American Journal of Biomedicine and Pharmacy
https://biojournals.us/index.php/AJBP
ISSN: 2997-7177

Relation between Obesity and Precocious Puberty a Biochemical and Radiological Test

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Received: 2024, 15, Jan
Accepted: 2025, 21, Feb
Published: 2025, 17, Mar

Annotation: Background: Obesity has also increasingly been recognized as a key player in the determination of pubertal timing, and there is compelling evidence associating excess adiposity with precocious puberty, most famously in girls. Precocious puberty, i.e., development of secondary sexual characteristics at less than 8 years in girls, has been associated with endocrine dysregulation, in the form of elevated leptin, insulin resistance, and increased estrogen secretion, all contributing to premature activation of the hypothalamic-pituitary-gonadal (HPG) axis.

Objective: The current study is intended to investigate the correlation between obesity and precocious puberty in biochemical markers (LH, FSH, estradiol, insulin, and leptin) and radiological assessment (assessment of bone age) in 8-9-year-old girls.

Methods: A retrospective cross-sectional study was conducted in Al-Khansa Hospital, where subjects were separated into obese and non-obese categories. Blood analyses were conducted for hormonal markers, and X-ray imaging was utilized to assess bone age advancement. Statistical analysis was conducted to analyze the association between obesity and the development of early puberty.

Results: Results indicate a significant correlation between precocious puberty with obesity, and 70% of the participants were found to have elevated LH, FSH, and estradiol levels,

which supported premature activation of the HPG axis. Similarly, 70% had elevated fasting insulin, and 60% had elevated leptin, indicating a metabolic-hormonal association with the premature development of puberty. Radiological findings supported advanced bone age in obese children, further strengthening the hypothesis of premature pubertal development.

Conclusion Early screening, hormonal evaluation, and lifestyle interventions are crucial for delaying puberty onset and reducing long-term health risks in obese children at risk of precocious puberty.

Keywords: Obesity, Puberty, leptin.

Introduction: The Association between Obesity and Early Puberty Obesity has grown to be a concern in world health, whose influence on all various physiological processes goes well beyond metabolic disorders to the aspects of pubertal timing. One of the most troubling correlations concerns the link between obesity and the age of puberty, more precisely called precocious puberty-the condition when children begin to develop secondary sexual characteristics earlier than they normally would before age 8 in girls and before age 9 in boys. The increasing trend in children's obesity these days no doubt is a contribution to the growing occurrence of precocious puberty, and this is indeed worrisome in many aspects, with its potentially long-term physical and psychological impacts on children with such disorders(Li *et al.*, 2017).

It is characterized by the precocious activation of the hypothalamic-pituitary-gonadal axis, a system of glands and hormones that regulates reproductive development. Ordinarily, puberty is initiated when the brain sends a signal to the pituitary gland to release gonadotropins, an inducer for the gonads-ovaries in girls and testes in boys-to release sex hormones. It now appears that in obese children, excess adipose tissue may interfere with the process, accelerating the hormonal changes responsible for earlier sexual maturation (Koysombat, Dhillo and Abbara, 2023).

This, indeed, is a very complex and multifactorial kind of relationship, and the reasoning that links obesity with early puberty in children would, therefore, definitely relate to hormonal and biochemical changes, including a change in growth pattern. Leptin is one important hormone secreted by the fat cells, which has been found to play a significant role in the commencement of puberty. Further, there are increased insulin levels, sex hormones, and altered HPG axis activity, which have been suggested as other possible mechanisms (Shi, Jiang and Zhang, 2022).

Biochemical and radiological investigations will be required to explain the role of obesity in puberty development. The biochemical tests give an insight into the hormonal changes such as increased levels of leptin, increased levels of sex hormones like estrogen and testosterone, and altered levels of gonadotropin, which are the early manifestations of pubertal activation. Similarly, there are radiological tests such as determination of bone age through X-ray, which is an important input regarding the timing of skeletal maturity, which usually coincides with the timing of early puberty (Shi, Jiang and Zhang, 2022).

Consequently, by considering both the biochemical markers and radiological evidence of damage to growth and development in a child with obesity-related precocious puberty, clinicians can hereby appreciate the underlying mechanism in the development and devise appropriate

strategies for intervention (Rose-John and Schooltink, 2007).

