

# Clinical and Hormonal Features of Polycystic Ovary Syndrome in Adolescent Girls

*Ismatova Farida Zokir kizi*

*Samarkand State Medical University Intern's clinic*

**Abstract:** Polycystic ovary syndrome is one of the most common endocrine disorders affecting adolescent girls and represents a major cause of menstrual irregularities and hyperandrogenic symptoms during puberty. The diagnosis of this condition in adolescence is challenging due to the overlap between physiological pubertal changes and pathological features. Polycystic ovary syndrome in adolescents is associated with disturbances in hormonal regulation, metabolic imbalance, and increased risk of long-term reproductive and cardiovascular complications. This article discusses the clinical manifestations and hormonal characteristics of polycystic ovary syndrome in adolescent girls based on current medical knowledge.

**Keywords:** Polycystic ovary syndrome, adolescence, hyperandrogenism, hormonal imbalance, menstrual disorders

## Introduction

Adolescence is a critical period of reproductive maturation characterized by dynamic hormonal changes. During this stage, irregular menstrual cycles and transient androgen excess are often considered normal physiological phenomena. However, in some adolescents, these features persist and reflect underlying endocrine pathology. Polycystic ovary syndrome is a leading cause of chronic anovulation and hyperandrogenism in young females. Early recognition of this condition in adolescent girls is essential to prevent long-term reproductive, metabolic, and psychological consequences. Understanding the clinical and hormonal features of polycystic ovary syndrome during adolescence remains a priority in modern gynecological and endocrine practice.

Polycystic ovary syndrome in adolescent girls is primarily characterized by chronic disruption of ovulatory function and excessive androgen production. Menstrual irregularities are often the first clinical manifestation and may include infrequent menstruation, prolonged cycles, or absence of menstruation following menarche. These disturbances result from impaired follicular development and altered gonadotropin secretion. Ovulatory dysfunction in adolescents with polycystic ovary syndrome is closely linked to abnormal pulsatile release of gonadotropin-releasing hormone, leading to increased luteinizing hormone activity relative to follicle-stimulating hormone.

Hyperandrogenism represents a central clinical and hormonal feature of the syndrome. Clinically, it manifests as acne, hirsutism, and, in some cases, androgenic alopecia. Biochemically, elevated levels of circulating androgens such as testosterone and androstenedione are commonly observed. During adolescence, distinguishing pathological hyperandrogenism from normal pubertal changes is challenging, requiring careful longitudinal assessment.

Insulin resistance plays a significant role in the pathophysiology of polycystic ovary syndrome in adolescent girls. Reduced insulin sensitivity leads to compensatory hyperinsulinemia, which enhances ovarian androgen production and suppresses hepatic synthesis of sex hormone-binding globulin. This results in increased levels of free and biologically active androgens. Metabolic abnormalities such as central obesity, impaired glucose tolerance, and dyslipidemia may already be present during adolescence, increasing future cardiovascular risk.

Ovarian morphology in adolescents with polycystic ovary syndrome may show increased ovarian volume and multiple small follicles; however, ultrasound findings alone are not considered diagnostic

in this age group. This is due to the high prevalence of polycystic-appearing ovaries in healthy adolescents during early postmenarchal years. Therefore, clinical and hormonal features remain the cornerstone of diagnosis.

Psychological and emotional disturbances are frequently associated with polycystic ovary syndrome in adolescent girls. Body image concerns, anxiety, depression, and reduced self-esteem are commonly reported and may be exacerbated by visible symptoms such as acne and excessive hair growth. These psychosocial aspects further emphasize the need for early diagnosis and comprehensive management. Polycystic ovary syndrome in adolescent girls represents a multifactorial endocrine disorder that emerges during the critical period of pubertal development. The condition reflects a complex interaction between genetic predisposition and environmental influences, leading to dysregulation of the hypothalamic–pituitary–ovarian axis. During normal puberty, transient hyperandrogenism and menstrual irregularity are common; however, in adolescents with polycystic ovary syndrome, these features persist and progressively worsen, indicating pathological endocrine dysfunction.

