Case Report

Long-Term Growth and Pubertal Outcomes After Seven Years of Treatment for Peripheral Precocious Puberty in a Girl with Mccune–Albright Syndrome: A Case Report

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ABSTRACT

Background: McCune–Albright syndrome (MAS) is a rare sporadic disorder commonly associated with peripheral precocious puberty (PPP) in girls due to autonomous ovarian estrogen production. PPP often presents early, progresses unpredictably, and poses significant challenges to growth preservation and pubertal control.

Case summary: This is a report of the long-term growth and pubertal outcomes of an eight-year medical treatment course in a girl who presented at 15 months of age with breast and pubic hair development and cyclical vaginal bleeding. She had café-au-lait macules, markedly elevated estradiol levels with suppressed gonadotropins, advanced bone age, and pelvic ultrasonographic findings consistent with estrogen exposure, confirming PPP in the setting of MAS. Treatment with the aromatase inhibitor, anastrozole led to marked reduction in vaginal bleeding frequency and eventual cessation, stabilization of pubertal progression, and moderated bone age advancement. Anastrozole was discontinued after seven years of therapy when she was 8 years and nine months. Menarche occurred within two weeks of stoppage of therapy, and regular menstrual cycles ensued. By 16 years of age, the patient attained a near-final height of 153 cm, close to her mid-parental target height.

Conclusion: This case highlights the effectiveness of prolonged aromatase inhibitor therapy and sustained follow-up in optimizing growth and pubertal outcomes in MAS-associated PPP, particularly in resource-limited settings.

Keywords: McCune–Albright syndrome, peripheral precocious puberty, aromatase inhibitor, growth outcome

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INTRODUCTION

McCune-Albright syndrome (MAS) is a rare sporadic disorder caused by post-zygotic somatic gain-of-function (activating) mutations in the *GNAS* gene (guanine nucleotide-binding protein, alpha-stimulating activity polypeptide), which encodes the alpha subunit of G proteins, leading to constitutive activation of several G protein-coupled receptors (GPCRs) including the Luteinizing hormone (LH), follicle-stimulating hormone (FSH), Thyroid-stimulating hormone (TSH), Growth hormone-releasing hormone (GHRH), and adrenocorticotropic hormone (ACTH) receptors. [1] McCune-Albright Syndrome is a rare disease with an estimated prevalence between 1/100,000 and 1/1,000,000. [2] MAS is not inherited, and there are no reported cases of vertical transmission. There are no known genetic or environmental risk factors implicated, and the disease appears to occur in all ethnic groups. [3]

The classical triad of the disease comprises *café-au-lait* skin pigmentation, fibrous dysplasia of bone, and endocrine abnormalities. [1] The presence of two of these three classical signs in a patient establishes the clinical diagnosis. [1, 4] There are several hyper-functioning endocrinopathies seen in this disease occurring alone or in combination. They include gonadotropin-independent precocious puberty, growth hormone excess, non-autoimmune hyperthyroidism, hyperprolactinemia, or neonatal hypercortisolism. Among the endocrine abnormalities, peripheral precocious puberty (PPP) is the most common in girls. [2, 5]

Peripheral precocious puberty in MAS is driven by estrogen secretion from autonomous ovarian cysts and is independent of hypothalamic–pituitary activation. [6, 7] In the ovaries, this mutation causes **autonomous follicular cysts** that secrete estrogen irregularly and in high amounts, driving breast development, growth acceleration, and *vaginal bleeding at a very young age*. Therefore, MAS is characterized by episodic or recurrent vaginal bleeding, breast development, advanced skeletal maturation, and accelerated linear growth, often beginning in infancy. [8] Repeated estrogen exposure leads to rapid bone age advancement and early epiphyseal fusion; thus, affected girls are at high risk of short stature or compromised final adult height if they are not treated. [9] There is also associated parental anxiety and psychosocial stress on the child and parents necessitating treatment. [3]

Management of MAS-associated PPP remains challenging. Aromatase inhibitors and estrogen receptor modulators have been used with variable success, but evidence is largely derived from small case series, and long-term outcome data remain limited, especially in sub-Saharan Africa. [8, 9] Gonadotropin-releasing hormone analogues are ineffective unless secondary central precocious puberty (CPP) develops. [8, 9]

There are few case reports [10-12] of McCune-Albright syndrome in Africa and they were on children having majorly fibrous dysplasia and *café-au-lait* macules without emphasis on associated endocrinopathies. In addition, none of these cases had a longitudinal follow-up of the patients to evaluate for growth and pubertal outcome.

