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Original Article

Association Between Body Mass Index and Precocious Puberty Among Girls in Sulaymaniyah: A Retrospective Cross-Sectional Study

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Abstract

Background: Precocious puberty, characterized by the early onset of secondary sexual characteristics before age 8 or menarche before 10, is a growing global health concern. There is no evidence about the correlation between childhood obesity and precocious puberty in the Kurdistan Region of Iraq. This paper explores the correlation between early pubertal indicators and body mass index (BMI) in girls living in Sulaymaniyah.

Methods: A cross-sectional study was employed from September 2024 to June 2025 at Dr. Jamal Ahmad Rashid Pediatric Hospital, involving 93 females aged 4–10 years with complete medical records. Participants were assessed for clinical signs of puberty, BMI, bone age, and hormonal levels. Exclusion criteria included non-pubertal conditions, chronic illnesses, and medication-induced cases. Statistical analyses were performed using SPSSv27.

Results: A significant portion of the participants was overweight (26.9%) or obese (33.3%). Dieting was highly correlated with overweight and obesity (χ^2 = 45.38, p < 0.001) and physical inactivity was significantly prevalent among overweight and obese girls (χ^2= 53.27, p = 0.001). Basal LH (p = 0.015), stimulated LH (p = 0.013) estradiol (p = 0.014) and increased bone age advancement (p =0.004) were significantly associated with elevated BMI categories. Basal LH (p = 0.005), stimulated LH (p = 0.008) and estradiol (p = 0.005) in obese participants were significantly more elevated when compared to normal weight peers. A family history of obesity was closely related to higher BMI (χ^2 = 19.508, p < 0.001).

Conclusion: High BMI has a strong association with hormone and skeletal markers of precocious puberty among this cohort, and obesity is a key modifiable risk factor of early pubertal development in Sulaymaniyah.

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Introduction

Precocious puberty (PP), defined as the development of secondary sexual characteristics before the age of eight in girls or menarche before the age of ten, is a common global health concern that has profound and significant long-term sequelae [1]. The timing of puberty is orchestrated by the sophisticated interplay of the hypothalamic-pituitary gonadal (HPG) axis.

This process is initiated by the pulsatile release of gonadotropin-releasing hormone (GnRH), which stimulates the secretion of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). These hormones, in turn, promote gonadal maturation and the production of sex steroids [2]. However, this finely tuned neuroendocrine system is highly susceptible to metabolic signals, particularly those originating from adipose tissue. Leptin, an adipokine, plays a crucial role in this interaction, with elevated levels in obese individuals potentially disrupting the normal timing of puberty [3]. Furthermore, the insulin resistance commonly associated with obesity can also contribute to the premature activation of the HPG axis [4]. PP has implications beyond physical well-being, influencing psychological and social development. Girls undergoing early puberty are at greater risk for behavioral and cognitive issues (such as ADHD and depression), metabolic syndrome, cardiovascular disease, and breast cancer later in life [5, 6]. PP can be broadly categorized into two types, Central PP (CPP) (GnRH dependent), and Peripheral PP (PPP) (GnRH independent) [1]. Most cases of CPP are idiopathic (over 90%), indicating that the underlying cause remains unidentified [5].

The significant health issue faced by the world today is childhood obesity, which has been constantly related to the earlier pubertal development, mature bone age, and changes in gonadotropin and sex steroid profiles [7, 8]. The recent research in the Kurdistan Region of Iraq has revealed a relatively high prevalence of overweight and obesity in young children, which can be ascribed to blistering urbanization, changes to energetically-rich diets, and decreased physical activity [9, 10]. In spite of this, there is little evidence on the association between BMI and PP in Sulaymaniyah and other underrepresented populations.

In order to fill this gap, our research paper explores the relationship between BMI and the symptoms of PP (hormonal profiles (basal and stimulated LH, estradiol), bone age development, and clinical progression) in girls in Sulaymaniyah. We also address the possibility of how this relationship may be mediated by the lifestyle (diet, physical activity) and family history of obesity. It is worth noting that we examine how socioeconomic and demographic factors alter the BMI-PP association in this particular regional set up. This study would help to enlighten specific preventive measures and early clinical intervention based on these connections, and the high-risk group.

