The Clinical and Biochemical Markers of Congenital Adrenal Hyperplasia in Basra City

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Abstract*—Congenital adrenal hyperplasia (CAH) comprises a group of inherited enzymatic defects affecting adrenal steroidogenesis, with 21-hydroxylase deficiency being the most common subtype. This study explored the clinical variability, biochemical markers, and diagnostic challenges of CAH in Basra Governorate. A retrospective cross-sectional record-based study was conducted at the Al-Faiha Specialized Diabetes, Endocrine, and Metabolism Center in Basra, Iraq. Medical records from 2010 to 2025 were reviewed for 240 patients, of whom 160 met the diagnostic criteria for CAH. Data were analyzed using SPSS version 26, and descriptive and comparative statistics were applied to assess demographic patterns, clinical presentations, hormonal markers (17-OHP, ACTH, cortisol, and DHEA), and growth outcomes. Females constituted 74.4% of the cohort. Atypical genitalia (72.9% in females) and precocious puberty (37.1% in males) were the most common presentations in classic CAH. In contrast, non-classic CAH cases were more likely to present later with hirsutism (55%) and menstrual irregularities. Hormonal profiling revealed significantly elevated 17-OHP and ACTH levels in classic cases ($p < 0.001$ and $p = 0.018$, respectively). Karyotyping identified discordance between genetic and phenotypic sex in several cases, contributing to variability in gender assignment. Short stature affected 25.0% of patients, and bone age advancement was most pronounced in males with classic CAH. Overall, the clinical and biochemical profile of CAH in this Basra cohort mirrors global patterns but is influenced by delayed diagnosis, limited neonatal screening, and sociocultural factors. Classic CAH typically presents early with more severe manifestations, whereas non-classic CAH is likely underdiagnosed. Early hormonal and genetic testing, together with appropriate psychological support, is essential for optimal management.

Keywords*—Congenital Adrenal Hyperplasia (CAH), 21-Hydroxylase Deficiency, 17-Hydroxyprogesterone (17-OHP), Pediatric Endocrinology.

INTRODUCTION

Congenital adrenal hyperplasia (CAH) is a group of autosomal recessive disorders caused by genetic mutations that impair adrenal steroidogenesis and lead to defective cortisol biosynthesis (1). The most common etiology is 21-hydroxylase deficiency, which accounts for approximately 95% of cases (2). Disruption of the steroidogenic pathway results in cortisol deficiency and excess androgen production, producing virilization and a range of metabolic complications. CAH is broadly classified into classic and non-classic forms according to the severity of enzyme deficiency (3). Classic CAH includes the salt-wasting and simple virilizing types: the salt-wasting form is the most severe, characterized by combined cortisol and aldosterone deficiency, whereas the simple virilizing form retains sufficient aldosterone to avoid salt loss but still causes androgen excess (4). Non-classic CAH represents a milder phenotype that typically presents later in life with features such as hirsutism, menstrual irregularities, infertility in females, or early puberty in males (1). Less common enzymatic defects include 11β-hydroxylase, 17α-hydroxylase, and 3β-hydroxysteroid dehydrogenase deficiencies, each associated with distinct hormonal and clinical profiles (3).

The global prevalence of CAH varies widely, with higher rates reported in populations where consanguinity is common (5). The incidence of classic CAH is estimated at approximately 1 in 10,000 to 1 in 15,000 live births, although this differs by ethnicity and geographic region (6). In Iraq, precise prevalence data are lacking because of limited large-scale

epidemiological studies. However, a report from the Pediatric Endocrine Consultation Clinic in Baghdad identified CAH in 62 children, with 82% linked to consanguineous marriages, indicating a significant regional burden (7). Comparable data from neighboring countries show a prevalence of about 1 in 6,400 in Saudi Arabia and 1 in 9,030 in the United Arab Emirates, suggesting that Iraq may have similar rates due to shared genetic and sociocultural factors (7).

