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Precocious Puberty Beyond the Common Forms: A Moroccan Pediatric Case Series

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Abstract

Objectives:

To describe the clinical, etiological, therapeutic, and evolutionary characteristics of children with precocious puberty treated at a Moroccan tertiary center.

Materials and Methods:

A retrospective descriptive study of 12 children with precocious puberty followed at a Moroccan tertiary pediatric endocrinology unit between 2022 and 2026. Clinical, auxological, hormonal, radiological, therapeutic, and follow-up data were extracted from medical records.

Results:

The cohort included 10 girls (83.3%) and 2 boys (16.7%). Mean age at first evaluation was 67.6 ± 18.4 months (range, 39–108); it was 74.6 ± 19.4 months in central precocious puberty and 62.6 ± 17.2 months in peripheral forms. Five children (41.7%) had central precocious puberty and seven (58.3%) had peripheral precocious puberty. Breast development was documented in 9/10 girls (90%), pubic or axillary hair in 9/11 patients with available data (81.8%), premature body odor in 10/12 (83.3%), and vaginal bleeding or menstruation in 3/10 girls (30%). Four patients (33.3%) had virilization; the two girls with genital ambiguity were Prader stage III. Bone age was advanced at the first available assessment in 10/12 patients (83.3%). Triptorelin was used in seven patients, while congenital adrenal hyperplasia was managed with etiology-specific glucocorticoid therapy. Follow-up showed regression or partial regression in five patients, stabilization/non-progression in six, and age-appropriate pubertal progression in one adolescent with congenital adrenal hyperplasia.

Conclusion: Causes of early puberty are variable. Idiopathic forms were predominant but organic and adrenal causes were also frequent, highlighting the need for a complete clinical, hormonal and radiological assessment.

Keywords: Precocious puberty; Central precocious puberty; Peripheral precocious puberty; Congenital adrenal hyperplasia; Pediatric.

INTRODUCTION

Precocious puberty is the development of secondary sexual characteristics in girls before the age of 8 years and in boys before the age of 9 years [1]. It may accelerate growth and bone maturation, leading to premature epiphyseal fusion, reduced adult height, and psychosocial difficulties.

Central precocious puberty is caused by early activation of the hypothalamic-pituitary-gonadal axis. In girls it is often idiopathic but central nervous system lesions may be implicated. Peripheral precocious puberty is due to gonadotropin-independent sex-steroid production and may be associated with congenital adrenal hyperplasia (CAH), McCune–Albright syndrome, gonadal or adrenal tumors, severe hypothyroidism or exogenous hormone exposure [1,3,4].

Differentiation of these mechanisms requires clinical evaluation, Tanner staging, assessment of growth and bone age, hormonal studies, and directed imaging.

Given the scarcity of data in North Africa, the aim of this study was to describe the clinical, hormonal, radiological, etiological, therapeutic and evolutionary profile of 12 children with precocious puberty followed in a Moroccan tertiary center.

Materials and Methods

We conducted this retrospective, descriptive, single-center case series in the Pediatric Endocrinology Unit of the Department of Pediatrics of the Hassan II University Hospital of Fez, Morocco. Medical records of children followed in the unit between 2022 and 2026 were reviewed.

Children presenting with secondary sexual characteristics before eight years in girls or nine years in boys and having sufficient clinical, hormonal, and radiological data to establish or orient the underlying mechanism were included. Patients with incomplete records or insufficient follow-up were excluded.

Demographic, clinical, auxological, hormonal, radiological, etiological, therapeutic, and follow-up data were extracted from the medical records using a standardized form. Variables included age, sex, age at symptom onset, Tanner stage, breast and pubic-hair development, vaginal bleeding/menstruation, body odor, acne, genital virilization and Prader stage when applicable, anthropometric measurements, BMI, growth pattern, bone age, LH, FSH, estradiol, testosterone, DHEAS, 17-hydroxyprogesterone, pelvic or testicular ultrasonography, hypothalamic-pituitary MRI, diagnosis, treatment, and clinical evolution. Serial growth velocity and repeat bone age were analyzed when sufficiently documented.

Statistical analysis was performed using IBM SPSS Statistics, version 26.0. Categorical variables were reported as numbers and percentages. Continuous variables were summarized as means ± standard deviations and ranges. No inferential analysis was performed because of the small sample size.

Written informed consent for the use of anonymized clinical data was obtained from the parents or legal guardians of all patients. Data were anonymized to preserve confidentiality.