This report describes a girl with MAS who presented as a toddler with severe PPP and was followed for over a decade, highlighting long-term growth and pubertal outcomes following prolonged aromatase inhibitor therapy.

This case is notable for the exceptionally long duration of follow-up and therapy, spanning early toddler through adolescence, demonstrating the feasibility and benefit of sustained aromatase inhibitor use in a resource-constrained setting. It adds to the limited long-term data on growth and pubertal outcomes and underscores the importance of early diagnosis, individualized therapy, and prolonged follow-up in optimizing outcomes for girls with MAS-associated peripheral precocious puberty.

CASE SUMMARY

History

A 15 months old girl presented at the Paediatric Endocrinology clinic of the University of Calabar Teaching Hospital on 30 November 2010. She presented with a seven months history of breast and pubic hair development and a five months history of cyclical vaginal bleeding. There was no history of trauma, sexual assault, or exogenous estrogen exposure. There was no history of bone pains. The parents initially sought spiritual and traditional remedies before hospital presentation.

She was the third child of non-consanguineous marriage, with two older healthy male siblings. Pregnancy and delivery were uneventful, and there was no family history of precocious puberty. The mother attained menarche at 13 years of age. Parents have tertiary level of education and are employed in the public service.

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Physical examination: She was well nourished, afebrile, anicteric, and not pale. She weighed 10.2 kg and measured 81cm in length (at 90th percentile on CDC growth chart). Mid-parental height was calculated as 160.5 cm and target height was 160.5 ± 8 cm. She had a large *café-au-lait* macule with irregular borders ("coast of Maine") on the left lower back measuring 5 x 4 cm and a smaller one on the dorsum of the left hand and forearm. Breast and pubic hair development were both Tanner stage II. The labia majora and minora were normal, with an intact hymen. No signs of sexual abuse or trauma to the area or presence of foreign body.

Investigations

A complete blood count was normal. Abdomino-pelvic ultrasonography showed a bulky uterus with visible fundus and cervix with well delineated endometrium, and bilateral ovarian follicles. Hormonal evaluation demonstrated markedly elevated estradiol, 99 pg/ml (n= < 10) with low to suppressed gonadotropins, LH 3.8 µIU/ml (n = 5.0-20), FSH 4.4 µIU/ml (n= < 10), consistent with peripheral precocious puberty. Prolactin was 25ng/ml (n= 8.39-20.15). Bone age was 4 years, advanced for age. Cranial CT scan showed no intracranial or pituitary mass. Thyroid function tests revealed normal T3, T4 and TSH. The IGF-1 and growth hormone levels were not done because of cost.

Diagnosis: McCune–Albright syndrome with peripheral precocious puberty.

Treatment and follow-up: She was commenced on anastrozole 1 mg daily, six months after presentation. The delay in starting treatment was because of the high cost of investigations which parents were paying from out of pocket and the fact that Anastrazole was not available in Nigeria at the time and was sourced from outside of Nigeria. A repeat hormonal profile after 3 months of starting anastrozole showed a reduced estradiol level of 25 pg/ml (n= <10), LH 5.0 µIU/m (5.0-20), FSH 7.5 µIU/m (n= < 20). Estradiol was not suppressed completely throughout the treatment period. However, there was no vaginal bleeding again. The serial LH, FSH and estradiol levels over the period of treatment are shown in Table I. The bone age at this time was 5 years. Letrozole, a newer third generation aromatase inhibitor was considered to see if the estradiol will be suppressed but the cost was prohibitive for the parents and therefore, we continued with the Anastrazole therapy.

Table I: Serial Hormonal profile in the course of treatment

Date	Age of child	Duration on treatment	LH (5-20 µIU/ml)	FSH (< 20 µIU/ml)	Estradiol (< 10 pg/ml)
14/12/2010	15 months	Nil	2.5	5.0	66
12/04/2011	19 months	Nil	3.8	4.4	99
29/09/2011	2 years	3 months	5.0	7.5	25
26/05/2015	5 years	$3^{1/2}$ years	7.4	2.4	72
19/07/2016	6 years	5 years	2.3	9.7	50
26/05/2017	7 years	6 years	1.5	4.4	99

Following treatment initiation, the frequency and volume of vaginal bleeding progressively reduced, with intermittent scanty episodes monthly and eventual cessation 15 months after start of treatment. Pubertal progression slowed, though not completely arrested.