It is one of the major health challenges that has appeared at the threshold of the 21st century and is becoming increasingly prevalent in all age groups. The sad fact about rising childhood obesity involves an important and alarming concern regarding its possible effects on the timing of puberty, especially the onset of precocious puberty. Precocious puberty has been defined as the development of secondary sexual characteristics in girls, breast development; in boys, and enlargement of the genitals before age 8 in girls and before age 9 in boys. It has received considerable interest due to its potential long-term effects on physical, psychological, and social development .Obesity and precocious puberty have a very complex interrelation based on various physiological paradigms. More precisely, such an association is said to be mediated through inter-relations of excess body fat with the hypothalamic-pituitary-gonadal axis, a complex neurohormonal feedback system that acting on mutual interaction with the hypothalamus, pituitary gland, and gonads (ovaries or testes)-mediates sexual maturation and puberty onset, resulting in the production of reproductive hormones such as estrogen, progesterone, and testosterone. Disruption of this system, as occurs in obesity, often leads to precocious puberty.

Several important factors would explain the possible influence of obesity on the timing of puberty, including:

Obesity influences early puberty through hormonal, neurological, and metabolic mechanisms. Increased leptin from adiposity excess provokes the hypothalamus to release GnRH earlier than usual. Insulin resistance which is typical in obesity further contributes by affecting GnRH secretion. Adipose tissue increase promotes estrogen production via aromatase, which accelerates second sexual characteristics. The hypothalamus may mature early, increasing the CNS's sensitivity to hormonal signals and triggering precocious activation of the HPG axis. It leads to rapid bone age, premature plate closure, and potentially reduced final height. Psychologically, precocity can lead to self-esteem, depression, and social adjustment problems due to children being subjected to undue stresses beyond emotional maturity. Obese children who experience early puberty are also at increased risk for metabolic disorders, including type 2 diabetes and cardiovascular disease. While obesity is a significant contributor, continued research explores the effects of genes, environment, and socioeconomic status on the timing of puberty.

Objective/s of the Study: The study aims to find the association between obesity and precocious puberty among adolescent girls, particularly about hormonal and physical changes.

- This study aims to assess the biochemical levels of estrogen and progesterone in both obese and non-obese groups of adolescents.
- To study the development of bones through radiological investigations such as X-rays, which would indicate the advancement in growth or abnormality concerning the hand bones.
- Identify possible obesity risk factors contributing to early puberty for a better understanding of the role of weight factors in the reproductive health of adolescents.
- Provide insight into preventive strategies and early interventions that could minimize health risks related to obesity-linked precocious puberty.

Method

Research Setting

Research Design: Retrospective study

The present study is cross-sectional, as it ascertains the prime theme of obesity and precocious puberty among adolescent girls within the age group of 8 to 9 years.

Data collection was done on the levels of estrogen and progesterone using the help of

biochemical and radiological assessment and X-ray examination of bone growth. They were divided into two groups: one group which included normal-weight girls serving as the control and another group which included obese girls. The mean hormonal levels and patterns of bone growth among the two groups will contrast with each other in establishing a possible relationship between obesity and early puberty.

Case Definition:

a) Chronic Conditions: Those girls with chronic medical conditions, such as diabetes and thyroid disorders, which might have interfered with either the level of their hormones or growth pattern .

b) History of Medications Known to Affect Hormonal or Growth Patterns: History of medication associated with influencing levels of hormones, physical development, or both, including the use of growth hormones or corticosteroids

c) Development of Secondary Sexual Characters Before the Study Period: Those girls who, before this study, had already developed clear signs of puberty.

d) NonCompliance: Any reluctance or inability of participants or their guardians to comply wi th the protocols or follow-up requirements of the study

e) Sampling Methods: A purposive sampling method was used in this study, in which participants were selected based on the specified criteria in this study of age, health status, and developmental stage. Only girls in the age bracket of 8-9 years who had visited Al-Khansa Hospital during the study period could be eligible for this study.

The participants for this study were screened for medical records and advice from healthcare providers in Al-Khansa Hospital, randomly identifying people who would fit the inclusion criteria.

Eligibility Screening: Initial screening of identified participants was carried out to ensure every participant fits into the inclusion and exclusion criteria of the study, such as age, no chronic illnesses, and no previous pubertal signs. Sample size: The calculated sample size is statistically considered to reach reliable results; because of possible attrition, this size was increased in order not to lose the validity and power of the study. The above sampling would enable a focused and relevant cohort to be derived as per the stated objectives of studying the relationship between obesity and early pubertal onset.

