Case Report

Congenital Adrenal Hyperplasia in Two Sisters in Calabar, Nigeria: Case Report and Challenges of Management

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ABSTRACT

Background: Congenital adrenal hyperplasia (CAH) is a group of autosomal recessive disorders resulting from defects in enzymes involved in cortisol biosynthesis. It is a rare but important cause of virilization and disorder of sexual development, particularly in resource-limited settings where diagnostic and treatment challenges persist. Familial clustering of CAH, though expected from its autosomal recessive inheritance, is only reported few times in Nigeria.

Case summary: We report two sisters aged 7 and 14 years from Calabar, Nigeria, who presented with symptoms and signs of androgen excess, virilization, deepening of voice, acne, hirsutism and latter with menstrual irregularities. Laboratory evaluation revealed elevated serum 17-hydroxyprogesterone and androstenedione levels. A diagnosis of classical (simple virilizing) congenital adrenal hyperplesia was made. Both patients were managed with hydrocortisone replacement therapy, leading to clinical improvement.

Conclusion: These cases highlight the familial occurrence of CAH in Nigeria and underscore the diagnostic and therapeutic challenges in low-resource settings. Training of Paediatric Endocrinologists, strengthening diagnostic capacity, ensuring drug availability, and instituting national newborn screening are essential for early diagnosis and improved outcomes.

Keywords: Congenital adrenal hyperplasia, 21-hydroxylase deficiency, virilization, hydrocortisone, Nigeria.

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INTRODUCTION

Congenital adrenal hyperplasia (CAH) comprises a group of autosomal recessive disorders resulting from defects in enzymes involved in steroid biosynthesis within the adrenal cortex. The most common form, accounting for over 90% of cases, is 21-hydroxylase deficiency caused by mutations in the CYP21A2 gene.[1, 2] Impaired cortisol synthesis leads to excessive adrenocorticotropic hormone (ACTH) production and therefore chronic overstimulation of the adrenal glands leading to adrenal hyperplasia, and accumulation of precursors proximal to the blocked enzymatic step.[3, 4] Clinically, CAH presents as two major types, the severe form called classical congenital adrenal hyperplesia (CCAH) and the mild or late-onset form called non-classical congenital adrenal hyperplesia (NCCAH).[5, 6] The CCAH has two types, the "salt-wasting" and the "simple virilizing" forms depending on the degree of aldosterone deficiency such that there may be failure to maintain sodium balance which is often potentially fatal if not treated in the "salt wasting" form. In the "simple virilizing" form, aldosterone is produced even though not optimal and therefore sodium balance is maintained but the child will have symptoms and signs of virilization from accumulation of precursor androgens proximal to the enzymatic block. On the other hand, the non-classical congenital adrenal hyperplesia (NCCAH) is a milder form of the disease and a late-onset form, manifesting later with variable degrees of virilization, menstrual irregularities, infertility, or hirsutism.[5, 6] These patients have compensated aldosterone and cortisol production but present late with symptoms of androgen excess.

The worldwide incidence of classic CAH is roughly 1:14,000 to 1:18,000 births, while the NCCAH, is relatively common, with an overall prevalence of 1:200 in the Caucasian US population and a higher frequency among Ashkenazi Jews, Hispanics, Mediterranean, Middle Easterners, and Eskimos.[7, 8]

However, the true burden in sub-Saharan Africa remains largely unknown due to under-diagnosis, limited hormonal assay capability, and absence of neonatal screening programs.[9-11] In Nigeria, only a few isolated reports exist, and many cases are detected late when virilization, precocious puberty or ambiguous genitalia becomes obvious to parents.[9, 12]

Familial clustering of CAH, though expected from its autosomal recessive inheritance, is only reported few times in Nigeria.[10, 12] Most of the time, it is isolated cases that are reported.[9] This may reflect limited endocrine diagnostic services, economic constraints, and poor follow-up compliance in affected families.[9, 10] Therefore, follow up and screening of families of affected patients is not the norm. Furthermore, effective management is often hampered by drug stock-outs, distance from tertiary care, and lack of sustained hormonal monitoring[10]. This often leads to long-term outcomes in patients affected by CAH, including psychosexual well-being of the patient and stress on the parents.[2]

We report the case of two sisters from Calabar, Nigeria, both presenting with virilization from birth and subsequently diagnosed with classical (simple virilizing) congenital adrenal hyperplasia. These cases highlight diagnostic and therapeutic challenges in a resource-constrained setting and underscore the need for improved awareness, neonatal screening, and training of more Paediatric Endocrinologists, hormonal diagnostic capacity, and genetic counseling. A brief review of the relevant literature is also presented.