Materials and Methods

1. Study Design and Setting

The study was a retrospective cross-sectional study, conducted from September 2024 to June 2025 at the endocrine department of Dr. Jamal Ahmad Rashid Pediatric Teaching Hospital (DJAR) in Sulaymaniyah, Iraq, a tertiary teaching hospital on pediatric endocrine diseases in this region. Since this type of design is retrospective, it is possible to identify the associations, but not to prove the cause-effect relationships between BMI and evidence of PP.

2. Participants

During the study period, 93 girls aged 4-10 years who had CPP were selected, and they were under active follow-up in DJAR. A non-probability consecutive sampling method

was used to enroll girls diagnosed with CPP who attended the endocrinology department during the study period. Although this strategy has made the study feasible in the clinical environment, the study might be constrained in its ability to generalize its findings to the wider population of girls with PP in Sulaymaniyah especially those who attend the private-managed clinics or remain undiagnosed. Inclusion criteria for the study were: female sex, age between 4 and 10 years at the time of diagnosis, and the availability of complete medical records. Patients were excluded if they presented with premature thelarche (isolated breast development without other signs of puberty), PPP, organic diseases (e.g., ovarian tumors, hamartomas), chronic diseases (e.g., chronic kidney disease, asthma, epilepsy), genital abnormalities, or if their PP was induced by medications (e.g., glucocorticoids).

3. Data collection

Hospital records were used to retrieve clinical and demographic data. Lifestyle data, namely dietary (frequency of fruit/vegetable consumption, frequency of fast food, frequency of sugary drinks) and physical activity (duration of moderate-to-vigorous physical activity, frequency of moderate-to-vigorous physical activity) were gathered using a structured questionnaire, which was completed by the parent or guardian on a routine visit. It is important to note that this questionnaire was written to be used clinically in our department and has not been formally validated; hence the reliability and validity of that questionnaire is unknown, and this can lead to bias in measurement.

4. Ethical Considerations

The College of Medicine Review Committee, University of Sulaimani (Ref: 335, October 23, 2024) approved the study and registered the study with the Department of Clinical Sciences (Ref: 132, October 8, 2024). Written informed consent was obtained from the parental or legal guardians.

5. Physical Examination

Experienced pediatric endocrinologists and pediatricians jointly evaluated the sexual development of each participant. Breast development was assessed through both inspection and palpation, with particular attention paid to the presence of nodules in obese girls. The Tanner staging method was utilized to classify breast and pubic hair development into stages 1 through 5 [11]. For the purpose of this study, PP was specifically defined as reaching Tanner breast stage 2 (B2) or higher before the age of 8, or experiencing menarche before the age of 10. Height and weight measurements were meticulously obtained using standardized equipment. Participants' body mass index (BMI) was calculated and subsequently categorized according to the World Health Organization (WHO) Child Growth Standards [12]. These classifications were: severe thinness (BMI < -3 standard deviations [SD]), thinness (BMI < -2 SD), normal (BMI between -2 and +1SD), overweight (BMI between +1 SD and +2 SD), and obesity (BMI ≥ +2 SD).

6. Hormonal parameters

The hormonal evaluation of PP in girls aims to distinguish central CPP from PPP causes and identify underlying conditions. Initial screening includes assessing the HPG axis and adrenal functions through key hormone tests: basal LH (prepubertal level <0.1-0.3 IU/L) and FSH (typically < 4.0 mIU/mL),

estradiol (levels >20pg/mL suggests puberty), and adrenal androgens like DHEA-S and 17-OHP (to rule out adrenal disorders). The GnRH stimulation test, with a peak LH ≥5 IU/L post-stimulation is the gold standard for diagnosing CPP and an LH/FSH ratio >0.63 is indicative of progressive CPP [13]. Thyroid-stimulating hormone (TSH) and free thyroxine (FT4) are assessed to exclude hypothyroidism, which can mimic precocious puberty. Prolactin levels are also measured to exclude hyperprolactinemia, although they generally remain within normal physiological ranges during pubertal development. Additional tests such as Inhibin B, AMH, and hCG, and ACTH stimulation are used to identify specific causes. Hormonal analyses are typically conducted in the early morning, as recommended by the pediatric endocrinologists at the Endocrinology Department of DJAR Hospital. However, we cannot specify the exact methods used for these analyses, as they were performed across different laboratories. Additionally, due to the retrospective cross-sectional nature of my study, detailed methodological information was not available.