Results

Patient Characteristics

The cohort included 10 girls (83.3%) and 2 boys (16.7%), with a female-to-male ratio of 5:1. Mean age at first evaluation was 67.6 ± 18.4 months (range, 39–108 months). Mean age was 74.6 ± 19.4 months (range, 60–108) in the central group and 62.6 ± 17.2 months (range, 39–86) in the peripheral group. The youngest patient was 39 months old. Age at onset of the first pubertal manifestations ranged from approximately 2 to 7 years.

Central precocious puberty was diagnosed in five patients (41.7%), whereas seven (58.3%) had peripheral precocious puberty. Three central cases were idiopathic, one was associated with a hypothalamic hamartoma, and one with a pituitary adenoma. Four peripheral cases had congenital adrenal hyperplasia due to 11β-hydroxylase deficiency; one of these was additionally associated with mosaic 47,XYY

syndrome. The remaining three peripheral cases were non-progressive or idiopathic after exclusion of an identifiable central, adrenal, gonadal, or tumoral cause.

Characteristic	n (%) or value
Total patients	12
Girls	10 (83.3%)
Boys	2 (16.7%)
Mean age at first evaluation	67.6 ± 18.4 months (39–108)
Central precocious puberty	5 (41.7%)
Mean age, central group	74.6 ± 19.4 months (60–108)
Peripheral precocious puberty	7 (58.3%)
Mean age, peripheral group	62.6 ± 17.2 months (39–86)
Idiopathic central precocious puberty	3 (25%)
Idiopathic/non-progressive peripheral forms	3 (25%)
CAH due to 11β-hydroxylase deficiency	4 (33.3%)
Hypothalamic hamartoma	1 (8.3%)
Pituitary adenoma-associated precocious puberty	1 (8.3%)
Mosaic 47,XYY syndrome	1 (8.3%)

Table 1. General, age, and etiological characteristics of the study population

Clinical and Paraclinical Findings

Breast development was documented in 9 of 10 girls (90.0%). Pubic or axillary hair was present in 9 of 11 patients with documented data (81.8%). Premature body odor was reported in 10 of 12 patients (83.3%), whereas acne was not documented in any patient. Vaginal bleeding or menstruation occurred in 3 of 10 girls (30.0%). Virilization or genital enlargement was present in four patients (33.3%). The two girls with genital ambiguity (20% of girls; 16.7% of the cohort) were both classified as Prader stage III. Both boys had marked penile enlargement (8 cm) with genital Tanner stage IV and pubic/axillary hair; exact testicular volumes in milliliters were not consistently recorded. Height SDS was available in 11 patients, of whom 7 (63.6%) were above +2 SDS. BMI recalculated from the paired height and weight values ranged from 12.0 to 20.8 kg/m² (mean 16.6 ± 3.3 kg/m²). Because the exact age at the time of each anthropometric measurement was not consistently documented, BMI-for-age obesity classification could not be reconstructed reliably.

Bone age was advanced relative to chronological age at the first available assessment in 10 of 12 patients (83.3%), with the most marked skeletal advancement observed in the congenital adrenal hyperplasia (CAH) cases. Interpretable follow-up skeletal information was available in six patients. Bone-age advancement persisted in all six (100%); four of six (66.7%) had no further documented skeletal acceleration, whereas two of six (33.3%) remained markedly advanced without sufficient serial measurements to quantify the rate of progression. Formal growth velocity in cm/year could not be reconstructed reliably because paired serial height measurements with exact dates were unavailable;

however, height SDS was above +2 in 7 of 11 patients with available baseline auxological data (63.6%), supporting frequent statural acceleration at presentation. Baseline hormonal values were heterogeneous according to mechanism: LH ranged from 0.04 to 5.05 IU/L (n=12), FSH from 0.30 to 6.93 IU/L (n=11), estradiol from 12.24 to 373 pg/mL (n=9), testosterone from <0.13 to 1.35 ng/mL (n=10), DHEAS from 1.05 to 140 µg/dL after unit harmonization where possible (n=10), and 17-hydroxyprogesterone from 0.20 to 8.70 ng/mL (n=10). Within the four 11β-hydroxylase-deficiency cases, LH ranged from 0.88 to 3.20 IU/L and FSH from 2.60 to 5.50 IU/L, values that were relatively low compared with the degree of virilization and adrenal androgen excess. Testosterone ranged from <0.13 to 1.35 ng/mL and 17-hydroxyprogesterone from 2.7 to 8.7 ng/mL. DHEAS was variably documented (5.7–36.9 µg/dL in the three cases with recoverable units) and was not uniformly elevated. In one boy, deoxycorticosterone (DOC) was markedly elevated at 1586 pg/mL. In patient 8, biochemical suppression under triptorelin was documented, with LH decreasing from 5.05 to 0.23 IU/L, FSH from 3.67 to 2.06 IU/L, and estradiol from 373 to <10 pg/mL.

