REPRODUCTIVE COMPLICATIONS IN POLYCYSTIC OVARY SYNDROME AND THEIR CLINICAL SIGNIFICANCE

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Abstract

Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age, with a significant impact on reproductive function. This thesis analyzes the main reproductive complications observed in PCOS, including chronic anovulation, infertility, menstrual irregularities, pregnancy-related complications, and their underlying pathogenetic mechanisms. Particular attention is paid to the clinical significance of these complications, as well as to the importance of early diagnosis and comprehensive management strategies in clinical practice.

Keywords:

Polycystic ovary syndrome; reproductive complications; infertility; chronic anovulation; hyperandrogenism; insulin resistance; menstrual dysfunction; pregnancy complications; endometrial dysfunction.

Significance of the Study

Polycystic ovary syndrome (PCOS) is a highly prevalent endocrine disorder among women of reproductive age and represents a major public health concern due to its substantial impact on female reproductive health and demographic indicators. PCOS is recognized as one of the leading causes of female infertility and occupies a central position in the structure of reproductive dysfunctions. In recent years, an increasing prevalence of PCOS among adolescents and young women has further emphasized the relevance of this issue.

Chronic anovulation, persistent menstrual cycle disturbances, and hyperandrogenism associated with PCOS significantly limit natural reproductive potential and often lead to long-term infertility. Moreover, insulin resistance and metabolic syndrome commonly observed in PCOS exacerbate reproductive complications and increase the risk of gestational diabetes, preeclampsia, and adverse perinatal outcomes during pregnancy.

In contemporary clinical practice, PCOS is frequently considered solely as a gynecological disorder, while its systemic endocrine and metabolic nature is often underestimated. This approach contributes to delayed diagnosis of reproductive

complications and reduced treatment effectiveness. Therefore, in-depth investigation of the pathogenetic mechanisms underlying reproductive complications in PCOS, assessment of their clinical significance, and development of individualized, comprehensive management strategies remain among the most pressing challenges in modern medicine.

Main Body

The development of reproductive complications in polycystic ovary syndrome (PCOS) is associated with complex, multilevel pathogenetic mechanisms involving hormonal, metabolic, and molecular disturbances that are closely interconnected. Central to the pathophysiology of PCOS is dysfunction of the hypothalamic–pituitary–ovarian axis.

An increased secretion of luteinizing hormone (LH) relative to follicle-stimulating hormone (FSH) leads to impaired folliculogenesis. As a result, maturation of primary and secondary follicles is arrested, dominant follicle formation does not occur, and chronic anovulation develops. Clinically, this condition manifests as oligomenorrhea or amenorrhea and represents one of the primary causes of infertility in women with PCOS.

Hyperandrogenism is a hallmark pathophysiological feature of PCOS. Enhanced androgen production by ovarian theca cells not only suppresses ovulation but also disrupts endometrial hormonal receptor sensitivity. Imbalance between proliferative and secretory transformations of the endometrium reduces implantation potential and contributes to early pregnancy loss.

Metabolic disturbances, particularly insulin resistance and compensatory hyperinsulinemia, play a crucial role in the progression of reproductive complications in PCOS. Insulin enhances ovarian androgen synthesis, thereby aggravating hyperandrogenism. Simultaneously, reduced hepatic production of sex hormone-binding globulin (SHBG) increases circulating levels of biologically active free androgens, forming a pathological feedback loop that further impairs ovulatory function. Even when pregnancy is achieved, women with PCOS remain at increased reproductive risk. Numerous studies demonstrate higher rates of early spontaneous miscarriage, gestational diabetes mellitus, hypertensive disorders of pregnancy, preeclampsia, and preterm birth compared to the general population. These complications are associated with placental dysfunction, chronic low-grade inflammation, and persistent metabolic imbalance.

Additionally, prolonged anovulation and progesterone deficiency in PCOS result in continuous estrogen stimulation of the endometrium. This condition significantly

increases the risk of endometrial hyperplasia and, in the long term, endometrial carcinoma, posing a serious clinical concern for women of reproductive age.

Thus, reproductive complications in PCOS extend beyond infertility and encompass adverse pregnancy and perinatal outcomes, underscoring the necessity for early diagnosis, thorough pathogenetic evaluation, and individualized, multidisciplinary management.

Conclusion

Polycystic ovary syndrome is a clinically significant endocrine disorder characterized by a wide spectrum of reproductive complications that substantially impair women's quality of life and reproductive potential. Chronic anovulation, infertility, and pregnancy-related complications represent the primary clinical consequences of PCOS. Early identification of these disturbances and implementation of personalized, pathogenetically based therapeutic approaches are essential for improving reproductive outcomes. Comprehensive assessment of hormonal and metabolic status in women with PCOS should therefore be considered a priority in modern clinical practice.

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