hg, or ≥ 140/90 mm Hg (whichever is lower). For children ages ≥ 13 yr normal BP is <120/<80 mm Hg, elevated BP is 120/80 t0 129/80 mm Hg, stage 1 hypertension 130/80 to 139/89 mm Hg, and stage 2 hypertension is ≥ 140/90 mm Hg.

This study had several limitations which hindered its ability to find significant differences in ABPM variables between children with primary hypertension and children with secondary hypertension. These included the unexpectedly low prevalence of secondary hypertension (leading to a small sample size), the retrospective nature of the investigation, and the lack of a predefined protocol for identifying secondary etiologies of hypertension. Our study also carried unique advantages compared to prior pediatric ABPM research, such as the omission of children who were already on anti-hypertensive therapy (thereby avoiding any iatrogenic alterations in blood pressure and heart rate trends), and the near-universal application of ABPM on children referred to our pediatric nephrology clinic for evaluation of hypertension. Further prospective studies with larger sample sizes are needed to better clarify whether ABPM can be reliably used as a predictive tool of secondary hypertension. Nonetheless, the paradigm of hypertension in the pediatric population may be shifting. The previously observed prominence of secondary hypertension as a primary etiology is now being overshadowed by the rising incidence of essential hypertension, likely due to obesity and other metabolic risk factors. The notably high prevalence of white-coat hypertension in our study also reinforces the value of ABPM to further evaluate office hypertension in children.

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Acknowledgment

Atena Asiaii for developing the initial study database.

Disclosures

Authors disclosed no financial or conflicts of interest.

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