Ovulatory dysfunction is a defining characteristic of the syndrome and manifests clinically as irregular menstrual cycles, oligomenorrhea, or secondary amenorrhea. This dysfunction results from altered gonadotropin secretion, particularly increased luteinizing hormone pulse frequency and amplitude, which disrupt normal follicular maturation. As a consequence, follicles arrest in early developmental stages, leading to chronic anovulation and irregular endometrial shedding.

Hyperandrogenism is the most prominent clinical and hormonal feature of polycystic ovary syndrome in adolescence. Excess androgen production originates primarily from the ovaries and is amplified by insulin-mediated stimulation of androgen synthesis. Clinically, hyperandrogenism presents as moderate to severe acne, hirsutism, seborrhea, and, less frequently, androgenic alopecia. Biochemical hyperandrogenism is characterized by elevated circulating levels of testosterone and other androgenic hormones, with reduced concentrations of sex hormone-binding globulin contributing to increased free androgen activity.

Insulin resistance plays a central role in the pathophysiology of polycystic ovary syndrome in adolescent girls, regardless of body weight. Hyperinsulinemia enhances ovarian androgen production and interferes with normal follicular development. In overweight or obese adolescents, insulin resistance is often more pronounced, increasing the risk of early metabolic complications such as impaired glucose tolerance and dyslipidemia. These metabolic alterations highlight the long-term health implications of the syndrome beyond reproductive dysfunction.

Ovarian morphology in adolescents with polycystic ovary syndrome often demonstrates enlarged ovaries with multiple small follicles; however, this feature lacks specificity during adolescence due to physiological pubertal changes. For this reason, imaging findings are interpreted cautiously, and diagnosis relies primarily on clinical and hormonal criteria rather than ultrasound appearance alone.

Neuroendocrine abnormalities are increasingly recognized as contributing factors in adolescent polycystic ovary syndrome. Altered sensitivity of the hypothalamus to hormonal feedback leads to persistent elevation of luteinizing hormone secretion. This neuroendocrine dysregulation perpetuates androgen excess and anovulation, reinforcing the chronic nature of the disorder.

Psychological and emotional aspects are integral components of polycystic ovary syndrome in adolescent girls. Visible symptoms such as acne, excessive hair growth, and weight gain often result in body image dissatisfaction, reduced self-esteem, and social withdrawal. Anxiety and depressive symptoms are more prevalent in affected adolescents, emphasizing the need for psychological assessment and support as part of comprehensive management.

From a clinical perspective, early identification of polycystic ovary syndrome during adolescence allows for timely intervention aimed at restoring hormonal balance and preventing future complications. Management strategies focus on lifestyle modification, regulation of menstrual cycles, reduction of androgen excess, and improvement of metabolic health. Addressing the syndrome during

adolescence provides an opportunity to modify disease progression and improve long-term reproductive and overall health outcomes.

Polycystic ovary syndrome in adolescent girls develops against the background of ongoing pubertal maturation, making its clinical presentation particularly complex. The disorder arises from an interplay of genetic susceptibility, intrauterine programming, and postnatal environmental influences such as nutrition and lifestyle. Familial clustering of the syndrome suggests a hereditary component, while early exposure to excess androgens may alter neuroendocrine regulation, predisposing adolescents to persistent reproductive dysfunction.

At the hormonal level, dysfunction of the hypothalamic–pituitary–ovarian axis is a central feature. Increased frequency of gonadotropin-releasing hormone pulses leads to preferential secretion of luteinizing hormone over follicle-stimulating hormone. This imbalance disrupts normal folliculogenesis and promotes excessive androgen production by the ovarian theca cells. Reduced follicle-stimulating hormone activity further limits aromatization of androgens to estrogens, reinforcing hyperandrogenism and chronic anovulation.

