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Levels of 17-Hydroxyprogesterone, Renin, Testosterone, And Electrolytes in Congenital Adrenal Hyperplasia Patients

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ABSTRACT

Introduction: Congenital adrenal hyperplasia (CAH) is an autosomal-recessive condition primarily caused by 21-hydroxylase deficiency. It is the most common cause of ambiguous genitalia in infants and children. It can be a fatal condition if left untreated.

Objectives: The study's objective was to estimate the levels of 17-hydroxyprogesterone, plasma direct renin, testosterone, and electrolytes in patients with CAH.

Place and Duration of Study: This cross-sectional study was conducted at the University of Child Health Sciences, Lahore between March 2021 till April 2022.

Material & Methods: This study included ninety CAH patients up to 14 years of age who underwent clinical and physical testing without gender restriction. Patients without confirmed CAH but with elevated levels of 17-hydroxyprogesterone were excluded. An ultrasound examination was done to confirm the presence of ambiguous genitalia. ELISA was used to estimate the values of 17-hydroxyprogesterone. Plasma direct renin and testosterone were measured by chemiluminescence immunoassay. The values of sodium, potassium and chloride were measured using an electrolyte analyzer. Data was analyzed using IBM SPSS version 26.0.

Results: Of the total 90 patients, 46 (51.11%) were boys and 44 (48.88%) were girls. 55 (61.10%) CAH patients had high levels of 17-OHP. High testosterone and plasma direct renin levels were observed in 31 (34.40%), and 43 (47.80%) CAH patients respectively. Mostly, CAH patients had normal levels of sodium 70 (77.80%), potassium 54 (60.0%), and chloride 57 (63.30%). The difference in 17-OHP (ng/mL) levels was found to be statistically significant (p=0.002) in both genders.

Conclusion: There is a significant association between elevated 17-OHP and CAH in the pediatric population, indicating that the 17-OHP can be used as a diagnostic tool for CAH patients.

Keywords: Congenital adrenal hyperplasia, 17-hydroxyprogesterone, plasma direct renin, testosterone, serum electrolytes

INTRODUCTION

Genetic diseases affecting the adrenal glands are known as congenital adrenal hyperplasia (CAH). Despite being uncommon, steroid 21-hydroxylase deficiency (21-OHD), an autosomal-recessive condition of adrenal steroidogenesis resulting from CYP21A2 mutations, is the cause of the most frequent type of CAH[1]. The 21-OHD CAH is classified into moderate non-classic CAH and severe classic CAH[2].

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According to newborn screening, the classic CAH is an uncommon condition that affects 1 in 14,000 to 1 in 18,000 infants globally. In classic CAH, cortisol, aldosterone, and adrenaline are deficient, and compensatory mechanisms cause excess adrenal androgens. These three critical nutrients (cortisol, aldosterone, and adrenaline) play important roles in several homeostatic processes.

Based on the severity of the disease, classical CAH is further classified into two forms: simple virilizing CAH, and salt-wasting CAH[4]. The salt-wasting CAH is related to CYP21A2 mutations that ablate enzyme activity which leads to aldosterone and cortisol deficit. Infants with salt-wasting CAH have a potentially fatal adrenal crisis in the first two weeks of life if treatment is not received early[5]. Elevated precursor steroids are transferred to the androgen pathways which are unaffected by the lack of negative feedback on the hypothalamic pituitary adrenal axis, that results in an excess of adrenal androgen production. Infants with this type of CAH rapidly experience hyponatremia, hyperkalemia,

acidosis, shock, and possibly deadly "salt-wasting crises"[3]. Although newborns with increased aldosterone production are less likely to experience an acute salt-wasting crisis, they still have a significant cortisol shortage and significantly higher adrenal androgen production. With residual enzymatic activity ranging from 1% to 5% of normal, they are considered to have "simple virilizing" CAH. The simple virilizing CAH is related to CYP21A2 mutation[5]. Non-classical CAH is a less severe, and more prevalent type of disease that appears later in adulthood, lacks genital ambiguity, and is not immediately life-threatening. It might continue to show no symptoms or be misdiagnosed as other diseases[6]. With an estimated frequency of 1 non-classic CAH case per 200 people in the USA and a carrier rate of 4.0–7.5% in many European countries[7].

CAH is also the most typical reason why a baby has ambiguous genitalia[8]. In the Pakistani population, CAH often goes untreated during the neonatal period for a variety of reasons, including ignorance, lack of access to accurate diagnosis, erroneous societal attitudes, and religious dogmas[9]. There is ethnic variety in Pakistan, and various ethnic groups may have varying rates of CAH[10]. Many children, especially boys, are asymptomatic, but others may exhibit signs such as acne, early adrenarche, and rapid skeletal maturity as a result of higher adrenal androgens. Subnormal fertility, acne, oligomenorrhea, and hirsutism can all be present in adolescent girls or adult women. In addition to adrenal insufficiency, patients with CAH have virilization, early puberty, decreased fertility, acne, the development of testicular adrenal rest tumors in males, and a decreased standard of life as a result of chronic adrenal-derived androgen excess[3,11,12].