The underlying pathophysiology depends on the specific enzyme defect within the adrenal steroid biosynthesis pathway (1). In 21-hydroxylase deficiency, impaired cortisol production leads to loss of negative feedback at the hypothalamic–pituitary level, resulting in elevated adrenocorticotropic hormone (ACTH) and adrenal hyperplasia (2). In severe cases, aldosterone deficiency produces salt-wasting crises that can be life-threatening during the neonatal period (8). Early diagnosis is therefore critical to prevent adrenal crises and initiate timely hormone replacement (9). Measurement of serum 17-hydroxyprogesterone (17-OHP) remains the cornerstone of diagnosis, as levels are markedly elevated in affected individuals (10). Molecular confirmation through CYP21A2 mutation analysis further refines classification and guides management (10).

Clinical manifestations vary according to sex, disease severity, and timing of diagnosis. In genetic females (XX), excess androgen exposure commonly results in ambiguous genitalia at birth, including clitoromegaly and labial fusion, despite normal internal reproductive organs (11,12). Genetic males (XY) typically appear normal at birth but later develop signs of androgen excess such as early pubarche, rapid growth, and increased muscle mass (12). Salt-wasting CAH, the most severe form, presents in early infancy with vomiting, dehydration, and electrolyte imbalance and may be fatal if untreated (13,14). In contrast, non-classic CAH often presents during childhood or adolescence with hirsutism, menstrual irregularities, or hyperandrogenism that can mimic polycystic ovary syndrome (PCOS) and delay diagnosis (12,15). Males may develop early puberty and premature epiphyseal closure, resulting in short adult stature (15). Testicular adrenal rest tumors (TARTs) represent an important complication in males and may impair fertility and testosterone production (16). Rare forms such as 17α-hydroxylase deficiency present differently, with hypertension, hypokalemia, and delayed sexual development, highlighting the need for comprehensive hormonal and genetic evaluation (18).

Biochemical assessment is central to both diagnosis and monitoring. Elevated 17-OHP is the key marker in 21-hydroxylase deficiency (19), while increased androstenedione and testosterone reflect diversion of steroid precursors toward androgen synthesis (20). In salt-wasting disease, plasma renin activity (PRA) rises because of mineralocorticoid deficiency and is useful for assessing treatment adequacy (21). Emerging biomarkers, including 11-oxygenated androgens such as 11-ketotestosterone and 11β-hydroxyandrostenedione, show promise as more specific indicators of androgen excess (19). Additionally, 21-deoxycortisol has been proposed to improve the specificity of newborn screening and reduce false-positive results (22). Chronic ACTH elevation drives adrenal hyperplasia and androgen excess, contributing to virilization, hirsutism, and precocious puberty (23). Glucocorticoid therapy aims to suppress ACTH, but careful dose titration is essential to avoid both iatrogenic Cushing syndrome and persistent hyperandrogenism (24).

Electrolyte disturbances are a hallmark of salt-wasting CAH, which accounts for about 75% of classic cases (25). Aldosterone deficiency leads to hyponatremia, hyperkalemia, and metabolic acidosis, resulting in dehydration and shock if untreated (26). Patients may also experience hypoglycemia during illness or fasting because of cortisol deficiency (27). Pubertal disorders are common: untreated or poorly controlled patients often develop precocious puberty with accelerated skeletal maturation and reduced adult height (28–30), whereas rare forms such as 17α-hydroxylase deficiency may cause delayed puberty requiring hormone replacement therapy (31,32). Beyond physical complications, many patients experience significant psychological distress related to gender identity, body image, and social stigma, underscoring the need for multidisciplinary care (33,34). Fertility may also be affected; females may develop chronic anovulation and PCOS-like features (35), while

TARTs contribute to infertility in up to 56.7% of affected males (36).