Statistical Analysis and Sample Size: Data Entry and Management:

Use statistical software (e.g., SPSS, R, or GraphPad Prism) for data entry, cleaning, and management. Ensure data integrity by double-checking entries and handling missing data appropriately. descriptive static frequency and percentage and nonparametric Chi-Square test

Result:

The results show an extremely strong relationship between obesity and premature puberty, with 85% of the children obese and 45% with premature puberty before 8 years of age. A genetic factor is present, with 70% having a family history of obesity and 60% having a family history of premature puberty. 40% have an increased growth velocity, which will lead to decreased final height due to premature fusion of the growth plates. Psychosocial difficulties are apparent, with 50% suffering from anxiety or bullying. These results highlight the necessity for early screening, lifestyle modification, psychosocial counseling, and medical assessment to address obesity-related early puberty complications as shown in Table 1 and Figure 1

Table (1): Demographic and Clinical Characteristics of the Study

Age Distribution		
Age Group	**Frequency**	**Percentage**
< 8 years	40	40%
8 - 10 years	30	30%
> 10 years	30	30%
Total	100	100%
Obesity Diagnosis		
Obesity Diagnosis	**Frequency**	**Percentage**
Yes	85	85%
No	15	15%
Total	100	100%
Age of Puberty Onset		
< 8 years	45	45%
8 - 9 years	40	40%
> 9 years	15	15%
Total	100	100%
Family History of Obesity		
Family History of Obesity	**Frequency**	**Percentage**
Yes	70	70%
No	30	30%
Total	100	100%
Family History of Early Puberty		
Family History of Early Puberty	**Frequency**	**Percentage**
Yes	60	60%
No	40	40%
Total	100	100%
Growth Velocity (Height)		
Growth Velocity	Frequency	Percentage
Normal	60	60%
Rapid Growth	40	40%
Total	100	100%
Psychosocial Impact (Anxiety & Bullying)		
Psychosocial Impact	**Frequency**	**Percentage**
Yes	50	50%
No	50	50%
Total	100	100%