7. Imaging Procedures

Pelvic ultrasounds were performed by a sonographer. Bone age (BA) was assessed by taking x-rays of left hand and wrist joint, using the Tanner–Whitehouse 3 Method [14] interpreted by a pediatric endocrinologist or radiologist. Magnetic resonance imaging (MRI) of the pituitary or sellar region was conducted for special indications like patients younger than 6 years of age, particularly those with signs or abnormal neurological findings indicating central nervous system (CNS) tumors during follow-up.

8. Diagnostic Criteria

CPP was diagnosed based on established criteria [15]: presence of secondary sexual characteristics before age 8 (Breast Stage 2) or menstruation before 10, linear growth acceleration (>6 cm/year), advanced bone age (≥1 year), specific uterine/ovarian measurements, and initiation of gonadal axis function (basal LH ≥0.3 IU/L or positive GnRH provocation test; LH ≥ 5 IU/L). Idiopathic CPP was confirmed for patients meeting CPP criteria without identifiable secondary causes.

9. Statistical analysis

The statistical analysis was performed using IBM SPSS Statistics, version 27. Data distribution was assessed using the Kolmogorov-Smirnov and Shapiro-Wilk tests to determine the appropriate type of analysis: normally distributed continuous variables (e.g., age, BMI) were summarized with mean ± standard deviation, while non-normally distributed variables (e.g., hormones, bone age) used median with interquartile range. Categorical data were presented as frequencies and percentages.

The Mann-Whitney U test with Bonferroni adjustment assessed bone age variations across BMI groups. Chi-square or Fisher's Exact Tests analyzed relationships between BMI categories and categorical factors. Pearson and Spearman correlations examined associations among continuous variables. All statistical tests were two-tailed, and a p-value of < 0.05 was regarded as statistically significant. To control Type I error in multiple comparisons, a Bonferroni correction was applied to the six Mann–Whitney U tests, resulting in an adjusted significance threshold of $p < 0.0083$ (i.e., $\alpha =$

0.05/6). Under this criterion, p-values below 0.0083 were considered statistically significant, values between 0.0083 and 0.05 were interpreted as marginally significant, and p-values ≥ 0.05 were regarded as not statistically significant.

Results

1. Characteristics of the Study Population

The study included 93 girls with CPP. The demographic, clinical, and lifestyle characteristics of the participants are summarized in Table 1. The mean chronological age was 89.06 ± 16.5 months, and the median bone age was 132 months (IQR: 108–137), unequivocally indicating accelerated skeletal maturation across all participants with mean advancing bone age was 34.63 months ± 12.58 SD. The mean Body Mass Index (BMI) was 18.98 ± 3.44 kg/m², no girl was severely thin (≤ -3 SD), 3.2% (n = 3) were thin (BMI < -2 SD), 36.6% (n = 34) had normal weight (BMI between -2 SD and +1 SD), 26.9% (n = 25) were overweight (BMI between +1 SD and +2 SD), and 33.3% (n = 31) were obese (BMI ≥ +2 SD) (Figure 1).

2. Association between BMI Categories and Hormonal Parameters

The Kruskal-Wallis H test revealed statistically significant differences in several hormonal parameters across the defined BMI categories (Table 2). Baseline LH and estradiol levels differed significantly across BMI categories (p = 0.015 and p = 0.014, respectively): the obese group had the highest LH, while the Overweight group showed the highest estradiol. Baseline FSH showed a marginal trend (p = 0.057), with the Obese group having the highest and Overweight the lowest levels. After GnRH stimulation, both stimulated LH (p = 0.013) and FSH (p = 0.032) varied significantly by BMI—Normal and Obese groups had the highest S.LH and S.FSH, respectively. In contrast, the LH/FSH ratio and stimulated estradiol showed no significant differences across BMI groups (p = 0.198 and p = 0.499).

3. Bone Age Advancement and BMI

All patients in the study exhibited advanced bone age, meaning their skeletal maturation was ahead of their chronological age. Figure 2 visually illustrates the consistent correlation between chronological age and advanced bone age, indicating that bone age invariably surpassed chronological age in the study populations. The age progression of the bones was observed to be very much dependent on the types of BMI (Kruskal-Wallis, p=0.004). The mean rank of bone age was the highest among girls in the category of obese (57.6), then the overweight (48.4), normal-weight (39.8), and thin (8.2). The pairwise comparisons have shown that the level of bone age advancement was statistically significantly lower in thin girls compared to overweight and obese girls ($p < 0.0083$). In the same manner, the normal weight girls had a lower bone age progress compared to the obese girls (Table 3).

