Case Report

Idiopathic Central Precocious Puberty in a 6-Year-Old Girl with Advanced Secondary Sexual Characteristics: A Case Report

Chidinma M. Nwogu[1], Sunday I. Omisakin[2], Aloy O. Ugwu [1,3], Abraham O. Abati[1], George N. Ejiole[1], James Iniso[1], Osato F. Giwa-Osagie[2]

[1]*Assisted Conception Unit, Kingswill Specialist Hospital, Lagos, Nigeria*
[2]*Department of Obstetrics and Gynaecology, Lagos University Teaching Hospital, Nigeria*
[3]*Department of Obstetrics and Gynaecology, 68 Nigerian Army Reference Hospital, Yaba, Lagos, Nigeria*

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Corresponding author's email: okeyugwu92@gmail.com

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ABSTRACT

Central precocious puberty is an uncommon but important paediatrics endocrine condition that requires timely recognition and evaluation to prevent adverse auxological and psychosocial outcomes. We present the case of a 6-year-old girl with a 10-month history of progressive breast enlargement and pubic and axillary hair development. Clinical examination revealed markedly advanced secondary sexual characteristics, consistent with Tanner stage IV maturation. Endocrine evaluation demonstrated pubertal-range gonadotropin concentrations, and dynamic testing with a gonadotropin-releasing hormone agonist showed a pubertal luteinizing hormone response, supporting activation of the hypothalamic-pituitary-gonadal axis. Bone age assessment demonstrated advancement beyond chronological age, while pelvic ultrasonography showed pubertal uterine morphology. Brain magnetic resonance imaging revealed no hypothalamic-pituitary or other intracranial lesion. Following clinical, biochemical, radiological, and imaging assessment, a diagnosis of idiopathic central precocious puberty was made. The patient's parents were counselled on the diagnosis, treatment options, expected benefits, and need for follow-up, and treatment with a gonadotropin-releasing hormone analogue was commenced. This case underscores the importance of a structured approach to children presenting with early progressive pubertal development, including careful clinical assessment, bone age evaluation, endocrine testing, pelvic imaging, and neuroimaging where indicated.

Keywords: Central Precocious Puberty; Idiopathic Precocious Puberty; GnRH Agonist Stimulation Test; Bone Age; Tanner Stage IV; Nigerian Child

Introduction

Precocious puberty in girls is conventionally defined as the appearance of secondary sexual characteristics before 8 years of age or at an age approximately 2 to 2.5 standard deviations below the expected population mean [1].

It may be classified as central precocious puberty, caused by premature activation of the hypothalamic-pituitary-gonadal axis, or peripheral precocious puberty, resulting from gonadotropin-independent sex steroid production or exposure [2,3]. Among girls, central precocious puberty is more often idiopathic than pathological, although exclusion of central nervous system lesions remains an important part of evaluation, especially in younger children and those with rapid progression [2,3,4].

The diagnosis of central precocious puberty is based on a combination of clinical progression, pubertal staging, skeletal maturation, hormonal profile, and, where indicated, dynamic stimulation testing and imaging [3,4]. Bone age advancement and a pubertal luteinizing hormone response to gonadotropin-releasing hormone stimulation strongly support hypothalamic-pituitary-gonadal axis activation.3 Brain magnetic resonance imaging is frequently performed to exclude structural intracranial causes [4,5].

We present the case of a 6-year-old girl with advanced secondary sexual characteristics, elevated gonadotropins, advanced bone age, pubertal response to gonadotropin-releasing hormone agonist stimulation, and a normal brain magnetic resonance imaging study, consistent with idiopathic central precocious puberty. This case report was prepared in accordance with the CARE (CAse REport) guidelines to ensure completeness and transparency in reporting.