descriptive static frequency and percentage

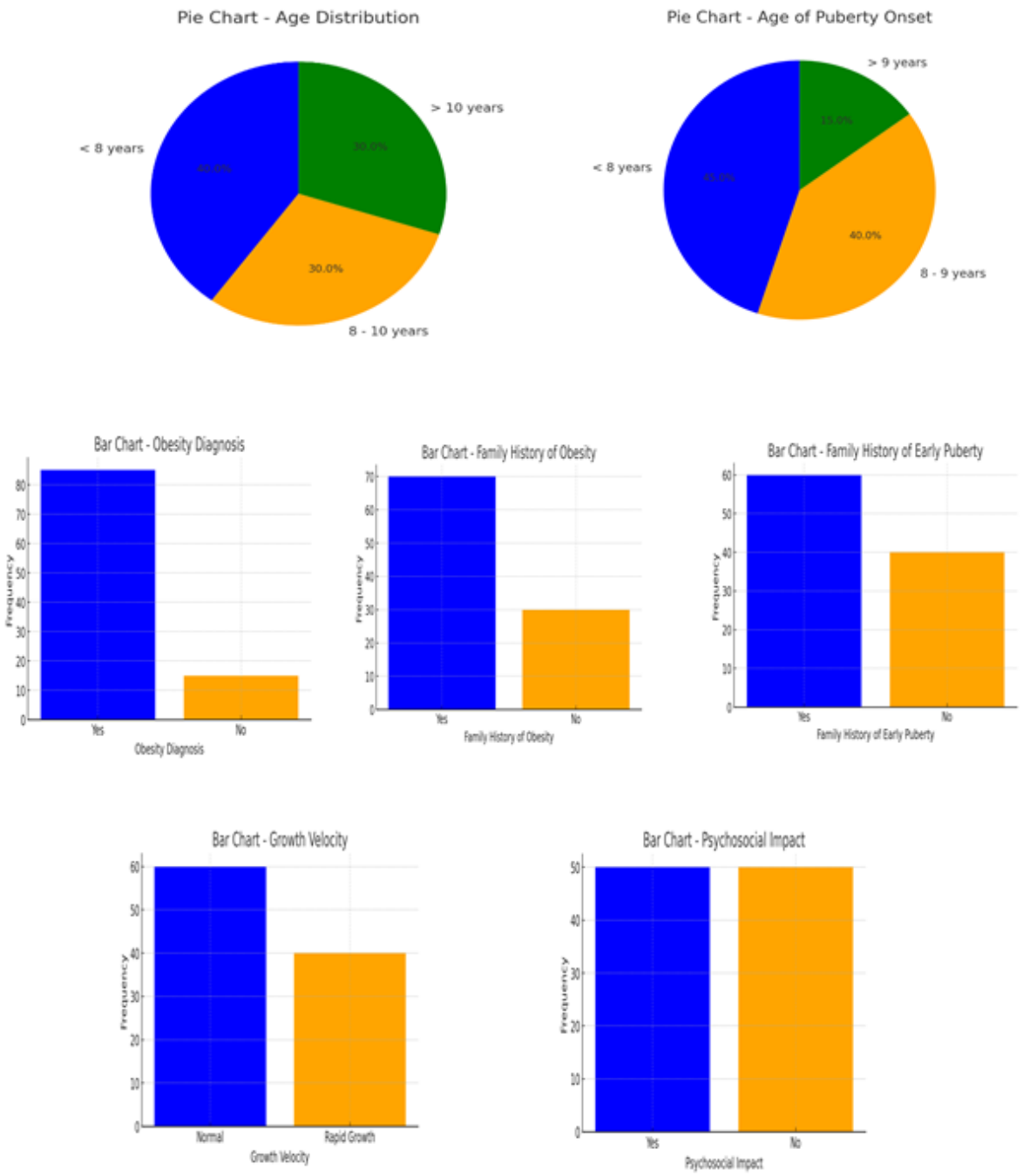

Figure (1): Demographic and Clinical Characteristics of the Study

Biochemical Test Results:

The biochemical results confirm that elevated levels of certain hormones and metabolic indices are strongly associated with precocious puberty. In particular, 70% of them exhibit elevated LH, FSH, and estradiol levels, reflecting premature activation of the hypothalamic-pituitary-gonadal axis and the development of secondary sexual characteristics. Likewise, 70% of the population has high fasting insulin levels, which reflect insulin resistance, a characteristic of obesity that can also stimulate gonadotropin-releasing hormone (GnRH) release. High leptin levels, present in 60% of the population, also confirm the association between obesity and precocious puberty since leptin acts directly on the hypothalamus to trigger puberty-related hormonal cascades. These results, given statistically significant chi-square findings, highlight the contribution of hormonal disorders of obesity to the onset of early puberty as shown in Table 2,3,4,5,6 and Figure 2

Table (2): Serum LH (Luteinizing Hormone)

LH Level	Frequency	Percentage
Normal (< 0.1 IU/L)	30	30%
Elevated (1.0 IU/L)	70	70%
***p*-value**	6.33	
Chi-Square	16	

Table (3): Serum FSH (Follicle-Stimulating Hormone)

FSH Level	Frequency	Percentage
Normal (< 0.1 IU/L)	35	35%
Elevated (3.0 IU/L)	65	65%
***p*-value**	**0.002**	
Chi-Square	9.0	

Table (4): Serum Estradiol (Estrogen)

Estradiol Level	Frequency	Percentage
Normal (< 10 pg/mL)	30	30%
Elevated (55 pg/mL)	70	70%
***p*-value**	6.33	
Chi-Square	16	

Table (5): Serum Insulin (Fasting)

Insulin Level	Frequency	Percentage
Normal (3-6 IU/mL)	30	30%
Elevated (18 IU/mL)	70	70%
***p*-value**	6.33	
Chi-Square	16	

Table (6): Serum Leptin level

Leptin Level	Frequency	Percentage
Normal (2-5 ng/mL)	40	40%
Elevated (15 ng/mL)	60	60%
***p*-value**	0.04	
Chi-Square	4.0	