Analyte	Available n	Recorded baseline range
LH (IU/L)	12	0.04–5.05
FSH (IU/L)	11	0.30–6.93
Estradiol (pg/mL)	9	12.24–373
Testosterone (ng/mL)	10	<0.13–1.35
DHEAS (µg/dL)	10	1.05–140
17-OHP (ng/mL)	10	0.20–8.70

Table 2. Summary of baseline hormonal values available in the medical records.

Hypothalamic–pituitary MRI was performed in six patients. Four examinations showed no causal lesion, one revealed a hypothalamic hamartoma, and one showed a 9 × 8 mm pituitary adenoma associated with precocious puberty. Pelvic ultrasonography supported estrogen exposure in central forms, while testicular ultrasonography in the evaluated boy did not reveal a tumor.

Patient	Type / etiology	Baseline clinical findings	Bone age (first available)
1	Central – idiopathic	B2P2; body odor; height +4 SDS	8 y at CA 5 y
2	Peripheral – 11β-hydroxylase CAH	B2P2; menstruation; virilization/DSD; Prader III; melanoderma	14 y at CA 6 y
3	Peripheral – 11β-hydroxylase CAH	B2P2; virilization/DSD; Prader III	10 y at CA 6 y
4	Central – idiopathic	B2P2; pubic hair; height +3 SDS	10–11 y at CA ~6 y
5	Peripheral – idiopathic	Vaginal bleeding at age 6 y; B1P1	10 y at CA 6 y
6	Central – hypothalamic hamartoma	B3; hydrocephalus/epilepsy/visual-neurological comorbidity	11 y at CA 5 y 2 mo
7	Peripheral – idiopathic	Thelarche with spontaneous regression; P3	7 y at CA 7 y 2 mo
8	Central – idiopathic	S3P3	11 y at CA 9 y

Patient	Type / etiology	Baseline clinical findings	Bone age (first available)
9	Central – pituitary adenoma associated	B4P5; menarche at 6 y	9–10 y at CA 5 y 11 mo
10	Peripheral – 11β-hydroxylase CAH	Penile length 8 cm; Tanner G4; pubic hair	10–11 y at CA 3 y 3 mo
11	Peripheral – idiopathic	B2P2; mild pubarche	4 y 5 mo at CA 4 y 3 mo
12	Peripheral – 11β-hydroxylase CAH + mosaic 47,XYY	Penile length 8 cm at 4 y; Tanner G4; pubic/axillary hair	14 y at 8 y

Table 3. Individual diagnoses, baseline manifestations, skeletal maturation, and follow-up outcomes

Treatment and Outcomes

Triptorelin therapy was administered to seven patients (58.3%): the five central forms and two peripheral/CAH cases with progressive or secondary central pubertal activation. The four CAH cases were classified as 11β-hydroxylase deficiency and were managed with etiology-directed glucocorticoid therapy, although the exact regimen was incompletely documented in one record. Three non-progressive idiopathic peripheral forms were managed by surveillance.

During the available follow-up, five patients (41.7%) showed regression or partial regression of pubertal manifestations, six (50.0%) showed stabilization or non-progression, and one adolescent with CAH showed age-appropriate pubertal progression at 13 years without evidence of pathological acceleration. No patient had documented pathological worsening during the recorded follow-up. The patient with hypothalamic hamartoma regressed from B3 to B1 under triptorelin. The patient with the pituitary adenoma-associated form achieved amenorrhea after three months, with stabilization of the remaining pubertal signs. Patient 8 showed both clinical and biochemical regression. In the CAH group, virilization was controlled or stabilized, but skeletal advancement persisted; patient 10 had unchanged advanced bone age at the three-month post-triptorelin assessment, and patient 12 showed stabilization without regression. In patient 2, melanoderma was documented and bone age remained advanced despite age-appropriate S4P4 development at 13 years.

Discussion

Epidemiological and Etiological Profile

Girls represented 83.3% of our cohort, consistent with the female predominance reported in precocious puberty [1]. Central and peripheral forms accounted for 41.7% and 58.3% of cases, respectively. In contrast, Chiu et al. reported 93% central and only 7% peripheral forms [6], while another series found 23.3% peripheral forms [7]. The high proportion of peripheral puberty in our study probably reflects the small sample size and referral of complex adrenal disorders to a tertiary center.