The patient's longitudinal linear growth pattern, from post-infancy period through adolescence while on Anastrazole therapy is illustrated in figure I. The curve demonstrates accelerated early growth prior to treatment initiation, followed by sustained linear growth with moderated velocity during prolonged anastrozole therapy.

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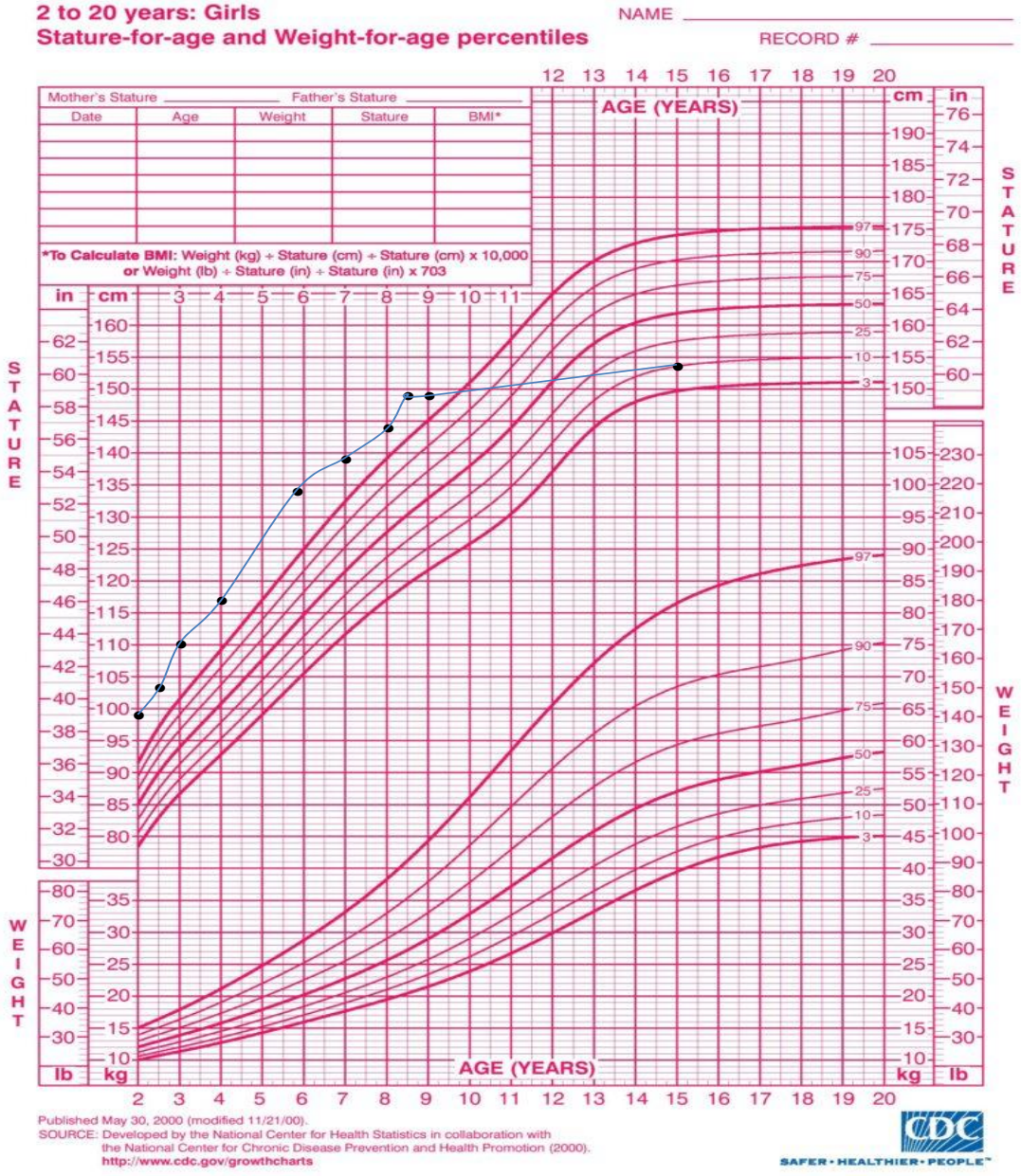

Figure I: Linear growth (Height) vs age during treatment.