Management of CAH relies on lifelong hormone replacement to correct glucocorticoid and, when necessary, mineralocorticoid deficiency (37). Glucocorticoids such as hydrocortisone or dexamethasone suppress excess ACTH and androgen production, while fludrocortisone and sodium supplementation are required in salt-wasting forms. Stress-dose steroids are essential during illness or surgery to prevent adrenal crisis. Selected female infants may undergo surgical correction of genital ambiguity based on ethical and clinical considerations (3). Novel therapies, including modified-release hydrocortisone, gene therapy, and enzyme inhibitors, are under investigation to improve disease control and minimize long-term complications (1).

This study aims to evaluate the demographic characteristics and clinical variability of CAH patients, analyze key biochemical and hormonal abnormalities, and assess puberty-related concerns, growth outcomes, and fertility challenges among affected individuals.

METHODS

This study was designed as a retrospective, record-based analysis conducted at the Al-Faiha Specialized Diabetes, Endocrine, and Metabolism Center in Basra, Iraq. Medical records of patients diagnosed with congenital adrenal hyperplasia (CAH) were reviewed over a 15-year period from 2010 to 2025. Data extraction was performed retrospectively between January 1st, 2025 and June 1st, 2025. The study focused on demographic characteristics, clinical features, biochemical profiles, hormonal markers, and pubertal development parameters associated with CAH. Ethical approval and official endorsement were obtained from the Basrah Health Directorate. As the study utilized deidentified archived medical records without direct patient contact, informed consent was not required.

A total of 240 patient records were initially reviewed using a convenient inclusive sampling approach. Eighty patients were excluded because they did not fulfill the established diagnostic criteria, leaving 160 eligible cases for final analysis. Inclusion criteria were based on recognized clinical and hormonal diagnostic standards for both classic and non-classic CAH, with particular emphasis on the classic form. Patients were included if they demonstrated characteristic clinical features such as ambiguous genitalia in newborn females, signs of precocious or delayed puberty, growth abnormalities including advanced bone age and accelerated linear growth, or hypertension, particularly in older children and adolescents. Hormonal criteria included elevated serum 17-hydroxyprogesterone (17-OHP) as the primary diagnostic marker, along with increased adrenal androgens such as dehydroepiandrosterone (DHEA) and androstenedione.

Data were categorized into multiple domains. Age was grouped into five categories according to World Health Organization (WHO) classification (38): infants (0–12 months), 1–3 years, 4–12 years, 13–18 years, and 18 years and above. Sex was recorded as male, female, or unknown. Residence was classified as urban (Basra city center), rural (peripheral areas of Basra Governorate), or other governorates within Iraq. CAH type was classified into classic and non-classic forms based on clinical presentation and 17-OHP levels (3). Classic CAH was defined as basal 17-OHP levels >10,000 ng/dL or ACTH-stimulated 17-OHP >10,000 ng/dL, while non-classic CAH was defined as basal 17-OHP levels between 200–10,000 ng/dL or ACTH-stimulated levels between 1,000–10,000 ng/dL.

The mode of presentation was documented and included menstrual problems (39), atypical genitalia (40), delayed puberty (41), hirsutism (42), acne (43), hyperpigmentation (44), hypertension (45,46), infertility (47), obesity (48), polycystic ovary syndrome (PCOS) (49), precocious puberty (50), and short stature (51). Family history of CAH among first-degree relatives was recorded as positive or negative. Biochemical parameters collected included 17-OHP, ACTH, cortisol, and DHEA levels. Karyotype results were documented according to the International System for Human Cytogenomic Nomenclature (ICNS) (52). Stature was classified as short, normal, or tall based on WHO child growth standards (53), and bone age was assessed as normal, delayed, or

advanced according to the Greulich and Pyle atlas (54).

Statistical analysis was performed using SPSS version 26.0. Descriptive statistics were applied to summarize demographic, clinical, and biochemical characteristics. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as median and interquartile range (IQR). Comparative analyses between classic and non-classic CAH groups were conducted using the Chi-square test or Fisher's exact test for categorical variables. Independent samples t-tests were used to compare continuous variables between groups. A p-value of less than 0.05 was considered statistically significant.

