

Short Notes on Polycystic Ovary Syndrome and its Complications

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Introduction

Polycystic Ovary Syndrome (PCOS) is a common multisystem disease with reproductive, metabolic, and psychological abnormalities, affecting more than 8-13 percent of women of childbearing age worldwide, with 5.6 percent prevalence in China. The clinical manifestations of PCOS are highly heterogeneous, primarily characterised by irregular menstruation or amenorrhea, hyperandrogenemia, polycystic ovarian change, and, to varying degrees, Insulin Resistance (IR), obesity, and anxiety and depression. In the early stages, PCOS is frequently complicated by infertility and poor pregnancy outcomes, but in the long run, the incidence of endometrial cancer, type 2 diabetes, and cardiovascular disease steadily rises, wreaking havoc on women's physical and mental health.

So far, there is no effective cure for PCOS, and the majority of treatment is symptomatic, including lifestyle changes, menstrual cycle adjustments, androgen reduction, and metabolic improvement. PCOS patients with ovulatory disorders were primarily treated with second-half cycle progesterone or a combination of short-acting oral contraceptives, but they were unable to restore PCOS spontaneous ovulation and were prone to relapse after drug withdrawal. Metformin [1-3] is used to treat PCOS metabolic disorder, but its ovulation rate is low, and it causes side effects such as diarrhoea, nausea, fatigue, headache, and so on, resulting in poor patient compliance. Letrozole and clomiphene are appropriate for PCOS patients who require fertility treatment.

Second, continuous stimulation of multiple follicles will result in follicular hyper stimulation syndrome. It primarily manifests as ovarian cystic enlargement, increased capillary permeability, and systemic edema, which is one of the most serious gynaecological diseases. Long-term clomiphene use also raises the risk of ovarian cancer, though the mechanism is unknown. It may be related to increased FSH. As can be seen, current treatment drugs can only target a specific pathological link of PCOS, and they have limitations. As a result, a multi-target, safe, and effective drug is desperately needed to provide a new idea for treating PCOS. Letrozole and clomiphene are appropriate for PCOS patients who require fertility treatment. Letrozole inhibits oestrogen synthesis, while clomiphene binds oestrogen receptors competitively, lowering oestrogen levels while increasing follicle-stimulating hormone (FSH) release and promoting follicular development. This low oestrogen status raises the chances of miscarriage or multiple pregnancies.

About the Study

Ovulation disorder is one of the most common types of PCOS, accounting

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for approximately 75% of cases, and is the leading cause of infertility in people with PCOS. At the moment, the mechanism of ovulation disorders is unknown. The hypothalamic-pituitary-ovarian (HPO) axis regulates follicle development, and abnormal secretion of hormones regulated by this gonadal axis affects follicular development. Granulosa cells are also a layer of somatic cells that surround oocytes and provide energy for follicular development and oocyte maturation via glycolysis.

According to research, the granulosa cell layers surrounding PCOS follicles exhibit atresia, hypertrophy, and degeneration, all of which are important causes of PCOS follicle development retardation. Hyperandrogenism is a significant phenotype of PCOS, and more than 80% of women with symptoms of androgen excess have PCOS. The pituitary gland is more sensitive to gonadotropin-releasing hormones [4,5] in PCOS patients, resulting in increased LH secretion and induction of androgen synthesis in theca cells. Furthermore, hyperandrogenism and IR interact as both a cause and an effect. Excess insulin can promote serum androgen synthesis, which in turn can promote adipose tissue decomposition, increase the production of free fatty acids and inflammatory factors, and aggravate IR. Excess androgen can also reduce granulosa cell sensitivity to follicle-stimulating hormone (FSH), resulting in stalled follicle development and a lower pregnancy rate. Finally, hyperandrogenemia is the primary lesion of PCOS.

Conclusion

Fatty acid oxidation is an important physiological process that occurs primarily in mitochondria to maintain energy balance in the body. By activating the AMPK pathway and accelerating fatty acid oxidation, QUR was found to reduce fat formation, accelerate fat decomposition, and maintain lipid homeostasis and energy balance in obese mice. Furthermore, QUR can increase fatty acid oxidation and improve lipid metabolism by upregulating the expression of the PPAR- gene. In PCOS patients, serum levels of pro-inflammatory cytokines such as Tumour Necrosis Factor (TNF) and Interleukin-6 (IL-6) were higher than in healthy subjects, indicating a state of chronic inflammation. The gut contains approximately 10¹⁴ resident microbes, with Firmicutes and Bacteroidetes accounting for more than 90% of the total. According to studies, the and diversity of intestinal flora decreased in PCOS, and the proportion of Firmicutes and Bacteroidetes was unbalanced.

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