Case 1

Patient profile: P01, is a 14-year-old girl, who was referred to our endocrine clinic on 16 April 2019 from another tertiary hospital in a neighboring state because their only paediatric endocrinologist had relocated overseas. She was the first of four children from a non-consanguineous marriage. They are of the Ibibio tribe of Nigeria and there was no history of similar illness in the Parents.

Clinical presentation: The patient's mother reported clitoral enlargement since birth, which progressively increased in size with age. Pregnancy and neonatal periods were uneventful. At age three, she was diagnosed to have CAH and the surgeons who saw at the time went on to perform a clitoral reduction surgery. Despite the procedure, she later developed deepening of voice, acne, and hirsutism (chin, axilla, and legs). She attained thelarche at 11 years and menarche at 13 years with irregular, light menstrual flow reported as spotting and lasting 2–3 days. There was also history of afternoon fatigue especially after returning from school.

The patient had previously been given hydrocortisone (10 mg mornings, 5 mg evening) by the referring hospital; however, the drug intake was irregular because of stock-out. Both parents have tertiary level of education; the father is a public servant while the mother, a fresh university graduate was undergoing the compulsory National Service in Nigeria.

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Examination findings: She was short for age (height 145.5 cm, –2.5 SD; weight 53 kg). She had acne, hirsutism, and a deep voice. Blood pressure was normal for age. Tanner staging: breast stage 3, pubic hair stage 4. External genitalia revealed a bifid, markedly enlarged clitoris with otherwise normal labia and vaginal opening. Patient did not give assent for clinical picture to be taken despite parent's consent.

Investigations: Serum hormone assays were done outside Nigeria due to unavailability locally showed:

- Testosterone: 0.91 nmol/L (reference 0.34–1.39)
- Androstenedione: 2.23 ng/mL (0.22–2.25)
- 17-hydroxyprogesterone (17-OHP): 1.44 ng/mL (<1.0)
- Early morning cortisol: 248 nmol/L (240-618)

Other investigations:

- Serum electrolytes, urea, and creatinine: normal
- Abdominopelvic ultrasound: normal uterus, fallopian tubes, and ovaries
- Bone age: advanced by 2 years

Diagnosis: Simple Virilizing Congenital Adrenal Hyperplasia (21-hydroxylase deficiency) with poor adherence to treatment.

Treatment and follow-up: She was continued on hydrocortisone but at 10 mg twice daily and to be reviewed in one month. However, she did not come for follow up until after four months (delayed by transport cost and poor roads). A big Pharmacy was contacted by us in the commercial city of Lagos, Nigeria, to ensure there is no stock out again. At review, she reported significant improvement in terms of afternoon fatigue and also a reduction in clitoral size. Physical exam confirmed marked regression of clitoral hypertrophy with normalization of vulvar appearance. However, voice change and hirsutism persisted. Repeat serum electrolytes remained normal. Parents were not able to repeat the hormonal assays again because of cost. She was continued on hydrocortisone 10 mg morning and 5 mg evening, with counseling on adherence and dose adjustment. Unfortunately, she was lost to follow-up due to financial constraints and distance from the facility. Phone calls to the parents to enquire about the child confirmed that she is doing well and happy with the outcome. She is now in the university and observes that once she stops the drugs the clitoral enlargement worsens and therefore, she is now conscious of taking her drugs as prescribed.

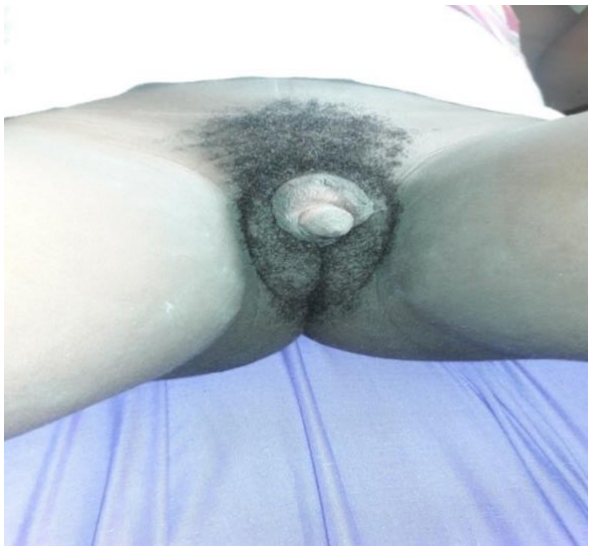

Figure 1: Clitoral enlargement seen in case 2

Case 2

Patient profile: P02, is a **7**-year-11-month-old girl, the younger sister of Case 1, presented on the same day (16 April 2019) with clitoral enlargement since birth. She was the fourth child in the same non-consanguineous family. Pregnancy and neonatal history were unremarkable.