RESULTS

The demographic distribution of 160 enrolled patients reveals a predominance of individuals aged over 18 years (39.4%), followed by children aged 4–12 years (18.8%). The infant group (0–12 months) constitutes 15.6%. Adolescents (13–18 years) and toddlers (1–3 years) represent 12.5% and 13.8%, respectively. The gender distribution is significantly skewed towards females, comprising 74.4% of the cross sectional. The residential distribution indicates a balanced representation from rural (44.4%) and urban (45.0%) regions, with a smaller proportion from other governorates areas (10.6%). As shown in table (1).

Table 1 Demographic parameters distribution of the enrolled patients

Variables		Frequency (No. 160)	Percentages
Age (years)	Infant (0-12 months)	25	15.6%
	1–3 years	22	13.8%
	4-12 years	30	18.8%
	13-18	20	12.5%
	>18	63	39.4%
Gender	Male	41	25.6%
	Female	119	74.4%
Address	Rural	71	44.4%
	Urban	72	45.0%
	Other governorates	17	10.6%

The data show a significant association between gender and classification type, with males more frequently represented in the Classic group (40.0%) compared to the non-classic group (11.25%) (p = 0.04). The family history of CAH shows a significant association with the classic subtype, where 18.8% report a positive history, compared to only 2.5% in the non-classic group (P = 0.001). The negative histories in non-classic cases (97.5%) . As shown in table (2).

Table 2 The association between gender and family history and the types of CAH

Variables		Classic	Non-classic	P value	Total
Gender	Male	32 (78.0%)	9 (22.0%)	0.04	41 (25.62%)
	Female	48 (40.3%)	71 (59.7%)		119 (74.38%)
Family history	Positive	15 (88.2%)	2 (11.8%)	0.001	17 (10.62%)
	Negative	65 (45.5%)	78 (54.5%)		143 (89.38%)

This table illustrates the distribution of clinical manifestations among patients with congenital adrenal hyperplasia (CAH) according to type and sex. Atypical genitalia represented the predominant presentation in classic CAH, particularly among females (54.7%) and to a lesser extent in males (23.4%). In contrast, non-classic females most frequently presented with hirsutism (93.6%) and menstrual irregularities (81.8%). Precocious puberty was observed more commonly in classic males (65%). Other manifestations such as infertility, obesity, and PCOS were confined mainly to non-classic females. As shown in table (3).

Table 3 Mode of presentation distribution among the types of CAH

Mode of presentation	Classic		Non-classic		P value
	Female	Male	Female	Male	
Menstrual problem	2 (18.2%)	0 (0.0%)	9 (81.8%)	0 (0.0%)	---------
Atypical genitalia	35 (54.7%)	15 (23.4%)	8 (12.5%)	6 (9.4%)	0.559
Delayed puberty	0 (0.0%)	0 (0.0%)	1 (100.0%)	0 (0.0%)	---------
Hirsutism	3 (6.4%)	0 (0.0%)	44 (93.6%)	0 (0.0%)	---------
Acne	0 (0.0%)	0 (0.0%)	2 (100.0%)	0 (0.0%)	---------
Hyperpigmentation	0 (0.0%)	1 (50.0%)	1 (50.0%)	0 (0.0%)	0.098
Hypertension	0 (0.0%)	2 (100.0%)	0 (0.0%)	0 (0.0%)	---------
Infertility	1 (25.0%)	0 (0.0%)	2 (50.0%)	1 (25.0%)	0.754
Obesity	0 (0.0%)	0 (0.0%)	6 (100.0%)	0 (0.0%)	---------
PCOS	0 (0.0%)	0 (0.0%)	3 (100.0%)	0 (0.0%)	---------
Precious puberty	3 (15.0%)	13 (65.0%)	2 (10.0%)	2 (10.0%)	0.519
Short stature	4 (44.4%)	3 (33.3%)	2 (22.2%)	0 (0.0%)	0.777
Total	48	34	80	9	

