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Original Article

Hypertension in children with congenital adrenal hyperplasia: Prevalence and associated factors

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ABSTRACT

Objective: Congenital adrenal hyperplasia (CAH) is a group of inherited disorders of adrenal steroidogenesis that may predispose affected patients to hypertension through both disease- and treatment-related mechanisms. Although hypertension represents an important comorbidity in CAH, its prevalence and contributing factors in the pediatric population, particularly across different CAH subtypes, remain incompletely defined. The aim of this study was to evaluate the prevalence and determinants of hypertension in patients with CAH.

Material and Methods: This retrospective, single-center study included 143 children and adolescents aged 1–21 years with a confirmed diagnosis of CAH who were followed at a tertiary pediatric center. Blood pressure measurements obtained during routine outpatient visits were evaluated according to age-, gender-, and height-specific pediatric reference values. Patients were classified by CAH subtype, and demographic, clinical, and treatment-related characteristics were compared between patients with and without hypertension. Correlations between blood pressure levels and clinical parameters were analyzed.

Results: A total of 143 patients with CAH (female-to-male ratio, 1.38) were included, with a median follow-up duration of 111.33 months (IQR; 144.70). Hypertension was diagnosed in 15 patients (10.5%). The prevalence of hypertension differed significantly according to CAH subtype ($p<0.001$), with the highest rate observed in patients with 11β-hydroxylase deficiency (88.8%), followed by those with 21-hydroxylase deficiency (8.4%). No cases of hypertension were observed in other CAH subtypes. Patients with hypertension received significantly higher hydrocortisone doses compared with normotensive patients ($p=0.012$), while fludrocortisone dose did not differ between groups. Blood pressure levels showed strong correlations with age at last visit and moderate correlations with follow-up duration and body mass index. Hypertensive cardiomyopathy was detected in 40% of hypertensive patients and was more frequent in those with 11β-hydroxylase deficiency.

Conclusion: Hypertension is a relevant clinical finding in children with CAH and is closely associated with disease subtype, particularly 11β-hydroxylase deficiency. Long-term follow-up, regular blood pressure monitoring and careful evaluation of glucocorticoid exposure may be important for cardiovascular risk assessment in this population.

Keywords: Congenital adrenal hyperplasia, hypertension, 11-beta-hydroxylase deficiency, 21-hydroxylase deficiency, glucocorticoids

Introduction

Congenital adrenal hyperplasia (CAH) comprises a group of autosomal recessive disorders characterized by complex hormonal imbalances resulting from deficiencies of enzymes involved in adrenal steroidogenesis, leading to impaired cortisol synthesis with variable disturbances in mineralocorticoid and androgen production (1). The clinical spectrum of CAH is heterogeneous and depends on the underlying enzymatic defect, with 21-hydroxylase deficiency being the most common subtype, followed by rarer forms such as 11β-hydroxylase, 3β-hydroxysteroid dehydrogenase, and 17α-hydroxylase deficiencies (2-4). The 21-Hydroxylase is a key enzyme in adrenal steroidogenesis,

and the severity of CAH due to its deficiency is determined by the level of residual enzyme activity (2). CAH due to 11beta-hydroxylase deficiency is characterized by androgen excess and hypertension and is autosomal recessively inherited (5). One of the most common clinical features of 11 beta-hydroxylase deficiency is hyporeninemic hypokalemic hypertension (4). Advances in hormone replacement therapy and neonatal screening have markedly improved survival in patients with CAH; however, long-term disease and treatment-related complications, particularly those affecting cardiovascular health, remain a major clinical concern (1,6).