Also, Height standard deviations scores (SDS), plotted using CDC female height-for-age references, were markedly elevated in early childhood, peaking above +3 SDS, before gradually declining during adolescence as **bone age advanced with epiphyseal maturation** (figure II). Despite this, the patient attained a near-final height SDS of approximately -1.5 (153cm).

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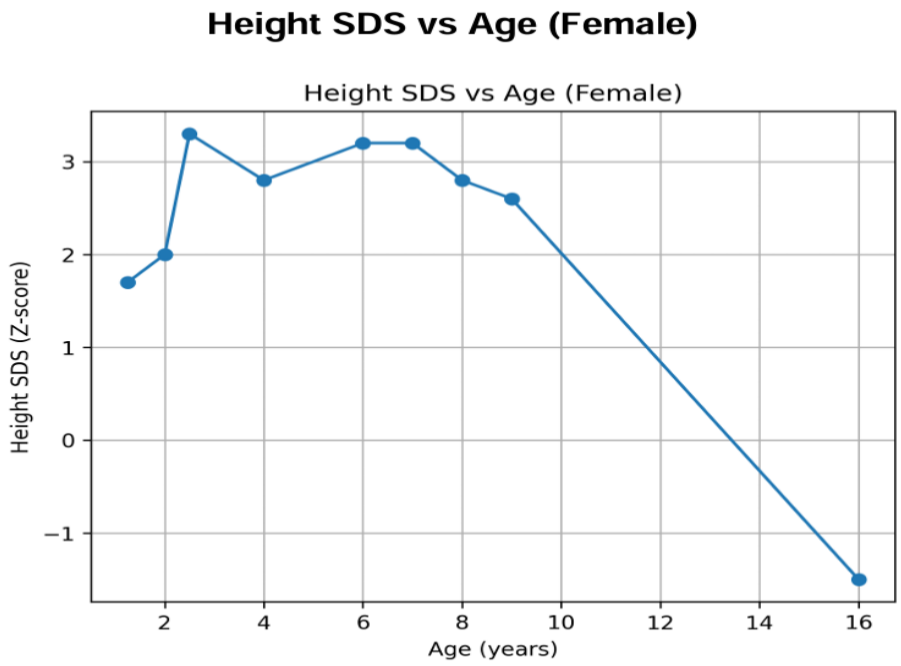
Figure II: Longitudinal height standard deviation score (SDS) plotted against age.



The approximate Height SDS at key time points of age (calculated using CDC female reference values of height-for-age) with corresponding bone ages and pubertal changes is shown in Table II. The Tanner staging for Breast (B1-B5) and pubic hair (P1-P5) for the corresponding longitudinal ages is also shown in Table II.

Table II: The height, bone age and pubertal stages for the corresponding ages

Age (years)	Height (cm)	Approx. Height SDS	Bone age (years)	Tanner Staging (Breast)	Tanner Staging (Pubic Hair)
1.25	81	+1.7	4	B2	P2
2.0	99	+2.0	-	-	-
2.5	103	+3.3	6	B3	P2
4.0	117	+2.8	-	-	-
6.0	134	+3.2	11	B3	P3
7.0	139	+3.2	12	B3	P4
8.0	144	+2.8	13.5	B4	P4
9.0	149	+2.6	14	B4	P4
16.0	153	−1.5	16	B5	P5

Also, the bone age progressed from the initial 4 years at presentation to 11 years at 6 years of chronological age and then stabilized gradually and was 14 years by 9 years of chronological age (Table II).

There was no reported history of joint or bone pain or dental pains. However, there was a history of a pruritic papular and scaly rash on the face and neck, six months after commencement of therapy, which resolved within two weeks with chlorpheniramine medication.

Anastrozole was discontinued after seven years of therapy in May 2018. Menarche occurred two weeks after discontinuation of therapy. This was followed by regular monthly menstrual cycles lasting 5-7 days.