Patients exhibit markedly elevated 17-OHP levels (Median + IQR) (2752.5 + 7125.25), significantly higher than non-classic cases (590.0 + 2182.0, P < 0.001), consistent with the diagnostic hallmark of CAH. ACTH is also significantly elevated in classic cases (P = 0.018), indicating adrenal hyperactivity. However, cortisol and DHEA levels do not differ significantly (P = 0.922 and P = 0.398, respectively). As shown in table (4).

Table 3.4 The association between the biochemical variables and the types of CAH

Variables	Classic	Non-classic	P value
17 OHP (Median)	2752.5 + 7125.25	590.0 + 2182.0	<0.001
Serum Cortisol (Median)	3.6 + 7.6	12.0 + 11.8	0.922
ACTH (Median)	161.0 + 480.525	50.0 + 79.580	0.018
DHEA (Median)	168.4 + 343.95	395.0 + 276.6	0.398

This table highlights the sex distribution within each age group, revealing a statistically significant skew (P < 0.001) towards male patients in infancy, with 29.3% males compared to 10.9% females. This disparity persists through early childhood but reverses dramatically in the >18 age group, where females comprise 52.1% compared to only 2.4% males. As shown in table (5).

Table 5 The association between sex and age groups in CAH patients

Variables	Female XX	Male XY	P value
Infant (0-12 months)	13 (10.9%)	12 (29.3%)	<0.001
1–3 years	11 (9.2%)	11 (26.8%)	
4-12 years	18 (15.2%)	12 (29.3%)	
13-18	15 (12.6%)	5 (12.2%)	
>18	62 (52.1%)	1 (2.4%)	

Karyotype analysis indicates a high rate of unknown results in both sexes (approximately 47%). Among known results, most females exhibit a 46XX karyotype (43.18%), with rare anomalies such as 45XO or 46XY. Interestingly, a substantial proportion of males are also 46XX (41.17%). As shown in table (6).

Table 6 Karyotypes in regard to sex in patients of CAH

Initial sex assignment	Karyotype	Frequency	Percent
Female	46xx	19	43.18%
	46xy	1	2.27%
	45XO	1	2.27%
	45 XX	1	2.27%
	Not done	21	47.73%
Total		43	100%
Male	46xx	14	41.17%
	46xy	4	11.76%
	Not done	16	47.06%
Total		34	100%

There is a significant divergence between initial sex assignment and current gender identity (P = 0.04), among those initially assigned female, 8.7% of whom now identify as male. Conversely, 69.2% of initially male-assigned individuals identify as female. As shown in table (7).

Table 7 Comparison of Initial Sex Assignment vs. Current Gender Identity among patients with CAH

Initial \ Current	Male (current)	Female (current)	P value	Total
Male (initial)	4 (30.8%)	9(69.2%)	0.04	13 (36.11%)
Female initial)	2(8.7%)	21 (91.3%)		23(63.89%)
Total	6(16.7%)	30(83.3%)		36 (100.0%)

The relationship between height measurement and age in CAH patients indicates that the majority of patients fall within the normal stature category (71.88%), with short stature affecting 25.0% and tall stature being exceedingly rare (3.13%). Short stature is consistently present across all age groups. As shown in table (8).