Table 1: Clinical and Demographic Characteristics of Participants (n = 93)

Variables	Values
Anthropometric Measures	
Chronological Age(months), mean ±SD	89.06 ± 16.5
Weight (kg), mean ± SD	30.99 ±7.79
height(cm) ,mean ± SD	127.2 ± 8.75
BMI(kg/m²) ,mean ±SD	18.98 ± 3.44
Hormonal Parameters	
Basal LH(mIU/mL), median(IQR)	1.30 (0.50-2.90)
Basal FSH(mIU/mL) ,median(IQR)	4.20 (2.88-6.50)
Stimulated LH (mIU/mL)median (IQR)	10.2 (5.95-14.35)
Stimulated FSH(mIU/mL) ,median(IQR)	10 (7.85-12.60)
LH/FSH ratio, median(IQR)	1.07 (0.80-1.34)
Estradiol(pg/mL),, median(IQR)	34.0 (17-57.5)
Stimulated Estradiol(pg/mL), mean ± SD	110.4 ± 42.66
Clinical data	
Bone Age(months), median(IQR)	132 (108-137)
Bone Age – Chronological Age Difference (months), mean ± SD	34.63 ± 12.58

Demographic Data. Lifestyle and Family Factors		**value**
Residency, frequency (%)	Inside	42(45.2%)
	0utside	51(54.8%)
Parent education level, frequency (%)	Illiterate	0(0%)
	Read and write	2(2.2%)
	Primary	24(25.8%)
	Secondary	33(35.5)
	College and postgraduate	34(36.6)
Socioeconomic state, frequency (%)	Low	32(34.4%)
	Moderate	54(58.1%)
	High	7(7.5%)
Diet habit, frequency (%)	High in fruits, vegetables, and whole grains	4(4.3%)
	High in processed foods, fast foods ,sugars, and fats	86(92.5%)
	Balanced diet	3(3.2%)
Exercise habit, frequency (%)	3-4 times per week	4(4.3%)
	Rarely	18(19.4%)
	Never	71(76.3%)
Family history of obesity, frequency (%)	Yes	22(23.7%)
	No	71(76.3%)
Family history of PP, frequency (%)	Yes	3(3.2%)
	No	90(96.8%)