Hypertension is one of the major contributors to the increased cardiovascular disease risk in patients with CAH (6,7). Previous studies have reported that 20–66% of children with CAH exhibit elevated systolic or diastolic blood pressure (8, 9). Moreover, 24-hour ambulatory blood pressure monitoring studies have demonstrated an absence of normal nocturnal dipping in blood pressure in many patients with CAH (10,11). Its pathophysiology is multifactorial and may include excess mineralocorticoid activity, accumulation of steroid precursors with mineralocorticoid properties, chronic glucocorticoid overtreatment, and altered regulation of the renin–angiotensin–aldosterone system (12). Certain CAH subtypes, particularly 11β-hydroxylase deficiency, are classically associated with a higher risk of hypertension due to the accumulation of deoxycorticosterone, whereas hypertension in patients with 21-hydroxylase deficiency is more often related to treatment-related factors (4,9).

Data on the prevalence and determinants of hypertension in pediatric CAH remain limited, and the relative contributions of disease duration, CAH subtype, and cumulative steroid exposure to blood pressure regulation are not fully understood. Therefore, the aim of this study was to evaluate the prevalence of hypertension in patients with CAH, to examine its distribution across CAH subtypes, and to investigate clinical and treatment-related factors associated with hypertension.

Materials and Methods

This retrospective, single-center study was conducted in the Pediatric Nephrology and Endocrinology Department of Ankara Child Health Hematology-Oncology Training and Research Hospital, and Ankara Bilkent City Hospital and the data were collected from the five-year period preceding June 2020. Patients with a confirmed diagnosis of CAH who were under regular follow-up during this period were eligible for inclusion. A total of 143 children and adolescents aged 1 to 21 years with diagnosis of CAH were included in the study. Clinical and laboratory data were obtained retrospectively from the hospital electronic medical records. Patients with incomplete medical records were excluded. Demographic and clinical data recorded included sex, date of birth, age at diagnosis of CAH, age at last visit, follow-up duration with CAH, height, and weight measurements, and BMI (kg/m^2) values were recorded. Systolic and diastolic blood pressure measurements were obtained using an oscillometric device by trained healthcare personnel, with an appropriately sized cuff and the child in a seated position after at least 5 minutes of rest. At each routine outpatient visit, blood pressure was measured at least two to three times, and the average of the last two readings was used for analysis. Hypertension was defined as systolic and/or diastolic blood pressure values ≥95th percentile for age, sex, and height, based on normative pediatric blood pressure reference values, documented on at least three separate clinic visits (13). For patients diagnosed with hypertension, age at hypertension diagnosis, interval between CAH diagnosis and hypertension diagnosis, follow-up duration with hypertension and medication data of anti-hypertensives were documented. In these patients, echocardiographic findings were also recorded to assess hypertensive cardiomyopathy. Hypertensive cardiomyopathy was assessed by transthoracic echocardiography during routine clinical follow-up. Left ventricular hypertrophy was identified based on left ventricular mass index, calculated using standard methods and interpreted according to age- and sex-adjusted reference values applied in clinical practice. Additionally, in patients with hypertension, daily hydrocortisone (mg/m^2/day) and fludrocortisone (mg/day) doses at the time of hypertension diagnosis were recorded, whereas in normotensive patients, doses of these medications at the last follow-up visit were documented.

Patients were classified according to CAH etiology based on clinical, biochemical, and genetic data when available. CAH subtypes included 21-hydroxylase deficiency, 11β-hydroxylase deficiency, 3β-hydroxysteroid dehydrogenase deficiency, 17α-hydroxylase deficiency, and unknown etiology.

Statistical analysis

All statistical analyses were performed using BM Statistical Package for the Social Sciences, version 22.0 (SPSS Inc., Armonk, NY, IBM Corp., USA). The normality of continuous variables was assessed using the Kolmogorov–Smirnov test. Normally distributed variables were expressed as mean and standard deviation (SD), while non-normally distributed variables were presented as median and interquartile range (IQR). Qualitative variables were presented as numbers and percentages (%).

Comparisons between patients with and without hypertension were performed using the Student's t-test for normally distributed variables and the Mann–Whitney U test for non-normally distributed variables. Categorical variables were compared using the chi-square test with exact methods (Fisher's exact test, Monte Carlo simulation) applied when appropriate.