According to a study conducted in Islamabad, Pakistan possesses comparatively less knowledge regarding CAH. Some of the traits seen were a high degree of consanguinity, an increase in family mortality, an inaccurate gender assignment at birth, and a delay in diagnosis[13]. Due to a delayed diagnosis, many CAH patients in Pakistan have genital anomalies. As the most prevalent cause of virilization at birth, 21-OHD may be diagnosed in neonates using the 17-hydroxyprogesterone (17-OHP) assay. Newborn screening programs in many countries use 17-OHP to diagnose 21-OHD CAH[14]. So, the present study was conducted to estimate the levels of 17-OHP associated with CAH in the pediatric population. The objective was to estimate the levels of 17-OHP, plasma direct renin, testosterone, sodium, potassium, and chloride in CAH pediatric patients and their association with CAH.

MATERIAL AND METHODS

This cross-sectional study was done between the period of March 2021 to April 2022 and included ninety patients. The study was conducted at the Department of Immunology and Serology, the Department of Medical Genetics, University of Child Health Sciences, Lahore, Pakistan after approval by the institutional review board (IRB No. 1436/SAHS dated 12/02/2021). The patients of CAH who underwent clinical and physical testing (by physician and geneticist), without gender restriction and with a range of up to 14 years were included. Patients without confirmed CAH but with elevated levels of 17-OHP were excluded. Ultrasound examination was done for those male patients whose testes were not palpable and to visualize the presence of the uterus and ovaries. Karyotyping was used to determine the subject's gender.

After obtaining written informed consent from the parents/guardian, about 5 ml of blood was taken from each patient in clotted vacutainers and 5 ml of blood was taken in an EDTA vacutainer. The samples were centrifuged at the speed of 3000 rpm for serum and plasma separation. The Enzyme-linked immunosorbent assay (ELISA) technique was used to estimate the values of 17-OHP (Kit reference No. DKO004). The plasma direct renin, and testosterone, were measured by chemiluminescence immunoassay (Maglumi X3). The levels of serum sodium (Na), serum potassium (K), and serum chloride (Cl) were measured by an automated electrolyte analyzer working on an ion-selective electrode technique. The plasma direct renin was measured in the EDTA plasma of all patients and the remaining parameters were measured in serum samples. The controls were also run with every parameter to monitor any analytical error. Data was analyzed using Statistical Package for the Social Sciences version 26.0 (IBM SPSS 26.0). The descriptive statistical analysis was done and the mean ($\pm$ SD), frequencies, and percentages were calculated. A chi-square test was applied to evaluate the association between variables. The p-value of ≤ 0.05 was considered statistically significant.

RESULTS

According to the chromosomal analysis, from the total 90 patients, 46 (51.11%) were males (46 XY) and 44 (48.88%) were females (46 XX). The patients had the ages up to 14 years. Hyperpigmentation was present in only 07 (7.80%) CAH patients while palpable gonads were present in 41 (45.60%). Scrotal rugae was absent in 06 (6.70%) CAH patients. The urethral opening was present in 81 (90.0%), vaginal opening was present in 26 (28.90%), urogenital sinus was present in 22 (24.4%), and clitoral enlargement was present in 29 (32.20%) CAH patients. The distribution of genital presentation in both genders is presented in Table 1.
The chi-square test was used to find the difference in presentation of genitalia between both genders identified through chromosomal analysis. Statistically significant difference was observed in the presence of gonads, vaginal opening, urogenital sinus, clitoral enlargement, scrotum, uterus, ovaries, testes, hypospadias, labial folds, and size of penis.
The 55 (61.10%) CAH patients were observed to have high levels of 17-OHP. The high testosterone level was observed in 31 (34.40%) CAH patients.
The plasma direct renin level was high in 43 (47.80%). Most CAH patients had normal serum Na 70 (77.80%) levels while serum K was high in 33 (36.70%). The serum chloride level was high in 23 (25.60%) CAH patients (Table III). The mean values of all parameters are presented in Table 2. The difference in 17-OHP (ng/mL) levels was found to be statistically significant (p=0.002) in both gender (male and female). The association of all parameters with gender is presented in Table 3.