Idiopathic forms were present in half of our cohort, and congenital adrenal hyperplasia (CAH) was the most common pathological cause found. Idiopathic central puberty has been reported in 74–96% of cases in larger female cohorts [6,8]. Differences from our findings should be interpreted cautiously because of variations in population, inclusion criteria, and referral patterns.

Physiological and Environmental Factors

Puberty onset is due to reinitiation of pulsatile hypothalamic GnRH secretion, followed by pituitary gonadotropin release and gonadal sex-steroid production. Genetic, nutritional, metabolic, and environmental factors may modify this process. Adiposity may favor earlier puberty through leptin, insulin, and peripheral estrogen pathways.

Bisphenols, phthalates, pesticides, and other endocrine-disrupting chemicals may interfere with central or peripheral pubertal signaling (Figure 1). In addition, published findings remain inconsistent and do not establish causality [5,18]. Systematic evaluation of potential environmental and iatrogenic exposures was conducted There was no exposure to exogenous sex hormones, to dietary supplements with hormones, to hair dyes or to any other products with potential endocrine disrupting effects in any of the patients, intake of biotin that may interfere with hormonal immunoassays was also excluded.

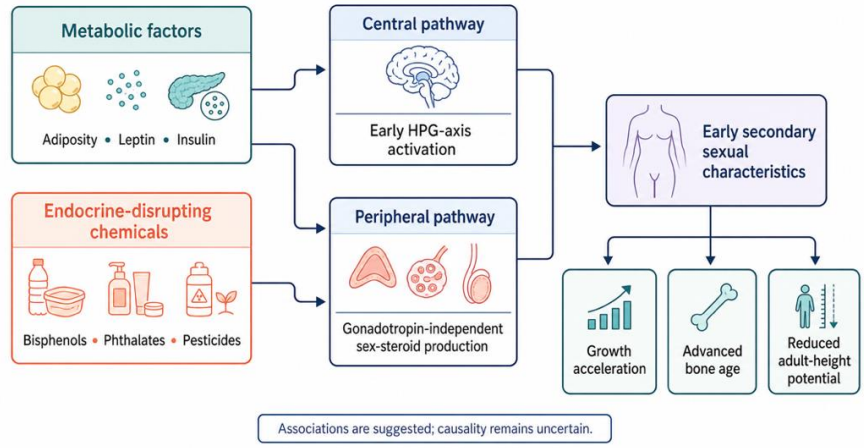

Figure 1. Potential contribution of metabolic factors and endocrine-disrupting chemicals to early pubertal development through central and peripheral pathways. These associations remain suggestive, and causality has not been definitively established.

Central Precocious Puberty

Three patients had idiopathic central precocious puberty, consistent with its predominance among girls in previous studies [6,8]. The 2026 Endocrine Society guideline recommends the ultrasensitive basal LH assay as the first hormonal investigation, and the GnRH stimulation test should be used only in inconclusive cases [1]. In our series, diagnosis relied mainly on clinical progression, basal gonadotropins, bone age, pelvic ultrasonography, and imaging. The limited availability of stimulation-test results reduced diagnostic standardization.

The guideline recommends clinical observation for isolated or slowly progressive Tanner B2 development. Rapid progression, accelerated growth, advanced bone age or neurological manifestations should prompt early evaluation and treatment [1]. Most treated patients in our series fulfilled these criteria. The use of three-monthly triptorelin was consistent with the preference for long-acting formulations. The main recommendations are summarized in Table 4 [1].

Clinical area	2026 recommendation
Initial observation	Monitor isolated Tanner B2 development every 4–6 months to distinguish slowly progressive from rapidly progressive puberty.

Immediate evaluation	Investigate Tanner B3 or higher, rapid progression, growth velocity >6 cm/year, or neurological manifestations.
Hormonal assessment	Use ultrasensitive basal LH first; reserve the GnRH stimulation test for inconclusive cases.
Brain MRI and genetics	Reserve MRI for younger children or neurological findings. Avoid routine genetic testing except in selected familial forms.
GnRH agonist treatment	Treat progressive forms threatening adult height and preferably use a long-acting formulation when prolonged therapy is expected.
Monitoring and discontinuation	Monitor Tanner stage, growth velocity, and bone age. Avoid routine hormonal testing or growth hormone addition. Individualize treatment discontinuation according to chronological age, bone age, and growth potential.

Table 4. Summary of the 2026 Endocrine Society recommendations for central precocious puberty [1]

Peripheral Precocious Puberty

Three patients were classified as having idiopathic peripheral precocious puberty after identifiable ovarian, adrenal, tumoral, and central causes were excluded. Transient ovarian follicular activity may produce intermittent estrogen exposure even without a persistent ovarian cyst.