Correlations between systolic and diastolic blood pressure levels and clinical parameters were evaluated using Spearman's correlation analysis. Correlation strength was interpreted as negligible (r=0.00–0.10), weak (r=0.10–0.39), moderate (r=0.40–0.69), strong (r=0.70–0.89), or very strong (r=0.90–1.00) (14). Statistical significance was set at $p<0.050$.

Results

A total of 143 patients with CAH (female-to-male ratio, 1.38) were included in the study, with a median follow-up duration of 111.33 months (IQR;144.70). During follow-up, 15 patients (10.5%) were diagnosed with hypertension. Baseline demographic and clinical characteristics of patients with and without hypertension are summarized in Table I. Age at diagnosis of CAH and age at last visit did not differ significantly between patients with hypertension and without hypertension. Follow-up duration with CAH tended to be longer in patients with hypertension; however, this difference did not reach statistical significance. Gender distribution and BMI were similar between groups. Patients with hypertension received significantly higher hydrocortisone doses than patients without hypertension (p=0.012), whereas fludrocortisone dose did not differ between the two groups (Table I).

Among patients with hypertension, the mean age at hypertension diagnosis was 11.15±6.45 years, with a median of 12.54 years (IQR; 9.18). The interval between CAH diagnosis and hypertension diagnosis was 10.50±6.40 years (median [IQR]; 11.92 years [9.53]). The median follow-up duration after hypertension diagnosis was 12.56 months (IQR; 43.80) (Table I). All hypertensive patients were receiving hydrocortisone therapy, whereas only six of the 15 (40%) patients were treated with fludrocortisone.

The prevalence of hypertension according to CAH subtype is shown in Table II. Hypertension was most frequently observed in patients with 11β-hydroxylase deficiency, followed by those with 21-hydroxylase deficiency. No cases of hypertension were observed in patients with 3β-hydroxysteroid dehydrogenase deficiency, 17α-hydroxylase deficiency, or unknown etiology. A significant association was observed

Table I: Demographic and clinical results of CAH with and without HT groups

Variable	Total patients*	Patients with HT*	Patients without HT*	p
Total number of patients	143	15	128	-
Age at diagnosis of CAH (years)*	2.54±4.44/0.08 (4.06)	0.65±1.39/0.06 (0.22)	2.76±4.62/0.08 (5.26)	0.523†
Age at last visit (years)*	11.84±6.16/13.49 (11.31)	13.04±6.28/15.90 (6.61)	11.70±6.15/13.20 (11.6)	0.414†
Follow-up duration with CAH (month)*	111.33±77.71/105.13 (144.70)	148.67±75.90/173.33 (101.87)	106.95±77.03/89.53 (137.68)	0.060†
Age at diagnosis of HT (years)*	-	11.15±6.45/12.54 (9.18)	-	-
Interval between CAH and hypertension diagnosis (years)*	-	10.50±6.40/11.92 (9.53)	-	-
Follow-up duration with HT (month)*	-	22.65±23.85/12.56 (43.80)	-	-
Gender (n) (female/male)	83/60	7/8	76/52	0.345‡
BMI (kg/m²)*	21.82±5.82/20.95 (7.44)	22.98±6.03/23.04 (10.15)	21.69±5.80/20.88 (7.35)	0.418‖
Systolic blood pressure (mmHg)*	108.54±15.87/106.0 (25.0)	136.80±16.26/140.0 (15.0)	105.23±12.13/105.0 (20.75)	>0.001†
Diastolic blood pressure (mmHg)*	65.0±12.29/64.0 (13.0)	88.13±14.39/90.0 (15.0)	62.29±8.69/63.0 (12.0)	>0.001†
Dose of hydrocortisone (mg/m²/day)*	14.62±4.80/14.20 (5.90)	17.90±4.67/17.30 (6.60)	14.33±4.74/14.0 (5.90)	0.012‖
Dose of fludrocortisone (mg/day)*	0.10±0.07/0.10 (0.05)	0.15±0.15/0.07 (0.28)	0.10±0.06/0.10 (0.05)	0.984†