At age of 16 years, she had attained a height of 153 cm and a weight of 52 kg, with Tanner stage V for both breast and pubic hair. There was no history of facial or jaw swelling. There was no symptom suggestive of malocclusion, pressure effects or functional impairment

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in the oral cavity. Dental examination reveals no facial asymmetry, scars or craniofacial deformities, no tooth displacement, spacing, rotation attributable to underlying osseous pathology, thus ruling out cranio-facial fibrous dysplasia. There was no history of bone pains in any part of the body to warrant skeletal X-rays or bone scanning. Bone age was 17 years and the abdomino-pelvic ultra sound showed essentially normal ovaries, fallopian tubes, and uterus. All abdominal organs were normal.

Discussion

This report provides rare long-term follow-up from 15 months to late adolescence in a girl with McCune–Albright syndrome presenting with peripheral precocious puberty. The key findings in this case were the challenge in the diagnosis of this rare disease of McCune-Albright syndrome, the manifestation of the disease as peripheral precocious puberty, the growth (skeletal) and pubertal advancement associated with the disease, treatment challenges, and the long-term growth and pubertal outcomes that were not severely affected.

This patient presented unusually early, at 15 months of age, with recurrent vaginal bleeding and rapid pubertal changes. Though LHRH stimulation tests was not done to differentiate between PPP and CPP because of high cost, the markedly elevated estradiol level, suppressed gonadotropins, advanced bone age, and typical *café-au-lait* macules strongly supported the diagnosis of MAS-associated PPP. Neuroimaging carried out excluded central causes (CPP), reinforcing the peripheral origin of puberty (PPP) in the child. The component of fibrous dysplasia (FD) was not seen in this patient throughout the course of the illness. Both dental and skeletal complaints were not reported and dental examinations were essentially normal. This finding is not surprising as the phenotypic spectrum of MAS is considerably wider and more complex, sometimes with one of the components being absent. [4] The new diagnostic criteria for MAS are the combination of FD and one or more extra-skeletal features, OR the presence of two or more extra-skeletal features, FD not always being associated as previously described. [13]

Peripheral precocious puberty (PPP) is the most frequent endocrine manifestation of McCune-Albright syndrome (MAS) in girls and often presents in infancy or early childhood. [2, 5] The patient presentation at **15 months of age** with breast development, pubic hair growth, and recurrent cyclical vaginal bleeding were consistent with precocious puberty. The hormonal evaluation showed **markedly elevated estradiol levels with suppressed gonadotropins (LH and FSH),** confirming **gonadotropin-independent (peripheral) precocious puberty.** Pelvic ultrasonography demonstrated a bulky uterus and ovarian follicles, indicating significant estrogen exposure. The bone age was markedly advanced (4 years at a chronological age of 15 months), indicating accelerated skeletal maturation and a high risk of compromised adult height if untreated. The neuroimaging excluded central nervous system pathology, supporting the diagnosis of MAS-associated peripheral precocious puberty.

The treatment of MAS-associated PPP is particularly challenging because the autonomous and intermittent nature of estrogen secretion leads to episodic vaginal bleeding and unpredictable pubertal progression. [13, 14] Also, if treatment is not given, repeated estrogen exposure accelerates skeletal maturation and significantly compromises eventual adult height. [2, 14]

Different treatments have been used to manage MAS, and they all aimed at reducing the recurrence of ovarian cysts, counteracting the manifestations of estrogen excess, and arresting the advancement of bone injuries in those who develop fibrous dysplasia. [15] There is varying degree of effectiveness with each treatment. In the past, medroxyprogesterone acetate (MPA) injections were given weekly to inhibit vaginal bleeding. However, it did not demonstrate an effect on countering bone maturation. [15] Tamoxifen, a selective estrogen receptor modulator, has also been used and it causes a reduction in vaginal bleeding events and an adequate control of growth rate and bone maturation, although it had no effect on ovarian volume and uterine size. [15] The newer treatment uses third generation aromatase inhibitors which reduces the rate of vaginal bleeding, the growth rate, and the advancement of bone age. [15] If the patients are untreated, they may end up with a short final adult height as bone age advancement lead to early epiphyseal closure. [9] In this case report, the patient was treated with anastrazole therapy for 7 years. The patient ultimately attained a near-final height of 153 cm, which is close to her mid-parental target height of 160.5 cm. The final height SDS of approximately **-1.5** still represents a **reasonable height preservation** given the severity and very early (infancy) onset of PPP in our patient. Estrada *et al* [16] documented similar growth outcomes with adult height Z-scores ranging from -1.5 to 1.7, which were increased in comparison with untreated historical controls. In his cohort of patients, letrazole, a newer