Table 1: Frequency and association of genital features with the gender based on chromosomal analysis

Variables	Male n=46	Female n=44	p-value*
Hyperpigmentation			
Present	02 (4.34%)	05 (11.36%)	0.190
Not present	44 (95.56%)	39 (88.63%)	
Gonads			
Palpable	32 (69.56%)	09 (20.45%)	0.001*
Not palpable	14 (30.43%)	35 (79.54%)	
Scrotal rugae			
Normal	40 (86.95%)	37 (84.09%)	0.785
Mild	03 (6.52%)	04 (9.09%)	
Absent	03 (6.52%)	03 (6.81%)	
Urethral opening			
Yes	41 (89.13%)	40 (90.90%)	0.529
No	05 (10.86%)	04 (9.09%)	
Vaginal opening			
Yes	08 (17.39%)	18 (40.90%)	0.013*
No	38 (82.60%)	26 (59.09%)	
Urogenital sinus			
Yes	03 (6.52%)	19 (43.18%)	0.001*
No	43 (93.47%)	25 (56.81%)	
Clitoral enlargement			
Yes	08 (17.39%)	21 (47.72%)	0.002*
No	38 (82.60%)	23 (52.27%)	
Scrotum			
Bifid	20 (43.47%)	04 (9.09%)	0.001*
Labio scrotal folds	05 (10.86%)	13 (29.54%)	
Normal	18 (39.13%)	01 (2.27%)	
No scrotum	03 (6.52%)	26 (59.09%)	
Uterus			
Present	06 (13.04%)	35 (79.54%)	0.001*
Not present	40 (86.95%)	09 (20.45%)	
Ovaries			
Present	04 (8.69%)	24 (54.54%)	0.001*
Not present	42 (91.30%)	20 (45.45%)	
Testes			
Present	38 (82.60%)	06 (13.63%)	0.001*
Not present	08 (17.39%)	38 (86.36%)	
Hypospadias			
Yes	20 (43.47%)	06 (13.63%)	0.002*
No	26 (56.52%)	38 (86.36%)	
Labial folds			
Present	05 (10.86%)	23 (52.27%)	0.001*
Not present	41 (89.13%)	21 (47.72%)	
Size of penis			
Short	29 (63.04%)	18 (40.90%)	0.002*
Enlarged	03 (6.52%)	01 (2.27%)	
Normal	11 (23.91%)	06 (13.63%)	
Absent	03 (6.52%)	19 (43.18%)	

***Chi-square test**

DISCUSSION

CAH refers to a series of recessively inherited enzyme abnormalities that affect adrenal steroid production, resulting in low cortisol and aldosterone levels and elevated androgens in the blood circulation of those with this condition[2]. Early pubescence and rapid growth in older children cause an early epiphyseal maturation that causes a short adult height. An excess of androgens in the body can cause irregular menstruation cycles, early virilization, female pubic hair development, and infertility in both sexes if left unchecked. Research on the psychological effects of high testosterone has been focused on its effects on the female brain; however, evidence of a worse quality of life has been found in adults with CAH. Even though steroid replacement therapy can stop adrenal crises and restore normal growth, CAH has a long-term impact

on health[15]. So, the present study was conducted to estimate the values of 17-OHP, plasma direct renin, testosterone, serum Na, serum K, and serum Cl in CAH patients. In the present study, gender confirmation was done through chromosomal analysis. From a total of 90 patients, 46 (51.11%) were male (46 XY) CAH patients, and 44 (48.88%) were female (46 XX) CAH patients, with the patients ranging in age up to 14 years. These findings were aligned with the findings of Mukhtar et al, who reported that from a total of 90 patients, 62 (68.89%) were males, and 28 (31.11%) were females, with age of patients were between 0-10 years[15]. The higher frequency of males over females in South Asian countries could be a result of societal preferences, leading to the assignment of male gender to females who had even slight resemblance with males. The study by Seneviratne et al., 2021 included 52 patients, of which 27 (51.92%) were girls and 25 (48.07%) were boys[16]. A study by Fatima et al presented that 63.2% of CAH patients had initially presented as males, and out of these, 44.8% were reassigned to female gender based on chromosomal analysis[11].

17-OHP is an important biomarker in the diagnosis and management of CAH, particularly in its most common form, 21-OHD[17]. The estimation of 17-OHP in newborns and children is very important. Early detection through elevated 17-OHP levels allows for timely intervention, which is crucial for preventing life-threatening adrenal crises and for initiating appropriate treatment early in life[18]. In the present study, 61.10% of CAH patients were observed to have high levels of 17-OHP, with a statistically significant association (p=0.002) with gender. The study by Fatima et al presented that 49% of CAH patients had high levels of 17-OHP. The mean (± standard deviation) value of 17-OHP was 3.99+8.05 in the present study. The study of Dehkordi et al presented a median (± Inter quartile range) value of 8.40±21.83, which is less than the present study. The study of Dehkordi et al also presented the median (± Inter quartile range) value of testosterone, as 0.66±2.00. which is also lower than the present study mean (± standard deviation) value of testosterone 9.61+8.42[19]. The difference in the mean values was due to the differences in the measuring units. The present study measured 17-OHP, and testosterone levels in ng/mL while the Dehkordi et al study measured them in ng/dL.

