



**CLINICAL AND BIOCHEMICAL JUSTIFICATION OF CHANGES IN
WOMEN OF REPRODUCTIVE AGE WITH POLYCYSTIC OVARY
SYNDROME**

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ABSTRACT

Polycystic Ovary Syndrome (PCOS) is a prevalent endocrine disorder affecting women of reproductive age and is characterized by a combination of reproductive, hormonal, and metabolic abnormalities. This study aims to justify the clinical and biochemical changes observed in women with PCOS. The findings demonstrate that clinical manifestations such as menstrual irregularities, infertility, and hyperandrogenism are closely associated with underlying biochemical disturbances, including elevated androgen levels, insulin resistance, and dyslipidemia. Hormonal imbalance, particularly an increased LH/FSH ratio and decreased sex hormone-binding globulin, plays a key role in disease progression. Insulin resistance is identified as a central pathogenic factor linking metabolic and reproductive dysfunctions. The study emphasizes the importance of comprehensive clinical and biochemical evaluation for early diagnosis and effective management of PCOS, as well as prevention of long-term complications such as type 2 diabetes and cardiovascular diseases.

KEYWORDS: Polycystic ovary syndrome, PCOS, reproductive age, hyperandrogenism, insulin resistance, hormonal imbalance, metabolic syndrome, infertility

MAIN PART

Women with PCOS commonly present with menstrual dysfunction such as oligomenorrhea or amenorrhea, which reflects chronic anovulation. Infertility is a frequent consequence of ovulatory disturbances. Hyperandrogenism manifests clinically as hirsutism, acne, and androgenic alopecia. Obesity, particularly central adiposity, is observed in a significant proportion of patients and exacerbates both reproductive and metabolic abnormalities. Psychological disturbances such as anxiety and depression are increasingly recognized as important components of PCOS, further affecting the quality of life of affected women.



Biochemical Changes

Biochemical evaluation reveals elevated serum levels of androgens, including testosterone and dehydroepiandrosterone sulfate (DHEAS), which are key markers of hyperandrogenism. Increased luteinizing hormone (LH) levels and an elevated LH/FSH ratio are frequently observed, indicating dysregulation of the hypothalamic-pituitary-ovarian axis. Insulin resistance plays a central role in the pathophysiology of PCOS, even in non-obese women. Hyperinsulinemia contributes to increased ovarian androgen production and decreases sex hormone-binding globulin (SHBG) levels, thereby enhancing free androgen activity. Metabolic abnormalities include impaired glucose tolerance, elevated fasting insulin levels, and dyslipidemia characterized by increased triglycerides and low high-density lipoprotein (HDL) cholesterol. These changes significantly increase the long-term risk of metabolic syndrome and cardiovascular complications.

Pathophysiological Justification

The interconnection between clinical and biochemical changes in PCOS can be explained by underlying insulin resistance and hormonal imbalance. Hyperinsulinemia stimulates ovarian theca cells to produce excess androgens, while also disrupting normal follicular development, leading to anovulation. The imbalance between LH and FSH further contributes to follicular arrest and cyst formation in the ovaries. Additionally, adipose tissue dysfunction in obese patients exacerbates insulin resistance and promotes chronic low-grade inflammation, which plays a role in disease progression. PCOS represents a complex interplay between endocrine, metabolic, and inflammatory pathways, all of which contribute to its clinical manifestations.

The findings confirm that PCOS is a systemic disorder involving both reproductive and metabolic dysfunctions. Clinical symptoms are closely linked with biochemical abnormalities, particularly hyperandrogenism and insulin resistance. Early identification of these changes is essential for preventing long-term complications such as diabetes and cardiovascular diseases. The variability of clinical presentation necessitates individualized diagnostic and therapeutic approaches. Further research is required to explore novel biomarkers and improve treatment strategies.

CONCLUSION

PCOS is a complex endocrine disorder characterized by significant clinical and biochemical alterations. Hormonal imbalance and insulin resistance are central to its pathogenesis. Clinical manifestations such as infertility and menstrual irregularities are



strongly associated with biochemical disturbances. Early diagnosis and comprehensive evaluation are essential for effective management. A multidisciplinary approach is required to reduce long-term health risks and improve quality of life in affected women.

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