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third generation aromatase inhibitor was used in the treatment of their patients while we used anastrazole, an older third generation aromatase inhibitor. Prolonged anastrozole therapy was associated with a gradual advancement of bone age. Bone age was markedly advanced at presentation, exceeding chronological age by more than two years in infancy, and continued to progress disproportionately during early childhood. Although advancement persisted during treatment, the slope of progression appeared moderated over time, reflecting partial control of estrogen-driven skeletal maturation during prolonged aromatase inhibitor therapy (anastrazole). [16]

Apart from the good growth outcome, prolonged anastrozole therapy was also associated with a reduction in the frequency and severity of vaginal bleeding, and stabilization of pubertal progression in our patient. Although pubertal signs continued to progress slowly, the tempo was moderated, allowing continued linear growth over several years. The prompt occurrence of menarche following discontinuation of anastrozole suggests restoration of normal hypothalamic–pituitary–ovarian axis function after prolonged suppression of estrogen synthesis, which has been described in previous reports. [13] The attainment of full pubertal Tanner stages of puberty and the normal pubertal organs seen on abdomino-pelvic ultrasound at 16 years of age is an indication that treatment with anastrazole does not affect final pubertal attainment. It is said that fertility is preserved in a majority of patients and therefore the hope that this will apply to this girl.

The patient developed an adverse skin reaction, a pruritic, papular and scaly rash six months after commencement of anastrazole which resolved within two weeks with an anti-histamin medication. We are not too sure if this was a reaction to anastrazole or just dermatitis from another cause. However, reaction to Anastrazole therapy, though rare, has been reported with onset from less than 5 days to 6 months; and the reaction was treated with anti-histamine or steroids. [17]

Strength of this report

This report provides rare long-term follow-up from infancy to late adolescence in a girl with McCune–Albright syndrome presenting with peripheral precocious puberty. It includes comprehensive clinical, hormonal, and auxological data, with objective assessment using height SDS and growth curves. The case also provides a practical experience with use of prolonged aromatase inhibitor (anastrozole) therapy in a resource-constrained setting and documents menstrual outcomes after treatment withdrawal, providing insight into recovery of reproductive function.

Limitations

As a single case, findings are not generalizable. Genetic confirmation was not performed, although diagnosis was clinically robust. Evidence for anastrozole in MAS is limited compared with letrozole. Some longitudinal data gaps exist, and growth chart plotting involved minor approximation. Potential confounders such as treatment adherence and intermittent ovarian activity could not be fully assessed.

Implications of the Findings

This case suggests that early and sustained therapy in MAS-associated peripheral precocious puberty can moderate skeletal maturation and preserve growth potential, even with infancy onset. It supports the practical utility of aromatase inhibitors, including anastrozole, in resource-constrained settings. The findings also show that pubertal function can normalize after treatment, as evidenced by timely menarche following drug cessation. Overall, the report highlights the importance of early diagnosis, long-term follow-up, and individualized management, while underscoring the need for larger prospective studies to guide optimal therapy.

CONCLUSION

Long-term medical management of peripheral precocious puberty in McCune–Albright syndrome can lead to satisfactory growth and pubertal outcomes when treatment is individualized and closely monitored. This case highlights the feasibility and benefit of extended follow-up and contributes to the limited body of literature on long-term outcomes in MAS-associated PPP.

Declarations

Authors' contribution: Michael Eteng Eyong conceived and designed the study and also wrote up the manuscript; Edu Michael Eyong first saw the patient and referred to the Endocrinologist and extracted the data; Ekaette Itam Nsa involved in the follow-up care and design of the study; Nonso Emmanuel Onyia and Samuel Archibong Efanga are the Dental surgeon and Radiologist who are involved in the follow–up care of patient contributed in overview of the manuscript in the area of their specialty; Maxwell Udo Anah proof read

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and edited the manuscript. All authors reviewed and approved the final manuscript.

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Disclosure in AI use: During the preparation of this manuscript, ChatGPT (OpenAI) was used to assist in generating the diagram of longitudinal height standard deviation score (SDS) plotted against age. The AI tool was not used for study design, data collection, data analysis or formulation of the study conclusions. The authors verified the scientific accuracy of the figure, made all necessary revisions, and take full responsibility for the final manuscript.

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