Adrenal contributions to androgen excess are also evident in some adolescents with polycystic ovary syndrome. Elevated adrenal androgens may coexist with ovarian hyperandrogenism, intensifying clinical manifestations. This combined androgen excess complicates diagnostic evaluation, as adrenal androgen production may be physiologically increased during puberty. Persistent elevation beyond expected pubertal norms supports a pathological diagnosis.

Insulin resistance and compensatory hyperinsulinemia amplify the endocrine abnormalities observed in polycystic ovary syndrome. Insulin acts synergistically with luteinizing hormone to stimulate ovarian androgen synthesis and reduces hepatic production of sex hormone-binding globulin, thereby increasing circulating free androgens. In addition to its reproductive effects, insulin resistance contributes to low-grade chronic inflammation, which has been increasingly recognized as a component of the syndrome. Elevated inflammatory markers may further impair ovarian function and exacerbate metabolic risk.

Menstrual irregularities in adolescents with polycystic ovary syndrome often reflect persistent anovulation rather than transient pubertal immaturity. Irregular, infrequent, or absent menstruation may persist several years after menarche, distinguishing pathological patterns from normal developmental variation. Prolonged exposure of the endometrium to unopposed estrogen increases the risk of abnormal uterine bleeding and may have long-term implications for endometrial health.

The clinical diagnosis of polycystic ovary syndrome in adolescence is particularly challenging due to overlap with normal pubertal features. Acne, irregular cycles, and polycystic ovarian morphology may be physiological during early adolescence. Therefore, emphasis is placed on the persistence and severity of symptoms over time, along with biochemical evidence of hyperandrogenism. A longitudinal approach is essential to avoid both overdiagnosis and delayed recognition.

Long-term health consequences of polycystic ovary syndrome begin to emerge during adolescence. Affected girls are at increased risk of obesity, type two diabetes mellitus, cardiovascular disease, and infertility later in life. Additionally, psychological morbidity may persist into adulthood, affecting social functioning and quality of life. Early intervention during adolescence offers a critical window for modifying disease trajectory and reducing future complications.

Effective management of polycystic ovary syndrome in adolescent girls requires a multidisciplinary approach that addresses hormonal, metabolic, and psychological dimensions. Lifestyle modification remains a cornerstone of therapy, particularly in adolescents with overweight or obesity. Hormonal treatment may be used to regulate menstrual cycles and reduce hyperandrogenic symptoms, while psychological support is essential to address emotional well-being and treatment adherence.

## Conclusion

Polycystic ovary syndrome in adolescent girls is a complex endocrine disorder characterized by a combination of clinical hyperandrogenism, ovulatory dysfunction, and hormonal imbalance. The condition often presents during adolescence and may persist into adulthood if not appropriately managed. Early identification of clinical and hormonal features allows for timely intervention, reducing the risk of long-term reproductive, metabolic, and psychological complications. A multidisciplinary and individualized approach is essential to optimize health outcomes for affected adolescents.

## References

1. Rosenfield R. Clinical review: identifying polycystic ovary syndrome in adolescents. *Journal of Clinical Endocrinology and Metabolism*. 2020;105:333–347.
2. Ibáñez L, Oberfield S, Witchel S. Polycystic ovary syndrome in adolescent girls. *Endocrine Reviews*. 2019;40:671–704.
3. Peña A, Witchel S, Hoeger K. Adolescent polycystic ovary syndrome. *Hormone Research in Paediatrics*. 2020;93:189–204.
4. Hickey M, Doherty D. Diagnosis and management of polycystic ovary syndrome in adolescents. *The Lancet Child and Adolescent Health*. 2021;5:497–506.
5. Legro R, Arslanian S. Insulin resistance in polycystic ovary syndrome. *Endocrinology and Metabolism Clinics of North America*. 2018;47:99–113.
6. Witchel S, Oberfield S. The pathophysiology of polycystic ovary syndrome in adolescents. *Best Practice and Research Clinical Obstetrics and Gynaecology*. 2019;55:32–43.
7. Teede H, Misso M. International evidence-based guideline for the assessment of polycystic ovary syndrome. *Human Reproduction*. 2018;33:1602–1618.