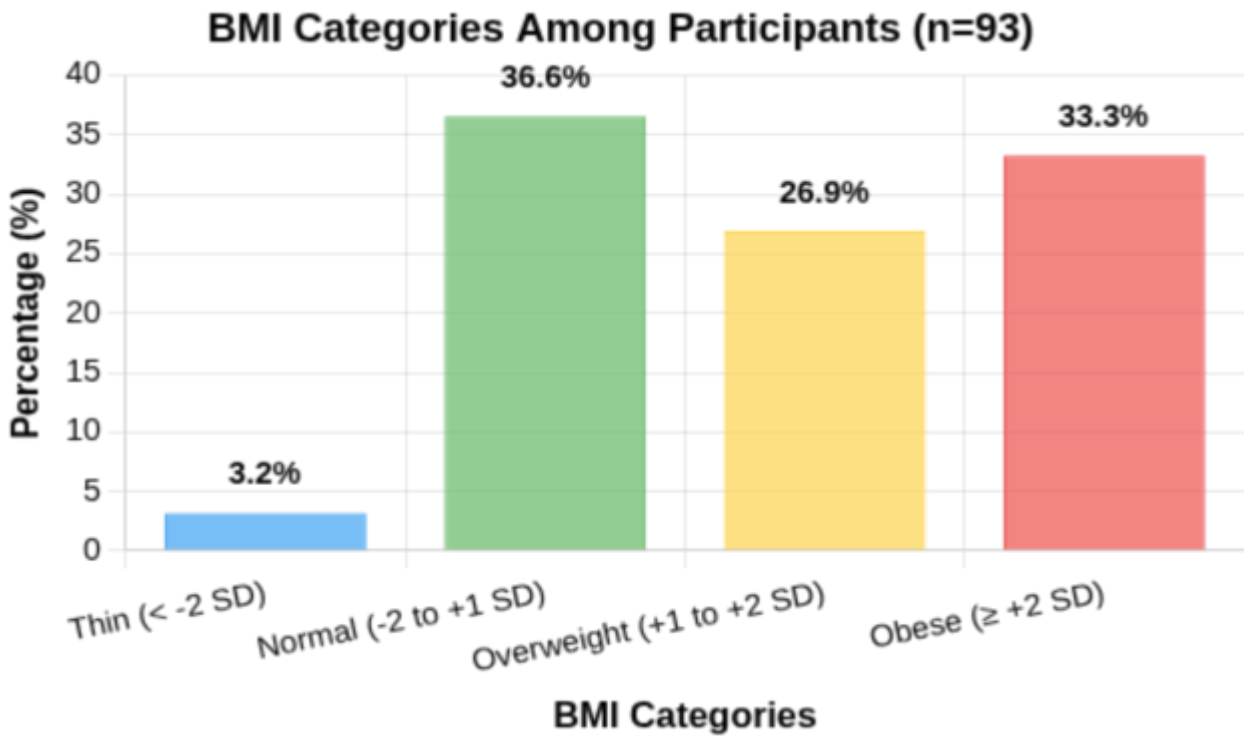

Figure 1: Distribution of BMI Categories among Participants

Table 2: Association of Hormonal Parameters with BMI Categories

Variable	Thin(n=3)	Normal (n = 34	Overweight (n = 25)	Obese (n = 31)	*p*-value
LH (mIU/mL)	0.25±0.14	1.69 ± 1.88	1.43 ± 1.24	2.3 ± 1.6	**0.015**
FSH (mIU/mL)	3.80 ±1.21	4.99 ± 2.62	3.67 ± 2.1	5.45 ± 2.59	0.057
E2 (pg/mL)	14.66±12.66	31.85 ± 25.95	77.79 ± 25.01	50.05 ± 27.36	**0.014**
S. LH(mIU/mL)	5.03±1.15	9.03 ± 6.06	6.58 ± 1.41	8.73 ± 2.34	**0.013**
S.FSH(mIU/mL)	5.9 ±3.25	9.6 ± 3.2	9.71 ± 3.16	12.83 ± 6.17	**0.032**
LH/FSH ratio	1.00±0.53	0.92 ±0.31	1.13±0.58	1.32±0.51	0.198
S.E2 (pg/mL)	93.66±45.62	107.3±47.43	104.38±46.02	124.07±32.73	0.499

Statistical test: Kruskal–Wallis test. Bold values indicate statistical significance ($p < 0.05$). S.LH (stimulated luteinizing hormone), S.FSH (stimulated follicular stimulating hormone), E.2 (estradiol), S.E2 (stimulated estradiol)