Table 3.8 The association between length/height measurement and age in CAH patients

Age	Gender	Short stature	Normal stature	Tall stature	Total	P value
(0-12months)	Male	1(8.3%)	11(91.7%)	0(0%)	12	0.425
	Female	1(7.7%)	12(92.3%)	0(0%)	13	
(1-3years)	Male	2(18.2%)	9(81.8%)	0(0%)	11	0.071
	Female	4(36.36%)	7(63.63%)	0(0%)	11	
(4-12 years)	Male	1(8.3%)	9(75%)	2(16.7%)	12	0.8
	Female	3(16.67%)	12(66.67%)	3(16.67%)	18	
(13-18years)	Male	3(60%)	2(40%)	0(0%)	5	0.062
	Female	4(26.67%)	11(73.33%)	0(0%)	15	
>18 years	Male	1(100%)	0(0%)	0(0%)	1	0.08
	Female	20(32.3%)	42(67.7%)	0(0%)	62	
Total		40(25%)	115(71.88%)	5(3.13%)	160	

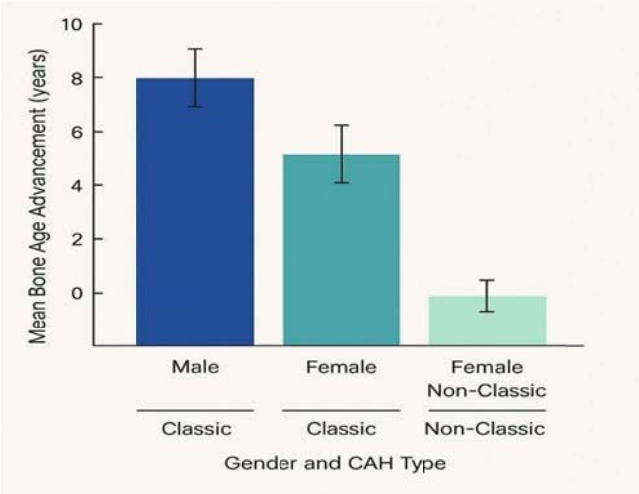

Figure 1 The mean bone age advancement by CAH type and gender

The figure shows that bone age advancement is highest in males with classic CAH (~8 years), followed by females with classic CAH (~5 years), and lowest in females with non-classic CAH (<2 years).

DISCUSSION

Congenital adrenal hyperplasia (CAH) represents a heterogeneous group of inherited endocrine disorders with wide variability in clinical presentation, hormonal profiles, and long-term outcomes (3). In settings such as Iraq, the management of CAH is further complicated by limited neonatal screening, delayed diagnosis, and sociocultural factors including high rates of consanguinity. This study examined the demographic, clinical, and biochemical characteristics of CAH patients managed at a specialized center in Basra.

A female predominance (74.4%) was observed, with a substantial proportion of patients presenting in adulthood (39.4%). Rural and urban distributions were nearly equal. The higher detection rate in females likely reflects earlier recognition due to visible genital ambiguity, consistent with findings from Mohsin et al. (2022) and Harshitha & Kalra (2022) (12,17). Boyareddy et al. (2023) similarly reported that male patients are often missed unless they present with adrenal crisis (55). The relatively high proportion of adult presentations in this cohort likely indicates delayed diagnosis, particularly in underserved areas.

In classic CAH, atypical genitalia was the dominant clinical feature, affecting 72.0% of females and 42.0% of males. Precocious puberty was also prominent, particularly in males (37.14%). By contrast, non-classic CAH predominantly affected adolescent and adult females, with hirsutism (55.0%) and menstrual irregularities (11.2%) being the most common presentations. These patterns closely mirror previous reports demonstrating that classic CAH typically presents early with virilization, whereas non-classic disease presents later with hyperandrogenic features (56,57). Boyareddy et al. (2023) also emphasized that PCOS-like features contribute to delayed recognition of non-classic CAH (55). Together, these findings reinforce the established phenotypic distinction between classic and non-classic disease.