Case Presentation

A 6-year-old girl presented with her parents to our paediatrics unit with concerns of early development of breast, pubic and axillary hair. This was of insidious onset and has remained progressive since onset 10 months ago. The child had no history of headaches, visual disturbance, behavioural change, or neurological impairment. There was no antecedent history of head trauma, neurosurgery, gelastic seizures, encephalitis, meningitis, central nervous system irradiation or anomaly, or steroid exposure. She had no history of birth injury or congenital anomalies, and her developmental milestones were age appropriate. There were no symptoms suggestive of hyperthyroidism or hypothyroidism, such as heat intolerance, and no galactorrhea. No similar history was noted among her parents or siblings.

Physical examination revealed advanced secondary sexual development, with breast, pubic hair, and axillary hair maturation consistent with Tanner stage IV. This degree of pubertal development was significantly premature for her chronological age. She weighed 38 kg and had a height of 135 cm. Abdominal examination was unremarkable. Endocrine evaluation showed basal follicle-stimulating hormone and luteinizing hormone concentrations in the pubertal range, whereas serum estradiol was within normal reference limits. Bone age assessment demonstrated advancement beyond chronological age, with a bone age of nine years compared with the patient's chronological age of six years. This finding supported progressive exposure to sex steroids and was relevant in assessing the potential risk to final adult height. Magnetic resonance imaging of the brain showed no lesion in the hypothalamic-pituitary axis.

A gonadotropin-releasing hormone agonist stimulation test was subsequently performed and elicited a pubertal gonadotropin response, with a peak LH-to-FSH ratio >0.66 confirming activation of the hypothalamic-pituitary-gonadal axis. Other potential causes of precocious puberty were actively considered. There was no history of exposure to exogenous oestrogen, and there were no symptoms or signs suggestive of central nervous system pathology. Thyroid function tests were assessed to exclude severe primary hypothyroidism, while adrenal androgen-related causes were considered in view of the presence of pubic and axillary hair. There were no clinical features suggestive of McCune-Albright syndrome, including café-au-lait macules, and no signs of virilization. Pelvic ultrasonography showed pubertal uterine morphology and increased uterine and ovarian volumes, with no ovarian cyst or tumour identified. Brain MRI showed no hypothalamic or pituitary lesion. Overall, the clinical, biochemical, (DHEAS, Serum 17-hydroxyprogesterone, TSH, free T4, androstenedione/testosterone, and hCG) radiological, and imaging findings supported idiopathic central precocious puberty rather than peripheral precocious puberty or an isolated benign pubertal variant

The patient's parents received counselling on the diagnosis, therapeutic options, and anticipated benefits of treatment, which included optimization of final adult height and mitigation of psychosocial challenges related to premature pubertal development. The need for strict adherence to follow-up was emphasized, including serial clinical assessments and periodic bone age evaluation. After counselling, treatment with a gonadotropin-releasing hormone

analogue was initiated, and the patient continued under follow-up at the time this report was prepared.

Ethics Approval

Ethics approval was obtained from 68 Nigerian Army Reference Hospital Yaba, ADM/NARHY/OG/781A. In accordance with institutional policy, written informed consent for publication of the anonymized clinical details and accompanying images was obtained from the patient's parent/legal guardian.

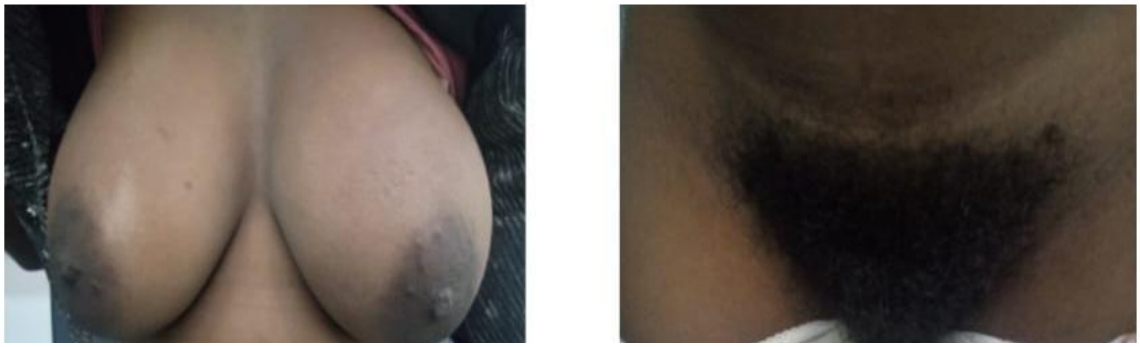

Figure 1. External genital and chest examination showing advanced secondary sexual characteristics, with breast and pubic hair development corresponding to Tanner stage IV.