Clinical presentation: She was diagnosed of CAH at the age of 6 years following a high level of 17OH Progesterone and a low cortisol level (the parents verbally said this as the referral note did not indicate the values). She had surgery shortly thereafter. The clitoris started enlarging again post-surgery. She also developed hirsutism, pubic hair, acne, deep voice, and post-school afternoon fatigue. Growth was accelerated, and she was taller than her peers. She had developed breast but had not attained menarche. She had earlier been started on hydrocortisone, which was discontinued when it became unavailable.

Examination findings: She was tall for age, muscular look, with waddling gait, bowing of legs, acne, and deep voice. Height: 139 cm (+2.5 SD); weight: 42 kg. Blood pressure was normal for age. Tanner stage: breast 3, pubic hair 4. External genitalia showed a markedly enlarged clitoris resembling a glans penis with otherwise normal labia and vaginal opening (see figure 1).

Figure 1: Clitoral enlargement seen in case 2

Investigations

- Testosterone: 1.98 nmol/L (reference 0.10–0.8)
- Androstenedione: 2.51 ng/mL (0.0–1.0)
- 17-OHP: 3.22 ng/mL (<1.0)
- Early morning cortisol: 206 nmol/L (240-618)

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- Serum electrolytes, urea, creatinine: normal
- Bone age: 10 years
- Abdominopelvic ultrasound: normal uterus, tubes, and ovaries

Diagnosis: Classical (simple virilizing) congenital adrenal hyperplasia (21-hydroxylase deficiency) and precocious puberty.

Treatment and follow-up: She was restarted on hydrocortisone 10 mg morning and 5 mg evening. Parents were counseled on adherence to treatment and prognosis. Arrangement was made on how to procure hydrocortisone medication. At a 4-month follow-up, she was lively, friendlier, happy, and reported resolution of afternoon fatigue. Clinical exam showed a markedly reduced clitoral size with normal vulvar appearance. Mother was happy with the outcome of treatment. She continued on the same hydrocortisone dose but was lost to follow-up due to distance and financial difficulty. Patient is however reported to be doing fine as she takes her drugs following counseling.

DISCUSSION

Congenital adrenal hyperplasia (CAH) remains a significant endocrine disorder with varying presentations, depending on the enzymatic block and degree of the enzyme deficiency. More than 90% of cases result from 21-hydroxylase deficiency, leading to impaired cortisol synthesis, variable aldosterone production, and increased androgen secretion. [1, 2] The two sisters reported here presented with features consistent with classical (simple virilizing) CAH (CCAH) - virilized external genitalia at birth which is a result of prenatal androgen excess. [1, 2] In the severe form of Classical CAH (salt wasting type), in addition to virilized external genitalia at birth, there is failure to synthesize sufficient aldosterone leading to failure to maintain sodium balance which is often potentially fatal if not treated. [1] Our patients had an uneventful neonatal period until their childhood, for which we presumed that aldosterone production was not severely affected and therefore had the simple virilizing disease. However, the clitoral enlargement was not recognized by the attending health workers at birth or even during infancy when they were receiving their childhood immunizations, to be investigated further. The postnatal androgen excess in the two sisters was the reason for the continued enlargement of the clitoris necessitating surgery to reduce clitoral size by the surgeons in the referring hospital. Despite the surgery to reduce clitoral size, the non-suppression of the androgen excess was the reason for the continued virilization in the two sisters resulting in acne, hirsutism, deep voice and advanced bone age.[3, 13] Menstrual irregularities, as seen in the elder sister who had attained menarche, was also a consequence of post-natal androgen excess. Until the excess androgens are suppressed, menstruation and fertility may be affected as seen in the elder sister in this report.[2, 13]

Precocious puberty resulting from the excess androgen can lead to accelerated growth as seen in the younger sister presenting with tall stature (height SD scores above +2). However, in the long run, final adult height is usually short as seen in the elder sister having a height below -2 SD scores.[2, 13]

The afternoon fatigue reported by both sisters is as a result of low cortisol production. [2] Low cortisol is known to decrease glucose availability, reduce stress tolerance and reduced vascular tone and because its production is also reduced as the day progresses, patients often report afternoon fatigue and weakness. [2] The level of cortisol result was low in the younger sister, confirming the reason for the unusual fatigue in the afternoons. Though cortisol was normal in the elder sister (which may be explained by the prior irregular use of hydrocortisone treatment before presentation), she still reported fatigue which improved with hydrocortisone medication dose adjustments.