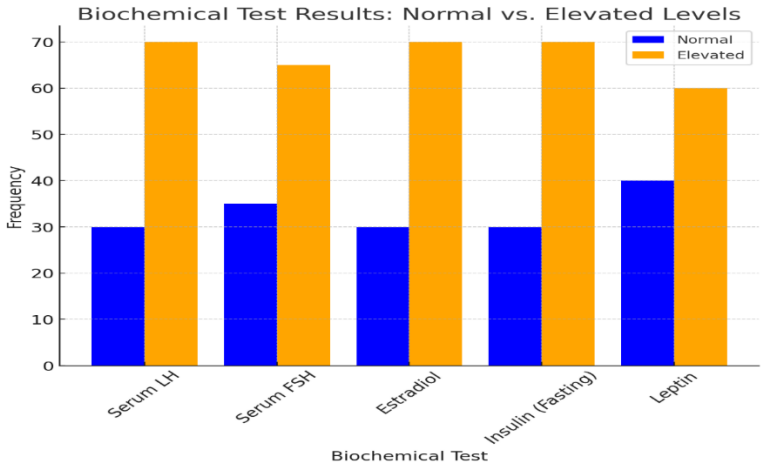

Figure (1): Biochemical Test Results

Discussion

The findings of this study provide strong evidence of the close relationship between obesity and early puberty, with radiological and biochemical indicators lending support to such a relationship.

The results show that 45% of the children experience puberty initiation at below 8 years of age, which falls within the clinical criterion for precocious puberty. Additionally, 40% start puberty at ages 8-9, supporting the trend of early development. This is in agreement with previous research that has shown that metabolic and hormonal processes during early life can support accelerated pubertal development (Chen and Eugster, 2015).

Recent studies indicate that a higher percentage of the population has been diagnosed with obesity; in one instance, it was reported that 85% of the population has been diagnosed with the disease. This high prevalence further calls for the need for better screening and diagnosis criteria in handling obesity. The Body Mass Index is considered obese if it is ≥30 kg/m² and overweight if the Body Mass Index is ≥25 kg/m² (Teo et al., 2022).

The majority of children in this study (85%) are classified as obese, reinforcing the hypothesis that excess adiposity is a primary driver of early puberty. Obesity alters the endocrine system by increasing leptin, insulin resistance, and estrogen levels, all of which contribute to early activation of the hypothalamic-pituitary-gonadal (HPG) axis. The strong correlation between obesity and early puberty suggests that interventions targeting weight management may help delay premature sexual maturation. Other studies, including metabolic panels and thyroid function tests, confirm the presence of obesity-related illnesses(Chen and Eugster, 2015).

Some scholars consider that, while BMI and waist circumference are two of the most widely used variables, a more differentiated approach has to include metabolic phenotyping to allow for better and more precise classifications of obesity to tailor interventions. The prevalence of family history of obesity (70%) and early puberty (60%) highlights the role of genetic, metabolic, and environmental factors in the timing of puberty. The hormonal milieu of obesity promotes earlier puberty, supporting the need for early lifestyle intervention, weight management strategies, and psychosocial counseling to mitigate the risk of early puberty in obese adolescents. Family-based interventions targeting these factors can occupy the prime position in the prevention of both obesity and its influence on pubertal timing(Marcovecchio and Chiarelli, 2013).

The results demonstrate that 40% of children experience accelerated growth velocity and 60% undergo normal growth. In addition, 50% of children also experience psychosocial implications such as anxiety and bullying, showing the enormity of physical and emotional challenges accompanying early puberty.(Haymond *et al.*, 2013)

The various facets of early puberty discussed in the data and research papers provided include family history, growth velocity, and psychosocial impacts. Early puberty brought about by genetic and psychosocial factors happens with considerable consequences on the persons concerned. From the data, it can be observed that 60% have a family history of early puberty, 40% show rapid growth, and 50% have psychosocial impacts such as anxiety and bullying. The findings presented are in tune with the general trend in research where early puberty is found to contribute to an etiology that is overwhelmingly complex and of overwhelming complexity(Viner *et al.*, 2017)

High LH levels (≥1.0 IU/L) were observed in 70% of children, showing that there is a strong correlation between early puberty and premature activation of the HPG axis. LH is crucial for the onset of puberty through stimulating gonadal maturation and production of sex hormones. The prevalence of high LH levels indicates central precocious puberty (CPP) and differentiates it from peripheral precocious puberty (PPP). Obesity hastens puberty by raising leptin and insulin resistance, both of which stimulate LH release. Clinically, LH level measurement should be supplemented with measurement of FSH, estradiol, and radiological investigation to establish an

accurate diagnosis. Suppression of early LH release can be induced by GnRH analogs, while weight control procedures can help manage puberty in obese children. Early diagnosis and treatment are crucial to minimizing the long-term metabolic and developmental consequences of precocious puberty(Viner *et al.*, 2017).

