ANALYSIS OF RISK FACTORS IN THE DEVELOPMENT OF POLYCYSTIC OVARY SYNDROME

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Introduction (Background). Polycystic ovary syndrome (PCOS) is one of the most common endocrine disorders among women of reproductive age, with an estimated prevalence of approximately 6–13% [4]. It is characterised by menstrual irregularities, hyperandrogenism, and infertility. In recent years, PCOS has increasingly been recognised not merely as a gynaecological condition but also as a metabolic disorder. Insulin resistance has been identified in many affected women, including those with a normal body weight [2, 3]. This condition is associated with an increased risk of type 2 diabetes mellitus and cardiovascular disease. Therefore, understanding the relationship between PCOS and metabolic disturbances is essential for early diagnosis and appropriate clinical management.

Keywords: polycystic ovary syndrome, risk factors, hyperinsulinaemia, prevention.

Objective. To analyse the risk factors associated with the development of PCOS and formulate recommendations for its prevention.

Risk Factors. Genetic predisposition and metabolic factors play important roles in the development of PCOS. A family history of PCOS or type 2 diabetes mellitus is associated with an increased risk. Insulin resistance and hyperinsulinaemia may stimulate androgen production and contribute to ovulatory dysfunction. Excess body weight, particularly visceral adiposity, can reduce insulin sensitivity and exacerbate metabolic disturbances; however, PCOS can also occur in women with a normal body weight. Physical inactivity and an unbalanced diet may increase metabolic risk, although they are not considered the sole or direct causes of PCOS. Sleep disturbances and chronic stress may also adversely affect metabolic health. The presence of these factors does not necessarily mean that PCOS will develop; rather, they should be evaluated in conjunction with individual susceptibility and the patient's clinical characteristics.

Materials and Methods. International clinical recommendations, including the 2023 International Evidence-based Guideline for the Assessment and Management of Polycystic Ovary Syndrome, and relevant scientific publications were reviewed. Analytical, comparative, and systematic approaches were used to evaluate and synthesise the available evidence.

Results and Discussion

1. Epidemiology and Diagnosis**.** PCOS is diagnosed according to the Rotterdam criteria when at least two of the following three features are present: oligo-ovulation or anovulation; clinical or biochemical hyperandrogenism; and polycystic ovarian morphology on ultrasonography [5]. Other potential causes, including thyroid disorders, hyperprolactinaemia, and adrenal disorders, must be excluded.

2. Risk Factors for the Development of PCOS. The aetiology of PCOS is multifactorial, with genetic predisposition and alterations in hormonal regulation playing important roles. Insulin resistance and compensatory hyperinsulinaemia may enhance ovarian androgen production and aggravate the clinical manifestations of the syndrome. Excess visceral adiposity and a sedentary lifestyle increase the risk of metabolic disturbances; however, PCOS may also occur in women with a normal body weight. A family history of PCOS or type 2 diabetes mellitus, as well as a predisposition to impaired glucose

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metabolism, is also clinically relevant. These factors do not necessarily cause the syndrome independently; rather, they interact and contribute to its development and clinical presentation.

3. Mechanisms of Insulin Resistance**.** In PCOS, abnormalities in post-receptor insulin signalling have been described, including excessive serine phosphorylation within components of the insulin-signalling pathway, which may impair intracellular insulin action [2]. In response to reduced insulin sensitivity, the pancreas compensates by increasing insulin secretion, resulting in hyperinsulinaemia. Insulin resistance is common among women with PCOS and tends to be more pronounced in those with obesity; however, it may also occur in lean women [1, 3].

4. Hyperinsulinaemia and Hyperandrogenism. An important feature of PCOS is that ovarian steroidogenesis may remain responsive to insulin despite insulin resistance in other tissues. Consequently, elevated insulin levels may continue to stimulate androgen synthesis. In conjunction with luteinising hormone (LH), insulin enhances androgen production by ovarian theca cells. In the liver, it may also reduce the synthesis of sex hormone-binding globulin (SHBG), thereby increasing circulating free testosterone concentrations [3]. Excess androgen production can disrupt follicular development, impair ovulation, and contribute to infertility. Furthermore, hyperandrogenism may promote visceral adiposity and exacerbate insulin resistance, creating a self-reinforcing pathophysiological cycle.

Table 1. The Role of Hyperinsulinaemia in the Pathogenesis of Polycystic Ovary Syndrome (PCOS)

Site of action	Effects of hyperinsulinaemia	Outcome
Ovary (theca cells)	Increased androgen synthesis in synergy with luteinising hormone (LH)	Hyperandrogenism and follicular growth arrest
Liver	SHBG sintezini kamaytiradi	Increased free testosterone levels
Adipose tissue	Promotes visceral fat accumulation	Exacerbation of insulin resistance

5. Metabolic Consequences. Women with polycystic ovary syndrome (PCOS) have a substantially higher risk of impaired glucose tolerance and type 2 diabetes mellitus than women without the syndrome [6]. Dyslipidaemia, arterial hypertension, metabolic syndrome, metabolic dysfunction-associated steatotic liver disease (MASLD), and obstructive sleep apnoea are also frequently observed. Furthermore, the risk of gestational diabetes mellitus is increased during pregnancy.

6. Assessment of Insulin Resistance. All women with PCOS should undergo assessment of glycaemic status, lipid profile, and, where appropriate, a 75-g oral glucose tolerance test (OGTT) [1]. The homeostatic model assessment of insulin resistance (HOMA-IR), calculated as fasting plasma glucose multiplied by fasting insulin and divided by 22.5 when glucose is expressed in mmol/L, is widely used in research. Although the hyperinsulinaemic-euglycaemic clamp is considered the reference method for assessing insulin sensitivity, its application is primarily limited to research settings.

7. Treatment. The first-line approach involves lifestyle modification. Regular physical activity and, where appropriate, a 5–10% reduction in body weight can improve insulin sensitivity, promote menstrual cycle regularity, and reduce androgen concentrations [1]. Metformin may be used to improve metabolic parameters, particularly in women with obesity or impaired glucose tolerance. Combined hormonal contraceptives can help regulate menstrual cycles and alleviate the clinical manifestations of hyperandrogenism. For women seeking pregnancy, letrozole is the recommended first-line pharmacological agent for ovulation induction. Treatment should be individualised according to the patient's clinical presentation, reproductive goals, and metabolic profile, under appropriate medical supervision.

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Conclusion.

1) Insulin resistance and compensatory hyperinsulinaemia are important pathophysiological components of PCOS and contribute to hyperandrogenism and anovulation.

2) Hyperandrogenism and obesity may further exacerbate insulin resistance, indicating a bidirectional relationship between hormonal and metabolic disturbances.

3) All women with PCOS should undergo assessment of their glycaemic status to facilitate the early detection of abnormalities in glucose metabolism.

4) Lifestyle modification and timely management of metabolic disturbances are important strategies for reducing the risk of type 2 diabetes mellitus and cardiovascular complications.

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