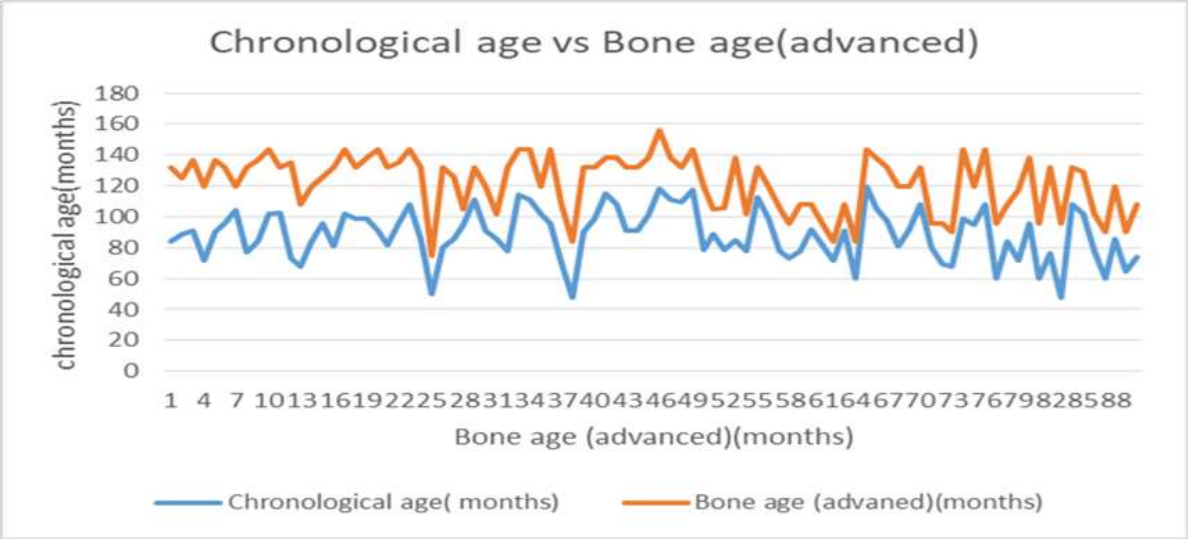

Figure 2: Advancing bone age in relation to chronological age

Table 3. Bone Age Advancement in Relation to BMI Categories

BMI Category	Mean Rank of Bone Age	Pairwise Comparison (vs. Other Groups)	p-value	Interpretation
Thin (< −2 SD)	8.17	vs. Normal: lower vs. Overweight: lower vs. Obese: lower	0.011 0.007 0.006	Significantly least bone age advancement
Normal Weight (−2 SD to +1 SD)	39.76	vs. Obese: lower	0.008	Moderate bone age advancement
Overweight (+1 SD to +2 SD)	48.40	vs. Obese: no difference	0.158	High bone age advancement
Obese (≥ +2 SD)	57.56	—	—	Greatest bone age advancement

Statistical Test: Kruskal–Wallis H = 0.004; ***Post-hoc Test:*** Mann–Whitney U with Bonferroni correction (significance threshold p < 0.0083)

4. Relation between BMI categories with and Lifestyle/Family Factors

Cross-tabulation analysis showed that there are strong relationships between the BMI category and the various behavioral and familial variables (Table 4). Girls with a family history of being obese were more likely to be considered obese among the sample ($\chi2$=19.508, p <0.001). Similarly, dieting was highly correlated with overweight and obesity ($\chi2$ = 45.38, p < 0.001) and physical inactivity was significantly prevalent among overweight and obese girls ($\chi2$= 53.27, p = 0.001). There were no statistically significant correlations found between BMI and the socioeconomic status, residential place, or family history of PP.

Table 4. Relationship Between BMI Category and Lifestyle/Family Factors

Variable	Thin(n=3)	Normal (n = 34)	Overweight (n = 25)	Obese (n = 31)	*p*-value
Physical Activity (≥3 times/week)	2(2.2%)	14(15.1%)	0(0%)	0(0%)	**< 0.001**
Fast Food Intake ≥ 2 times/week	0(0%)	15(16.1%)	25(26.9%)	31(33.1%)	**< 0.001**
Family History of Early Puberty	0(0%)	1(1.1%)	1(1.1%)	1(1.1%)	0.988
Family History of Obesity	0(0%)	1(2.9%)	6(24.0%)	15(48.4%)	**< 0.001**

***Statistical test**:* Chi-square test. Bold values indicate statistical significance ($p < 0.05$)

Discussion

The present research investigated the correlation between BMI and PP in girls living in Sulaymaniyah, thus making new contributions to the role of excess adiposity in hormonal control and skeletal growth in early puberty development. Through the evaluation of the hormones, anthropometrics, and lifestyle measures, the study contributes to the growing body of evidence demonstrating that obesity is a relevant and adjustable factor of premature pubertal onset.

The distribution of BMI categories revealed a notable prevalence of overweight and obese individuals, alongside a significant proportion with normal BMI. Specifically, we found that 33.3% of participants were classified as obese (BMI ≥ +2SD), representing the largest group, followed by 26.9% who were overweight (BMI ≥ +1SD). This distribution highlights a concerning trend toward higher BMI categories, particularly obesity, which may have implications for pubertal development and overall health outcomes in this population. Our finding aligns with contemporary pediatric endocrinology literature that consistently reports this association [16, 17]. The overweight and obese phenotypes in girls with PP are predominant, which is how the extreme need to conduct preventive interventions to prevent childhood obesity is evidenced. In addition, the increased levels of sedentary lifestyles and unhealthy eating habits such as consumption of fast foods, refined sugars and fats may indicate that environmental and lifestyle influences can contribute to the severity of the biological tendency to early puberty [18, 19].

The key finding of the study was a strong correlation between BMI and gonadotropic hormones. Obese girls exhibited elevated levels of both basal and stimulated LH, reinforcing previous research indicating that excess adiposity accelerates activation of the HPG axis. This acceleration is thought to be driven by heightened leptin and insulin signaling associated with increased body fat [6]. Leptin in obesity could inhibit central inhibition of GnRH neurons, thus promoting earlier pulsatile leapt to LH and earlier puberty. On the other hand, conflicting evidence has been found; El-Masry et al. found an inverse correlation between BMI and LH reaction in Egyptian girls that showed early signs of puberty [20]. Several differences can be explained by the

heterogeneity of pubertal subtype (central versus peripheral), leptin resistance or differences in metabolic health. Chronic low grade inflammation and hyperinsulinemia in obese children can block the responsiveness to GnRH resulting in the weakened LH release in spite of the advanced phenotypic maturation [21, 22]. These mechanisms underscore the complex endocrine interplay between adiposity and pubertal timing.