:** *mean±SD/median (IQR),* **†:** *Mann-Whitney U test,* **‡:** *Chi-Square test,* **‖:** *Student's t-test,* ***CAH: *congenital adrenal hyperplasia,* ***HT:*** *hypertension,* ***BMI:*** *body mass index*

Table II: Rate of HT according to type of CAH

CAH type	Total patients	Patients with HT	Patients without HT	p†
Total number of patients	143	15	128	-
21-hydroxylase deficiency*	83 (58.0)	7 (8.4)	76 (91.6)	<0.001
11β-hydroxylase deficiency*	9 (5.6)	8 (88.8)	1 (1.1)	
3β-hydroxysteroid dehydrogenase deficiency*	2 (1.4)	0	2	-
17α-hydroxylase deficiency*	1 (0.7)	0	1	-
Unknown*	48 (34.3)	0	48	-

:** *n(%),* **†:** *Fisher's exact test,* ***CAH: *congenital adrenal hyperplasia,* ***HT:*** *hypertension*

Table III: Correlations between systolic and diastolic blood pressure levels and the clinical parameters

Clinical parameters	Systolic blood pressure (mmHg)		Diastolic blood pressure (mmHg)	
	r*	p	r*	p
Age at diagnosis of CAH (years)	0.19	0.017	0.22	0.006
Age at last visit (years)	0.83	<0.001	0.82	<0.001
Follow-up duration with CAH (month)	0.65	<0.001	0.64	<0.001
BMI (kg/m^2)	0.56	<0.001	0.56	<0.001
Dose of hydrocortisone (mg/m^2/day)	0.39	<0.001	0.34	<0.001
Dose of fludrocortisone (mg/day)	-0.20	0.085	-0.18	0.104

:** *Spearman correlation,* ***CAH: *congenital adrenal hyperplasia,* ***BMI:*** *body mass index*

between CAH subtype, particularly 21- and 11-hydroxylase deficiencies, and the presence of hypertension (p<0.001). There was no significant difference in the age at diagnosis of hypertension between patients with 21-hydroxylase and 11β-hydroxylase deficiencies (p= 0.298).

Systolic and diastolic blood pressure levels were weakly correlated with age at CAH diagnosis but showed strong positive correlation with age at last visit. Moderate correlations were observed with follow-up duration and BMI, while hydrocortisone dose showed weak positive correlations with blood pressure levels. No significant association was found between fludrocortisone dose and blood pressure (Table III).

In hypertensive patients, seven patients (46.7%) were treated with amlodipine alone, three patients (20.0%) received combination therapy with amlodipine and spironolactone, one patient (6.7%) was treated with amlodipine and enalapril, and four patients (26.6%) were not on antihypertensive treatment. Among patients with hypertension, hypertensive cardiomyopathy was observed in six patients (40%); however, no cases of proteinuria or hypertensive retinopathy were detected. Hypertensive cardiomyopathy was significantly more frequent in patients with 11β-hydroxylase deficiency compared with those with 21-hydroxylase deficiency (50% vs. 28.5%; p<0.001).

Discussion

In this retrospective cohort study, we evaluated the prevalence of hypertension in children with CAH and found that 10.4% of the cohort had hypertension. By comparison, hypertension affects approximately 2–5% of the general pediatric population, a rate lower than that observed in our cohort of children with CAH (8,15). Previous studies have reported elevated systolic and/or diastolic blood pressure in 20–66% of patients with CAH (8). In a study by Bonfig et al. (16), the prevalence of hypertension among patients with classic CAH was 12.5%, which is comparable to the prevalence observed in our cohort.