Most of the autonomous ovarian cysts regress spontaneously, but recurrence and a secondary central activation can be seen [9]. Isolated prepubertal vaginal bleeding can also be benign in the absence of other pubertal or pelvic abnormalities [10]. However, surveillance was indicated in our non-progressive cases. Nevertheless, Tanner stage, growth velocity, bone age, basal LH, and pelvic ultrasonography should be reassessed because some incomplete peripheral forms may progress [11].

Congenital Adrenal Hyperplasia

Four (33.3%) patients had congenital adrenal hyperplasia due to 11β-hydroxylase deficiency. This high frequency is probably related to referral bias and the limited sample size.

11 β-hydroxylase deficiency constitutes approximately 5–8% of classic CAH and is characterized by virilization, acceleration of growth, advanced bone age, and hypertension [12]. A pediatric series reported a median bone-age advancement of 5.5 years [12], comparable to the severe advancement observed in our boys. Worldwide, 21-hydroxylase deficiency predominates, but 11β-hydroxylase deficiency is relatively more common in the Middle Eastern and North African populations [13]. Biochemically, elevated 11-deoxycortisol and deoxycorticosterone are the most informative markers of 11β-hydroxylase deficiency, whereas adrenal androgens and 17-hydroxyprogesterone may also be elevated but are less specific [12,13]. Prolonged adrenal androgen excess may initially cause peripheral puberty and subsequently trigger central activation. Late-onset 21-hydroxylase deficiency may be confused with primary central puberty [14]. Hydrocortisone suppresses adrenal androgen production but cannot completely reverse established skeletal advancement. GnRH agonist therapy may be required when secondary central activation develops.

Organic and Rare Associations

One patient had central precocious puberty secondary to a hypothalamic hamartoma, associated with hydrocephalus, epilepsy, visual impairment, and developmental abnormalities. In a series of 30 children

with hypothalamic hamartoma, 80% developed central precocious puberty, 50% had gelastic seizures, and 43.3% had developmental delay [15]. Our patient's breast development regressed under triptorelin, consistent with the reported efficacy of GnRH agonists .

Another patient had central precocious puberty associated with a 9 × 8 mm pituitary adenoma. Gonadotroph adenomas causing precocious puberty are extremely rare but LH-, FSH- and mixed-secreting tumors have been reported [16,17]. Because the presence of a pituitary lesion does not prove causality, the adenoma should be described as associated with precocious puberty unless autonomous secretion of hormones is demonstrated. Complete pituitary evaluation and serial MRI remain necessary.

One boy had peripheral precocious puberty due to 11β-hydroxylase-deficient CAH associated with mosaic 47,XYY syndrome. No established relationship between Jacob syndrome, CAH, and precocious puberty has been demonstrated. This coexistence is therefore probably fortuitous, with virilization and skeletal advancement mainly attributable to adrenal androgen excess. Long-term endocrine, neurodevelopmental, and reproductive follow-up is warranted.

Treatment, Strengths, and Limitations

Seven of the 12 patients received triptorelin, and the four patients with 11β-hydroxylase-deficient CAH were managed with etiology-directed glucocorticoid therapy, although the exact regimen was incompletely documented in one case. Available follow-up showed regression, partial regression, or stabilization of pathological pubertal progression. Current recommendations are for clinical monitoring using Tanner stage, growth velocity, and annual bone age, with hormonal testing reserved for those suspected of treatment failure [1].

This study illustrates the etiological diversity of precocious puberty and includes several uncommon conditions. However, the generalizability of the findings is limited by its retrospective design, small sample size, referral bias, incomplete hormonal data, and variable duration of follow-up. Exact serial growth velocity could not be reconstructed in all patients because paired heights and dates were incomplete; repeat bone age was also unavailable in several records. Testicular volumes were not consistently recorded, and some laboratory units required retrospective harmonization. Anthropometric measurements were not always linked to an exact visit age, limiting reliable BMI-for-age obesity classification. These limitations prevented assessment of final adult height and long-term reproductive outcomes.

Conclusion

Precocious puberty encompasses heterogeneous central and peripheral disorders. Idiopathic forms predominated, while CAH was the most frequent identified pathological cause. Hypothalamic hamartoma, pituitary adenoma, and mosaic Jacob syndrome represented uncommon but clinically relevant associations. Early etiological evaluation and individualized treatment are essential to control pubertal progression and skeletal advancement. Long-term follow-up is needed to assess growth and reproductive outcomes.

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