The GnRH agonist stimulation test demonstrated a pubertal pattern of gonadotropin secretion. LH increased from 2.9 mIU/mL at baseline to a peak of 9.7 mIU/mL at 120 minutes, while FSH increased from 4.8 mIU/mL to a peak of 5.6 mIU/mL. The resulting peak LH-to-FSH ratio of approximately 1.7 supports hypothalamic-pituitary-gonadal axis activation, consistent with central precocious puberty. The baseline estradiol level of 17.87 pg/mL was interpreted in conjunction with the patient's advanced Tanner staging, bone age advancement, and pubertal pelvic ultrasound findings. Table 1.

Table 1. Baseline and stimulated gonadotropin concentrations following administration of the GnRH agonist leuprolide acetate (20 μg/kg) in a 6-year-old girl with suspected central precocious puberty. The progressive rise in luteinizing hormone following stimulation is consistent with pubertal activation of the hypothalamic-pituitary-gonadal axis.

Hormone	Starting time	30 minutes	60minutes	90minutes	120 minutes
FSH (mUI/mL)	4.8	5	5.5	5.6	5.4
LH (mUI/mL)	2.9	4	6.8	9	9.7
Oestrogen (pg/mL)	17.87				

#Baseline estradiol was 17.87 pg./mL. Samples were collected at 0, 30, 60, 90, and 120 minutes.

Discussion

This case demonstrates a classic but clinically important presentation of idiopathic central precocious puberty in a young girl with markedly advanced sexual maturation.1 The diagnosis was supported by the combination of progressive pubertal signs, biochemical evidence of gonadotropin activation, advanced skeletal maturation, and a pubertal response on gonadotropin-releasing hormone agonist stimulation, while neuroimaging excluded an intracranial lesion.

In girls, breast development before 8 years of age should prompt evaluation for precocious puberty, especially when the findings are progressive or accompanied by pubic hair development, growth acceleration, or advanced bone age [5]. The presence of Tanner stage IV breasts and pubic and axillary hair in a 6-year-old strongly suggests true pubertal progression rather than a benign isolated variant such as premature thelarche or premature adrenarche.

In the present case, the patient was 6 years old at presentation and had a 10-month history of symptoms, a pattern similar to that described in earlier reports such as that of Helyaich et al. [6].

Similarly, previous reports from Nigeria have described precocious puberty in girls as young as 24 and 30 months of age although one of those were associated with oestrogen producing tumour of the ovary [7,8].

The elevated basal luteinizing hormone and follicle-stimulating hormone levels in this patient favored central activation of puberty. Dynamic testing further clarified the diagnosis [2,3]. A pubertal gonadotropin response following gonadotropin-releasing hormone agonist stimulation is widely regarded as strong evidence of central precocious puberty. In this case, the stimulation test was particularly helpful in establishing the diagnosis with confidence.

The bone age radiograph showed advancement beyond chronological age, which is consistent with sustained or progressive pubertal activation [4]. Bone age assessment remains valuable because it helps distinguish benign variants from progressive precocious puberty and provides insight into the possible effect on adult height potential [5,6]. Progressive sex steroid exposure accelerates epiphyseal maturation, and untreated cases may ultimately compromise final adult stature.