In recent study, hypertension showed a strong association with CAH subtype, with the highest prevalence observed in patients with 11β-hydroxylase deficiency, followed by those with 21-hydroxylase deficiency, while no cases were identified in the remaining subtypes. Comparative studies evaluating hypertension prevalence among different CAH subtypes remain limited. In the literature, the prevalence of hypertension has been reported to be approximately 9.1-12.5% in patients with 21-hydroxylase deficiency, whereas it reaches 50–59% in those with 11β-hydroxylase deficiency (18-20). In our study, the higher prevalence observed in 11β-hydroxylase deficiency is not unexpected when considering the underlying pathophysiology (16,17). Excess accumulation of mineralocorticoid-active steroid precursors in this subtype provides a plausible mechanistic explanation (18).

Additionally, the significantly higher frequency of hypertensive cardiomyopathy observed in patients with 11β-hydroxylase deficiency in our cohort further underscores the potential impact of prolonged mineralocorticoid excess and cumulative blood pressure burden. However, the absolute number of patients with 11β-hydroxylase deficiency in our cohort was small, and therefore subtype-specific prevalence estimates and cardiovascular outcomes, including hypertensive cardiomyopathy, should be interpreted with caution. In light of these findings, the significant association between CAH subtype and hypertension status and related complications highlights the importance of etiological classification when assessing cardiovascular risk in patients with CAH. Maccabee-Ryaboy et al. (8) performed a comparative analysis across CAH subtypes and reported early-onset hypertension, often before five years of age, among boys with salt-wasting CAH. In contrast, in our cohort, no significant differences were observed between patients with and without hypertension in terms of age or gender.

Beyond CAH subtype, treatment-related factors appear to play a critical role in blood pressure regulation. In this study, patients with hypertension received significantly higher hydrocortisone doses compared with normotensive patients, and hydrocortisone dose showed weak but significant correlations with blood pressure levels. Although patients with hypertension in our cohort received significantly higher hydrocortisone doses, this finding should be interpreted cautiously. Given the retrospective design of the study, a causal relationship between hydrocortisone dose and hypertension cannot be established. Higher glucocorticoid doses may reflect greater disease severity,

difficulties in achieving adequate hormonal control, or clinician-driven dose escalation in response to androgen excess rather than glucocorticoid overtreatment alone. Therefore, hydrocortisone dose in this context is more likely to represent a marker of cumulative disease and treatment burden than a direct determinant of hypertension. In contrast, fludrocortisone dose was not associated with hypertension or blood pressure levels in our cohort. Patients with classic 21-hydroxylase deficiency require lifelong glucocorticoid replacement therapy, with hydrocortisone being the most commonly used agent during childhood. Because impaired conversion of progesterone to deoxycorticosterone leads to aldosterone deficiency and salt wasting in many patients, mineralocorticoid replacement with fludrocortisone is frequently required, and salt supplementation is often recommended in early life (21).

Our finding supports the concept that chronic or supraphysiological glucocorticoid exposure may contribute to blood pressure elevation through multiple mechanisms, including increased vascular tone, enhanced sodium retention, adverse effects on body composition, and alterations in metabolic profile. Over time, these effects may lead to cumulative cardiovascular burden, particularly in patients requiring higher glucocorticoid doses for disease control. Despite appropriate consideration of age, sex, height, and weight, substantial interindividual variability in hydrocortisone dose requirements persists among patients with CAH, making it challenging to anticipate hydrocortisone-related side effects (21).

In contrast, fludrocortisone dose was not associated with blood pressure levels in our cohort. Supporting this finding, a study including 24 infants with CAH treated with fludrocortisone reported no elevation in blood pressure during the first year of life (22). Although mineralocorticoid therapy has been proposed to contribute to the development of hypertension in CAH, the lack of an observed association in our study may be related to the narrow dosing range maintained in clinical practice. Additionally, the limited number of patients receiving fludrocortisone and the heterogeneity of CAH subtypes within our cohort may have reduced our ability to detect a dose–response relationship.