In addition to the effect of gonadotropins, significant positive correlation between BMI and the levels of circulating estradiol was found. The group of obese female participants had the highest levels of estradiol which is in line with the previously conducted studies that have cited the adipose tissue as an extragonadal location of aromatase activity that converts androgens to estrogens [23]. This peripheral aromatization mechanism leads to the greater exposure to systemic estrogen and the acceleration of the development of secondary sexual traits. It is worth noting that the baseline estradiol levels continued to exhibit a significant correlation with the BMI, however, estradiol responses to gonadotropin stimulation did not showing any association meaning that the estradiol increase in obese girls is due to chronic peripheral aromatization, but not endocrine stimulation.

Bone age advancement was observed to be consistent among all participants of the research and showed a positive association with BMI categories. The females who exhibited the highest level of skeletal maturation were the obese girls followed by the overweight and normal weight. These findings are in line with previous studies that have found that increased adiposity is coupled with faster bone maturation by leptin- and estrogen-controlled mechanisms [24, 25]. It is theorized that leptin has a direct action on osteoblasts and, thus, increases endochondrial ossification and that high estrogen concentration accelerates epiphyseal plate closure. All of these mechanisms explain the correlation between obesity and an advanced bone age in girls with precocious pubertal initiation.

Other inputs to the temporal effect of pubertal development were based on lifestyle and family variables. There was also a significant correlation between a family history of obesity with both the BMI of the participants and the precocious puberty regardless of whether the child was currently overweight or not. The implication of this relationship is the existence of common genetic or epigenetic phenotypes that control metabolic regulation and pubertal timing. As it has been reported in the literature, poor dietary practices and lack of physical activity were common among the girls with high BMI [26, 27]. These lifestyle aspects should be put into consideration as a target in the solution of the problem of premature pubertal development using the approach of the public-health. Conversely, BMI failed to show significant relationships with socioeconomic, geographic residence, and family history of PP, indicating that biological and behavioral factors could override demographical factors in this cohort.

This study adds to the expanding body of evidence linking childhood obesity to disruptions in endocrine and skeletal development. Drawing on data from the Kurdish population in Sulaymaniyah—a group previously underrepresented in pediatric endocrinology research—the findings reveal a positive association between

higher BMI and elevated levels of luteinizing hormone (LH) and estradiol, as well as accelerated bone age progression in girls with precocious puberty (PP). These results underscore the critical need for early screening of BMI and metabolic markers in girls presenting with signs of pubertal onset. Furthermore, the study highlights the necessity of developing culturally tailored preventive interventions and calls on clinicians to incorporate nutritional guidance and physical activity counseling into routine pediatric care, recognizing obesity as a key driver of early puberty.

The current research has limitations that can be discussed. To begin with, cross-sectional design, as is, limits the ability to ascertain causality between the BMI and the studied hormonal or skeletal phenotypes. Secondly, non-probability consecutive sampling method in one geographical area creates doubts on selection bias and limits generalization to the rest of the population. Third, the hormonal tests were performed across different laboratories, we cannot specify the exact methods used for these analyses and due to the cross-sectional nature of the study, detailed methodological information was not available. Lastly, despite primary lifestyle covariates controls, there is still the possibility of residual confounding especially when dealing with unmeasured variables like sleep length, psychosocial stressors, or genetic polymorphs.

Conclusion

This study confirms that there is a strong correlation between high body mass index, as well as early puberty and rapid skeletal development in female respondents who experience precocious puberty in Sulaymaniyah. High levels of basal luteinizing hormone and estradiol in the obese participants indicate that a high adiposity can trigger the hypothalamic-pituitary-gonadal axis prematurely. The results highlight a biological nexus of obesity and pubertal timing and support the importance of lifestyle determinants that can be modified, such as dietary habits and physical sedentary activity. At this juncture, early identification and preventive interventions targeting maintenance of a healthy body weight in childhood is critical towards preventing the incidence of progressing precocious puberty and its associated long-term health consequences.

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Conflict of interest

The authors declare no conflict of interest

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