The study by Pofi et al estimated the plasma direct renin levels in CAH patients, which were elevated in 44.0%, normal in 26.0%, and low in 19.0% of cases[20]. In the present study, the plasma direct renin level was high in 47.80%, normal in 23.30%, and low in 28.88% of CAH patients. These findings are consistent with the Pofi et al study. The present study also estimated the mean (± standard deviation) values of serum Na (135.98+14.45), serum K (5.07+1.06), and serum Cl (104.76+8.90) in CAH patients.

A case of CAH was reported in a 2-year-old patient who presented with several abnormalities upon examination and laboratory analysis: thrombocytosis (795x103/uL), ambiguous genitalia, increased testosterone (110.1 ng/dL), increased 17-OHP (109.19 ng/mL), decreased morning serum cortisol levels (1.7 ug/dL), and very short stature. Gynecological ultrasonography revealed a uterus, hypertrophy of the left adrenal gland, no bilateral testicular structures, and indications of genital ambiguity[21].

Another case of CAH in a 4-month-old male patient was reported by Reddy et al. The male genitalia were correctly identified, and clinical symptoms of severe muscular wasting and potentially fatal dehydration were seen, along with hyperpigmentation of the overlying skin. Low serum sodium (119 mmol/L), low serum potassium (1.7 mmol/L), normal serum creatinine (61.89 µmol/L), low random blood sugar (0.89 mmol/L), normal serum magnesium (0.74 mmol/L), and normal ionized calcium (1.11 mmol/L) were observed on laboratory examinations[22].

The present study was conducted on a small cohort of CAH patients in a single-centered hospital. Some factors like consanguinity, and family history were also missing. Further studies should include more patients, consider other factors, and include more comprehensive biochemical workups should be done in the future.

Table 2: Observed and mean values of different parameters estimated in CAH patients

Para-meters	Normal ranges	Obser-ved mini range	Obser-ved maximum range	Mean± Standard deviation
17-Hydroxy-progesterone	0.26 to 16 ng/mL	0.10 ng/mL	24.30 ng/mL	13.99±8.05
Testosterone	2.20 to 10.50 ng/mL	0.20 ng/mL	29.30 ng/mL	9.61±8.42
Plasma direct renin	2.8 to 46.1 uIU/mL	0.20 uIU/mL	405.0 uIU/mL	91.05±106.97
Sodium	136 to 145 m.mol/L	99.0 m.mol/L	171.0 m.mol/L	135.98±14.45
Potassium	3.50 to 5.40 m.mol/L	2.30 m.mol/L	7.20 m.mol/L	5.07±1.06
Chloride	95 to 108 m.mol/L	78.0 m.mol/L	124.0 m.mol/L	104.76±8.90

Table 3: Frequency and association of parameters with gender distribution

Parameters	Male n=46	Female n=44	p-value (Chi-square test)
17-hydroxyprogesterone (ng/mL)			
High	20 (43.47%)	35 (79.54%)	0.002*
Normal	18 (39.13%)	05 (11.36%)	
Low	08 (17.39%)	04 (9.09%)	
Testosterone (ng/mL)			
High	20 (43.47%)	35 (79.54%)	0.989
Normal	18 (39.13%)	05 (11.36%)	
Low	08 (17.39%)	04 (9.09%)	
Plasma direct renin (uIU/mL)			
High	23 (50.0%)	20 (45.45%)	0.707
Normal	10 (21.73%)	11 (25.0%)	
Low	13 (28.26%)	13 (29.54%)	
Sodium (m.mol/L)			
High	01 (2.17%)	02 (4.54%)	0.262
Normal	39 (84.78%)	31 (70.4%)	
Low	06 (13.04%)	11 (25.0%)	
Potassium (m.mol/L)			
High	16 (34.78%)	17 (38.63%)	0.735
Normal	29 (63.04%)	25 (56.81%)	
Low	01 (2.17%)	02 (4.54%)	
Chloride (m.mol/L)			
High	12 (26.08%)	11 (25.0%)	0.107
Normal	32 (69.56%)	25 (56.81%)	
Low	02 (4.34%)	08 (18.18%)	

CONCLUSION

This study reveals that there is a significant association between 17-OHP and CAH is present in the pediatric population. So, the 17-OHP can be used as a diagnostic tool for CAH patients. Regular monitoring of 17-OHP levels can also help in assessing how well the disease is being managed with treatment.

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