In infants with CAH, higher fludrocortisone dose requirements and variable mineralocorticoid sensitivity have been suggested as potential risk factors for fludrocortisone-related hypertension (23). Bonfig et al. (17) reported the highest prevalence of hypertension among CAH patients receiving both glucocorticoid and fludrocortisone therapy in those aged 12–24 months. Considering the findings this study, the absence of a relationship between fludrocortisone dose and blood pressure in our cohort may be partly attributable to the relatively older age at hypertension diagnosis. Similarly, Lawrence et al. (21) reported a low regression coefficient in blood pressure prediction models, suggesting that a 100 µg increase in fludrocortisone dose was associated with only a modest (~1 mmHg) increase in systolic and diastolic blood pressure. This modest effect size is consistent with our findings and may further explain the lack of a significant association between fludrocortisone dose and blood pressure in our cohort.

In our study, the median age at hypertension diagnosis was 12.5 years, and the strong correlation between blood pressure levels and age at last visit is consistent with expected age-related increases in blood pressure during childhood and adolescence. This finding suggests that associations between blood pressure and other variables, such as hydrocortisone dose and follow-up duration, may be partially influenced by age and pubertal maturation. Because hydrocortisone exposure and disease duration inherently increase over time, these relationships are likely to reflect cumulative treatment and disease burden rather than age-independent effects. Given the relatively small number of hypertensive patients in our cohort, particularly across CAH subtypes, age-adjusted or partial correlation analyses were not performed. Future studies incorporating pubertal staging and larger sample sizes are warranted to better disentangle the independent contributions of age, treatment exposure, and disease duration to blood pressure regulation in CAH. In addition, blood pressure levels showed moderate correlations with follow-up duration and body mass index, further supporting a cumulative effect of long-term disease burden and treatment exposure. These findings suggest that hypertension in CAH may evolve as a chronic complication over time rather than being determined solely by early disease characteristics. The weak correlations observed with age at CAH diagnosis further support the notion that long-term disease burden, treatment exposure, and growth-related changes may be more relevant determinants of hypertension in this population.

Target organ involvement was assessed in hypertensive patients, with hypertensive cardiomyopathy identified in 40%, whereas no cases of proteinuria or hypertensive retinopathy were detected. This finding highlights the potential for early cardiovascular remodeling and underscores the importance of timely blood pressure monitoring in patients with CAH.

Limitations

Several limitations of this study should be acknowledged. The retrospective design and single-center setting may limit generalizability. Blood pressure classification was based on office measurements obtained during routine visits rather than ambulatory blood pressure monitoring, which precluded the assessment of white-coat and masked hypertension. Plasma renin levels were not available for analysis. In addition, the relatively small number of hypertensive patients limited the ability to perform more extensive multivariable analyses. Nevertheless, the strengths of this study include a well-characterized cohort with long-term follow-up and a detailed evaluation of CAH subtypes and treatment-related variables.

Conclusion

Hypertension is a clinically relevant finding in children with CAH. In this study, hypertension was closely associated with CAH subtype—particularly 11β-hydroxylase deficiency—and appeared to be influenced by long-term disease burden and glucocorticoid exposure rather than mineralocorticoid dose alone. The presence of early cardiac involvement in a subset of hypertensive patients highlights the importance of regular blood pressure assessment during follow-up. These findings support the value of etiological classification and individualized treatment strategies when evaluating cardiovascular risk in patients with CAH.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Ankara Child Health Hematology-Oncology Training and Research Hospital Ethics Committee (11.06.2019, reference number: 2019-180).

Contribution of the authors

Study conception and design: Mİ, GB; data collection: BA ,ZA; analysis and interpretation of results: Mİ, SAU, FŞÇ; draft manuscript preparation: Mİ, USB. All authors reviewed the results and approved the final version of the article.

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Conflict of interest

The authors declare that there is no conflict of interest.

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