The occurrence of CAH in siblings, as seen in this report, reflects its autosomal recessive inheritance pattern, where both parents are asymptomatic carriers of CYP21A2 mutations. [7, 11] Even though there was no prior history of similar symptoms reported in the parents, it is because the parents are likely asymptomatic carriers of the mutation gene. Familial clustering has been documented globally, but reports from sub-Saharan Africa are scanty. [9, 10] A similar occurrence in siblings have been reported by Oyenusi *et al* [10] and Agboola-Abu *et al* [12] in Lagos, Nigeria. In an earlier report in Benin, South-South of Nigeria, all the cases seen were sporadic with no familial clustering. [9] The rarity of such familial reports in Nigeria may not indicate low incidence but rather under-diagnosis and under-reporting due to lack of routine neonatal screening and limited endocrine diagnostic capacity. [9] Further screening of siblings and families of confirmed patients may reveal more cases in affected families. Therefore, diagnosis of a single case should necessitate evaluation of other family members.

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Both patients were initially diagnosed clinically and underwent clitoral reduction surgeries without waiting for outcome of the medical treatment over time and/or employing a multi-disciplinary approach to management. This sequence reflects a common pattern in resource-constrained settings, where early recognition of virilization often leads to surgical referral rather than to a paediatric endocrine evaluation.[9] Most of the patients reported by Osifo O *et al* [9] were referrals from neighboring General Hospitals in Benin City, Nigeria, and the neighboring states to his surgical out-patient clinic.

In our cases, some of the biochemical testing confirmation required sending samples abroad, causing delay, increased cost, and parental financial strain. This is similar to reports from Oyenusi *et al* [10] and Agboola-Abu *et al*[12] who highlighted the inaccessibility of hormonal assays in Nigeria. Karyotyping could not be performed due to cost, as well as mutational analysis which is not available in this clime. These reflect the financial and administrative barriers to comprehensive endocrine care and diagnosis in developing countries.[10, 14]

Hydrocortisone remains the cornerstone of treatment for both classical and non-classical CAH, acting by suppressing ACTH and thereby reducing androgen excess. [2, 3] Both sisters showed clinical improvement, notably reduced fatigue and regression of clitoral hypertrophy after several months on hydrocortisone therapy. However, persistent hirsutism and deepened voice are common residual features once virilization has progressed, as seen in these cases. [15, 16] There was no opportunity to further handle the hirsutism by considering adding other anti-androgens agents (spironolactone) or finasteride (that competes with di-hydro testosterone at the receptors), as the patients were lost to follow-up.[15]

A major challenge was poor follow-up adherence, attributed to transportation cost, drug stock-outs, and financial constraints; issues widely reported across Nigeria. [9, 12, 14] Drug unavailability and unsupervised dose interruption risk adrenal insufficiency and relapse of virilization.[2, 8] Therefore, counseling and continuous education of parents and patients on adherence, stress dosing and long-term monitoring are crucial.

Beyond the physical manifestations, CAH has important psychosocial consequences, including wrong gender assignment at birth, gender identity confusion, stigmatization, and low self-esteem, particularly in female patients with virilization. [2, 9, 17] Both girls and their mother expressed satisfaction with improvement in genital appearance following only four months of treatment, underscoring the positive psychological impact of medical therapy compared to surgery alone. Early diagnosis and hormonal therapy can thus prevent unnecessary surgical interventions and psychological distress.[15] The decision for genital reconstructive surgery in affected females should be taken at a multidisciplinary level when the child is in her adolescent age to contribute to the decision and to fully understand the complications of such a surgery. [2, 18] Unfortunately, the surgeons at the referring tertiary hospital did not follow this approach.