Brain magnetic resonance imaging was normal in this patient. This finding supports a diagnosis of idiopathic central precocious puberty rather than a neurogenic cause. In girls, especially those older than 6 years, idiopathic central precocious puberty is more common than central nervous system pathology. Nevertheless, imaging remains an important component of evaluation because hypothalamic or pituitary lesions must be excluded in appropriately selected patients [4, 5]

This case also underscores a broader clinical point: the diagnosis of central precocious puberty is best made through integration of clinical, radiologic, and endocrine findings rather than reliance on any single laboratory marker. Although estradiol was normal, the overall pattern clearly supported early activation of the hypothalamic-pituitary-gonadal axis. The case therefore illustrates the importance of a structured and comprehensive approach in young children presenting with advanced pubertal development.

Conclusion

We report a case of idiopathic central precocious puberty in a 6-year-old girl who presented with Tanner stage IV breast, pubic, and axillary hair development. The diagnosis was supported by elevated gonadotropin levels, advanced bone age, and a pubertal response to gonadotropin-releasing hormone agonist stimulation, while brain magnetic resonance imaging was normal.

Learning Points

1. Central precocious puberty should be suspected in girls younger than 8 years with progressive breast development and associated pubic or axillary hair.
2. Tanner stage IV secondary sexual characteristics at 6 years of age strongly suggest pathologic pubertal advancement rather than a benign isolated variant.
3. Normal estradiol concentration does not rule out central precocious puberty.
4. Advanced bone age supports progressive biological pubertal activation and may indicate risk to final adult height.
5. A pubertal luteinizing hormone and follicle-stimulating hormone response to gonadotropin-releasing hormone agonist stimulation is highly supportive of central precocious puberty.
6. Normal brain magnetic resonance imaging in this setting supports a diagnosis of idiopathic central precocious puberty.

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References

1. Yoon DY, Kim JH. An 11-month-old girl with central precocious puberty caused by hypothalamic hamartoma. Ann Pediatr Endocrinol Metab. 2016;21(4):235-239. doi:10.6065/apem.2016.21.4.235

2. Dallos-Lara MF, Mendoza V, Rojas A. Precocious puberty due to congenital adrenal hyperplasia: case report. Rev Fac Med. 2020;68(1). doi:10.15446/revfacmed.v68n1.72674
3. Joo EY, Yoo MJ, Kim SJ, Jang W, Lee JE. Case report: development of central precocious puberty in a girl with late-diagnosed simple virilizing congenital adrenal hyperplasia complicated with Williams syndrome. Front Endocrinol (Lausanne). 2024;15:1352552. doi:10.3389/fendo.2024.1352552
4. Castro C, Machado Morais J, Correia AL, Espada F. Hypothalamic hamartoma: a cause of precocious puberty. BMJ Case Rep. 2023;16(3):e254429. Published 2023 Mar 24. doi:10.1136/bcr-2022-254429
5. Suh J, Choi Y, Oh JS, et al. Management of Central Precocious Puberty in Children with Hypothalamic Hamartoma. Children (Basel). 2021;8(8):711. Published 2021 Aug 18. doi:10.3390/children8080711
6. Helyaich A, Elqadiry R, El Fakiri K, Rada N, Draiss G, Bouskraoui M. Case Report of Central Precocious Puberty in a Female: Role of Environmental Pollutants? Clin Med Insights Case Rep. 2020;13:1179547620933585. Published 2020 Jul 9. doi:10.1177/1179547620933585
7. Oluwayemi IO, Afolabi AA, Adeniji EO, Ayeni TO. Precocious puberty in a 24 month old Nigerian girl. Niger J Paediatr. 2024;47(4):358-360. doi:10.4314/njp.v47i4.10
8. Oni T, Adekoya A, Elizabeth G, Nwadiokwu J, Okebalama V, Anifowose O. Pseudo-precocious puberty secondary to juvenile granulosa cell tumour in a 30-month-old female toddler: a case report. Babcock Univ Med J. 2024;7(2):108-113. doi:10.38029/babcockuniv.med.j.v7i2.560