Family history showed a stronger association with classic CAH (18.8%) compared with the non-classic form (2.5%), supporting the known autosomal recessive inheritance pattern. Similar familial clustering has been reported in populations with high consanguinity. Al-Jurayyan et al. (2015) documented multiple affected children in over half of families with CAH in Saudi Arabia (58), while Shafaay et al. (2023) reported family history in 57.3% of Middle Eastern patients along with significant psychosocial burden (59). Sonawale et al. (2017) further illustrated genetic clustering in autosomal recessive CAH (60). The stronger familial signal in classic CAH likely reflects its more severe and recognizable phenotype, whereas milder non-classic cases may remain undiagnosed in relatives. Notably, Saroufim et al. (2023) reported that 19% of classic CAH cases were missed on newborn screening despite positive family history (61).

Biochemically, classic CAH showed markedly elevated 17-hydroxyprogesterone (17-OHP) and ACTH levels compared with non-classic disease, consistent with established diagnostic patterns. Prior studies confirm that 17-OHP remains the most reliable discriminator between CAH phenotypes (19), and ACTH stimulation improves diagnostic accuracy (64). The absence of significant differences in cortisol and DHEA likely reflects hormonal variability related to sampling timing, treatment adherence, and individual metabolic factors.

Karyotype analysis in a subset of patients revealed clinically meaningful discordance between phenotypic and genetic sex, with 30.56% undergoing reclassification. Similar observations have been reported by Adriaansen et al. (2024) and Neves et al.

(2021), highlighting the impact of prenatal androgen exposure on sex assignment and gender development (65,66). Gender identity shifts were particularly evident among individuals initially assigned male. These findings align with Chowdhury et al. (2015), who demonstrated that delayed diagnosis in resource-limited settings can influence gender identity outcomes (67). Collectively, this underscores the importance of early genetic confirmation and longitudinal psychological support.

Growth impairment remains an important long-term concern. Short stature was observed in 25.0% of patients, consistent with previous reports that CAH adversely affects linear growth (68). Most patients had normal height, while tall stature was rare, reflecting premature epiphyseal closure from chronic androgen excess (69). Similar prevalence of short stature has been reported in Saudi cohorts (71). Emerging adjunctive therapies such as anastrozole have shown promise in improving predicted adult height in selected patients (72). Bone age advancement was most pronounced in males with classic CAH, consistent with the known relationship between androgen excess and accelerated skeletal maturation (73). Multicenter studies by Troger et al. (2021) and Wasniewska et al. (2020) further confirm that bone age advancement is a key determinant of compromised adult height (74,75), reinforcing the need for individualized treatment strategies (76).

Overall, this study highlights the complex clinical spectrum of CAH in a regional context. Classic CAH is typically identified earlier because of severe manifestations such as ambiguous genitalia and salt-wasting crises, whereas non-classic CAH frequently presents later—particularly in females—with hyperandrogenic features that contribute to diagnostic delay.

CONCLUSION AND RECOMMENDATIONS

This study demonstrates that congenital adrenal hyperplasia (CAH) presents with marked variability according to age, sex, and disease severity. Classic CAH typically manifests early with severe features, whereas non-classic CAH often appears later with milder hyperandrogenic symptoms such as hirsutism and menstrual irregularities. Hormonal markers, particularly 17-OHP and ACTH, proved valuable in differentiating disease forms. The findings also highlight ongoing challenges related to sex assignment and gender identity, emphasizing the importance of early recognition and comprehensive care. Accordingly, implementation of national neonatal screening using 17-OHP is strongly recommended to enable early diagnosis and prevent adrenal crises. Establishing regional genetic and karyotyping services would improve diagnostic accuracy and support appropriate sex assignment. Management should involve multidisciplinary CAH teams, alongside provision of genetic counseling—particularly in consanguineous populations, to increase awareness of inheritance risks. Regular monitoring of growth and bone age is essential to detect early growth compromise, and the use of structured clinical algorithms is recommended to improve identification of non-classic CAH, especially in females presenting with PCOS-like features.

Conflicts of Interests: None

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Ethical Approvals: Ethical approval for the study was obtained from the relevant institutional review board, and informed consent was acquired from all participants prior to their inclusion in the study.

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