Strengths and limitations of this report

This case report highlights the familial nature of congenital adrenal hyperplasia and contributes to the limited literature from Nigeria and sub-Saharan Africa. The report provides detailed clinical descriptions, biochemical findings, and response to glucocorticoid therapy, providing valuable insights into the presentation and management of CAH in a resource-limited setting. However, the report has several limitations. It is only two patients and this limits generalizability of the findings. Genetic confirmation of the diagnosis was not performed due to financial constraints and availability of test. The long-term follow-up of the cases was limited because the patients were lost to follow-up. In addition, some recommended laboratory monitoring could not be repeated due to limited access and cost considerations.

Implications of the findings in this report

The cases underscore the importance of maintaining a high index of suspicion for congenital adrenal hyperplasia in female children presenting with clitoromegaly, virilization, or menstrual abnormalities. They also highlight the need to strengthen diagnostic capacity for hormonal assays in Nigeria, as delays in laboratory confirmation may affect timely management. The familial occurrence emphasizes the role of genetic counseling for affected families and the need for screening of siblings. Furthermore, the findings support advocacy for newborn screening programs and improved access to paediatric endocrine care, which would facilitate early diagnosis and prevent complications associated with delayed treatment.

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CONCLUSION

Congenital adrenal hyperplasia (CAH) remains an important though under recognized cause of virilization and disordered sexual development in Nigerian children. Familial cases of classical simple virilizing congenital adrenal hyperplasia are rarely reported in Nigeria but may be more common than recognized. These two sisters from Calabar illustrate the typical clinical presentation, diagnostic delays, and management challenges in resource-constrained settings. Early recognition, hormonal confirmation, sustained glucocorticoid therapy, and family education remain the cornerstone of effective care to prevent complications and unnecessary surgical interventions. Strengthening local manpower needs by training paediatric endocrinologist, improving local laboratory capacity and implementing newborn screening would significantly improve diagnosis, treatment outcomes, quality of life and prognosis of CAH in Nigeria and similar environments.

Ethics: Ethical clearance was given by the Health Research Ethics committee of the University of Calabar Teaching Hospital (UCTH/HREC/2026/VOL.IV/007).

Conflict of interest: None

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REFERENCES

1. White PC, Speiser PW. Congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Endocr Rev. 2000 Jun; 21(3):245-91. doi: 10.1210/edrv.21.3.0398. Erratum in: Endocr Rev 2000 Oct; 21(5):550. PMID: 10857554.
2. Claahsen-van der Grinten HL, Speiser PW, Ahmed SF, Arlt W, Auchus RJ, Falhammar H, Flück CE, Guasti L, Huebner A, Kortmann BBM, Krone N, Merke DP, Miller WL, Nordenström A, Reisch N, Sandberg DE, Stikkelbroeck NMML, Touraine P, Utari A, Wudy SA, White PC. Congenital Adrenal Hyperplasia-Current Insights in Pathophysiology, Diagnostics, and Management. Endocr Rev. 2022 Jan 12; 43(1):91-159. doi: 10.1210/endrev/bnab016. PMID: 33961029; PMCID: PMC8755999.
3. Uslar T, Olmos R, Martínez-Aguayo A, Baudrand R. Clinical Update on Congenital Adrenal Hyperplasia: Recommendations from a Multidisciplinary Adrenal Program. J Clin Med. 2023 Apr 26; 12(9):3128. doi: 10.3390/jcm12093128. PMID: 37176569; PMCID: PMC10179176.
4. Witchel SF, Azziz R. Nonclassic congenital adrenal hyperplasia. Int J Pediatr Endocrinol. 2010; 2010:625105. doi: 10.1155/2010/625105. Epub 2010 Jun 30. PMID: 20671993; PMCID: PMC2910408.
5. Adriaansen BPH, Schröder MAM, Span PN, Sweep FCGJ, van Herwaarden AE, Claahsen-van der Grinten HL. Challenges in treatment of patients with non-classic congenital adrenal hyperplasia. Front Endocrinol (Lausanne). 2022 Dec 12; 13:1064024. doi: 10.3389/fendo.2022.1064024. PMID: 36578966; PMCID: PMC9791115.
6. Kurtoğlu S, Hatipoğlu N. Non-Classical Congenital Adrenal Hyperplasia in Childhood. J Clin Res Pediatr Endocrinol. 2017 Mar 1; 9(1):1-7. doi: 10.4274/jcrpe.3378. Epub 2016 Jun 29. PMID: 27354284; PMCID: PMC5363159.
7. Jha S, Turcu AF. Nonclassic Congenital Adrenal Hyperplasia: What Do Endocrinologists Need to Know? Endocrinol Metab Clin North Am. 2021 Mar; 50(1):151-165. doi: 10.1016/j.ecl.2020.10.008. Epub 2021 Jan 9. PMID: 33518183; PMCID: PMC7863575.
8. Speiser PW, Arlt W, Auchus RJ, Baskin LS, Conway GS, Merke DP, Meyer-Bahlburg HFL, Miller WL, Murad MH, Oberfield SE, White PC. Congenital Adrenal Hyperplasia Due to Steroid 21-Hydroxylase Deficiency: An Endocrine Society Clinical Practice Guideline. J Clin Endocrinol Metab. 2018 Nov 1; 103(11):4043-4088. doi: 10.1210/jc.2018-01865. Erratum in: J Clin Endocrinol Metab. 2019 Jan 1; 104(1):39-40. doi: 10.1210/jc.2018-02371. PMID: 30272171; PMCID: PMC6456929.
9. Osifo O, Nwashilli N. Congenital Adrenal Hyperplasia: The Challenges of Management in a Developing country. Afr Jnl Uro. [Internet]. 2008 Oct. 23 [cited 2025 Nov. 3]; 14(3):138-42. Available from: https://www.ajol.info/index.php/aju/article/view/8241
10. Oyenusi, Elizabeth Eberechi; Oduwole, Abiola Olufunmilayo; Okorie, Obasi Onwuka1; Adekoya, Adesola Olubunmi1; Nwaogu, Nwaoma Tochukwu1; Asafo-Agyei, Serwah Bonsu1. Congenital adrenal hyperplasia at the Lagos University Teaching Hospital: A 10-year review. Journal of Clinical Sciences 13(4): p 178-186, Oct–Dec 2016. | DOI: 10.4103/2468-6859.192291