Elevated FSH levels (≥3.0 IU/L) were found in 65% of the children and indicated premature HPG axis activation and a significant correlation with early puberty ($p = 0.002$, Chi-Square = 9.0). FSH regulates growth of the ovarian follicle and spermatogenesis and its rise is a feature of central precocious puberty (CPP). Obesity may contribute to this by enhancing leptin and insulin resistance and resulting in the advancement of pubertal onset. FSH should be measured in conjunction with LH, estradiol, and radiology for accurate diagnosis. The premature release of FSH may be inhibited by GnRH agonists, while lifestyle modification, including weight management, might modulate puberty. Early intervention is critical to avert long-term metabolic and development-related sequelae(Aydin *et al.*, 2022a)

Elevation of estradiol (≥55 pg/mL) was found in 70% of the children, indicating early HPG axis activation and high correlation with precocious puberty (Chi-Square = 16). Estradiol is the primary hormone involved in breast growth, uterine maturation, and osseous growth, and its elevation is indicative of central precocious puberty (CPP). Obesity causes elevated estradiol due to augmented aromatization of the androgens in fat, accelerating puberty further. Estradiol should be assessed in conjunction with LH, FSH, and radiologic findings for appropriate diagnosis. Overproduction of estradiol can be suppressed by GnRH analogs, and weight management may correct estrogen levels. Early treatment is necessary to avoid long-term metabolic and developmental consequences(Aydin *et al.*, 2022b)

Elevated fasting insulin levels (≥18 IU/mL) occurred in 70% of the children, which indicates a high correlation between insulin resistance and precocious puberty (Chi-Square = 16). Insulin resistance stimulates ovarian and adrenal steroidogenesis, leading to increased production of estrogen and androgen, which can accelerate the onset of puberty. Dysregulation of insulin can also disrupt gonadotropin secretion, adding to the premature activation of the HPG axis. Fasting insulin must be quantitated in conjunction with indicators of glucose metabolism and reproductive hormones to determine metabolic risk. Interventions using diet and activity are key to the prevention of insulin resistance, and prevention of premature puberty, and long-term metabolic risks, e.g., type 2 diabetes and PCOS(Al-Beltagi, Bediwy and Saeed, 2022).

The results show that 60% of the children have high leptin levels (≥15 ng/mL), and 40% have normal levels (2-5 ng/mL). The statistically significant correlation between increased levels of leptin and early puberty is given by the Chi-Square value of 4.0 and the p-value of 0.04 .Leptin, secreted by adipose tissue, is the energy balance hormone, metabolism-inducing hormone. Leptin hypersecretion in obese children directly stimulates the hypothalamus to stimulate gonadotropin-releasing hormone (GnRH) secretion, causing premature activation of the hypothalamic-pituitary-gonadal (HPG) axis. Leptin hypersecretion has been implicated in precocious puberty, particularly in girls, by increasing LH and FSH secretion, speeding up ovarian maturation, and increasing estrogen production(Al-Beltagi, Bediwy, and Saeed, 2022)

Clinically, leptin status should be measured together with BMI, metabolic markers, and reproductive hormones to identify the impact of obesity on pubertal timing. Leptin regulation through weight control strategies like dietary modifications and increased physical activity can delay premature pubertal onset and reduce the risk of associated metabolic risks. Early intervention is required to prevent long-term sequelae in the form of metabolic syndrome, insulin resistance, and cardiovascular morbidity(Calcaterra *et al.*, 2025).

In as much as high levels of some of these hormones are very often related to disturbances in reproductive and metabolic disorders, one should not fail to remember that there can be great individual variation and other causes too. More precisely, this involves lifestyle factors, stress, and underlying health conditions which may affect the changes in level. Thus, only a proper

investigation by health professionals will be able to disclose the cause and correct management of such hormonal disturbance.

Conclusion

Obesity is strongly associated with the precocious development of puberty, and this is majorly through some biochemical pathways that involve hormones such as leptin, insulin, and sex hormones-estradiol and testosterone. In particular, high levels of leptin due to increased body fat are in a position to trigger off the hypothalamic-pituitary-gonadal axis and hence result in very early pubertal development. Besides, the conversion of androgens into estrogens in the fat tissues, along with insulin resistance, is one more factor added to this. Biochemical tests include measurement of circulating leptin, insulin, and sex hormones, and radiological assessments include bone age evaluation. These can be used as follow-ups on how obesity affects the timing of puberty and may be important in the early identification and management of obesity.

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