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11. Navarro-Zambrana AN, Sheets LR. Ethnic and National Differences in Congenital Adrenal Hyperplasia Incidence: A Systematic Review and Meta-Analysis. Horm Res Paediatr. 2023; 96(3):249-258. doi: 10.1159/000526401. Epub 2022 Aug 16. PMID: 35973409.
12. Agboola-Abu CF, Aligwekwe PK, Olowu AO, Kuku SF. Congenital adrenal hyperplasia due to 11-hydroxylase enzyme deficiency in three siblings. A brief report. West Afr J Med. 1999 Apr-Jun; 18(2):80-6. PMID: 10504861.
13. Speiser PW, White PC. Congenital adrenal hyperplasia due to steroid 21-hydroxylase deficiency. Clinical Endocrinology (1998) 49, 411–417.
14. Eyong, M. E, Nsa, E. I, & Etuk, I. S. (2021). Pediatric Endocrinology Practice in Nigeria: Challenges and Way Forward. Nigerian Journal of Medicine, 29(4), 542–547. Retrieved from https://www.ajol.info/index.php/njm/article/view/204204.
15. Peltek Kendirci HN, Ünal E, Dündar İ, Buluş AD, Odabaşı Güneş S, Şıklar Z. Treatment and Follow-up of Congenital Adrenal Hyperplasia Due to 21-hydroxylase Deficiency in Childhood and Adolescence. J Clin Res Pediatr Endocrinol. 2025 Jan 10; 17(Suppl 1):12-22. doi: 10.4274/jcrpe.galenos.2024.2024-6-26-S. Epub 2024 Dec 23. PMID: 39713876;
16. Nygren U, Nyström HF, Falhammar H, Hagenfeldt K, Nordenskjöld A, Södersten M. Voice problems due to virilization in adult women with congenital adrenal hyperplasia due to 21-hydroxylase deficiency. Clin Endocrinol (Oxf). 2013 Dec; 79(6):859-66. doi: 10.1111/cen.12226. Epub 2013 May 21. PMID: 23600848.PMCID: PMC11730096.
17. Agrimonti G, Spaggiari G, Menabò S, Vezzani S, Magnani E, Frasoldati A, Granata A R, Santi D. Simple-Virilizing Congenital adrenal hyperplasia sustained by five mutations on the CYP21A2 gene. Biomed. Res. Ther. 2024; 11(7):6583-6591.
18. Wang LC, Poppas DP. Surgical outcomes and complications of reconstructive surgery in the female congenital adrenal hyperplasia patient: What every endocrinologist should know. J Steroid Biochem Mol Biol. 2017 Jan; 165(Pt A):137-144. doi: 10.1016/j.jsbmb.2016.03.021. Epub 2016 Mar 16. PMID: 26995108.

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