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INSULIN RESISTANCE IN POLYCYSTIC OVARY SYNDROME: CLINICAL IMPLICATIONS AND IMPACT ON QUALITY OF LIFE — A NARRATIVE REVIEW

Martyna Sienicka *(Corresponding Author, Email: martyna.salamonska@o2.pl)*
MD, Instytut „Centrum Zdrowia Matki Polki" w Łodzi, Poland
ORCID ID: 0009-0009-8164-6236

Weronika Winiarska
MD, University Orthopaedic and Rehabilitation Hospital in Zakopane, Zakopane, Poland
ORCID ID: 0009-0004-2731-615X

Wiktoria Bełcikowska-Skowron
MD, Dr. Tytus Chałubiński Radom Specialist Hospital, Radom, Poland
ORCID ID: 0009-0001-1789-1845

Aleksandra Siegmund
MD, Medical University of Silesia in Katowice, Katowice, Silesia, Poland
ORCID ID: 0000-0002-6162-6096

Marzena Chrapczyńska
MD, Maria Skłodowska-Curie Provincial Specialist Hospital in Zgierz, Poland
ORCID ID: 0000-0002-3876-292X

Agnieszka Filarecka
MD, Tytus Chałubiński District Hospital in Zakopane, Zakopane, Poland
ORCID ID: 0009-0005-4200-7635

Piotr Przybyszewski
MD, Dr. Tytus Chałubiński District Hospital in Zakopane, Poland
ORCID ID: 0009-0000-3658-494X

Emilia Przybyszewska
DMD, "AVO-DENTAL" Dentistry & Aesthetic Medicine in Zakopane, Poland
ORCID ID: 0009-0002-4393-1967

Julia Gatniejewska
MD, Independent Researcher, Poland
ORCID ID: 0009-0001-1463-7497

Paulina Prudzic
MD, Independent Researcher, Poland
ORCID ID: 0009-0001-8746-8540

ABSTRACT

Polycystic ovary syndrome (PCOS) is a multifaceted endocrine and metabolic condition affecting women of reproductive age. While historically conceptualized as a reproductive disorder, current evidence indicates that PCOS extends beyond ovulatory dysfunction and hyperandrogenism to include significant metabolic, psychological, and quality of life consequences. Insulin resistance represents a core pathophysiological feature of the syndrome, contributing to hyperinsulinemia, exacerbation of androgen excess, menstrual irregularities, and long term metabolic risk. This narrative review synthesizes current literature on the mechanisms and clinical implications of insulin resistance in PCOS, drawing from peer reviewed studies, systematic reviews, meta analyses, and international guidelines.

Evidence shows that insulin resistance is prevalent across PCOS phenotypes, including in women without obesity, and is strongly associated with impaired glucose metabolism, dyslipidemia, metabolic syndrome, and increased cardiometabolic vulnerability. Psychological burden is also substantial, with many women reporting depressive and anxiety symptoms, body image concerns, fertility related distress, and reduced overall wellbeing. Interactions between metabolic dysfunction, emotional health, and lifestyle factors further contribute to diminished health related quality of life.

Collectively, these findings highlight the need for early metabolic assessment, individualized therapeutic strategies, and interdisciplinary care models addressing both physiological and psychosocial dimensions of PCOS. A comprehensive approach to insulin resistance may improve reproductive, metabolic, and psychological outcomes and support more patient centered long term management.

KEYWORDS

Polycystic Ovary Syndrome; Insulin Resistance; Quality Of Life; Hyperandrogenism; Metabolic Risk; Interdisciplinary Care

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1. Introduction

Polycystic ovary syndrome (PCOS) is a complex clinical condition characterized by reproductive, endocrine, metabolic, and psychological features (Azziz et al., 2016; Ehrmann, 2005; Escobar-Morreale, 2018). Although historically viewed through the lens of reproductive dysfunction, recent research emphasizes its broader significance as a chronic, multidimensional disorder that affects long-term metabolic and emotional health (Cooney et al., 2017; Teede et al., 2023)

Insulin resistance is recognized as a fundamental metabolic component of PCOS. It contributes to hyperinsulinemia, intensifies androgen production, and affects hepatic sex hormone-binding globulin synthesis, thereby amplifying the endocrine phenotype of the syndrome (Barber et al., 2015; Diamanti-Kandarakis & Dunaif, 2012). Importantly, insulin resistance may be present regardless of body weight, although excess adiposity can exacerbate metabolic abnormalities (Barber & Franks, 2021; Cassar et al., 2016).

The metabolic consequences of insulin resistance include increased risk of impaired glucose tolerance, type 2 diabetes, dyslipidemia, and components of metabolic syndrome (Lim et al., 2019; Wild et al., 2010). Psychological and quality-of-life consequences are also substantial, with many women reporting depressive and anxiety symptoms, concerns about body image and fertility, and reduced overall wellbeing (Bazarganipour et al., 2014; Cooney et al., 2017; Hollinrake et al., 2007).

PCOS also exhibits significant phenotypic variability, influenced by differences in adiposity, androgen concentrations, and metabolic profiles (Azziz et al., 2016; Goodarzi et al., 2011; Teede et al., 2023). Delayed diagnosis, fragmented care, and limited recognition of emotional burden can further worsen long-term outcomes (Azziz et al., 2016; Teede et al., 2018, 2023).

In addition to its relevance for individual clinical care, insulin resistance in PCOS also has implications at the level of healthcare systems and public health. (Cassar et al., 2016; Diamanti-Kandarakis & Dunaif, 2012). Given this complexity, PCOS requires an interdisciplinary, patient-centered approach (Azziz et al., 2016; Escobar-Morreale, 2018; Teede et al., 2018). This narrative review outlines the mechanisms and clinical implications of insulin resistance in PCOS, emphasizing its influence on metabolic health, reproductive function, quality of life, and therapeutic decision-making.

2. Methodology

This study was designed as a narrative review focused on the role of insulin resistance in polycystic ovary syndrome (PCOS), with particular attention to its clinical implications and its impact on quality of life. The review aimed to synthesize current knowledge from key clinical, pathophysiological, and guideline-based publications relevant to the topic.

Literature searches primarily included PubMed-indexed studies published in English between approximately 2000 and 2025. Particular emphasis was placed on international guidelines, systematic reviews, meta-analyses, and clinically influential studies related to insulin resistance and PCOS.

The literature included peer-reviewed journal articles, systematic reviews, meta-analyses, consensus statements, and international evidence-based guidelines addressing insulin resistance in PCOS. Particular emphasis was placed on studies concerning the pathophysiology of insulin resistance, its metabolic and reproductive consequences, methods of assessment, psychological burden, quality-of-life outcomes, and therapeutic approaches.

The selection of literature was based on relevance to the subject of the review and the scientific importance of the cited publications. Priority was given to widely cited and clinically significant papers, including foundational studies on insulin resistance measurement and major international guidelines for PCOS assessment and management. The analysis also included publications addressing depression, anxiety, obesity, metabolic syndrome, and interventions aimed at improving insulin sensitivity in women with PCOS.

Because this study is a narrative rather than a systematic review, the objective was not to provide an exhaustive quantitative synthesis of all available evidence, but rather to present an interpretative and clinically relevant overview of the current literature. The collected studies were analyzed thematically and grouped into the following domains: pathophysiology of insulin resistance in PCOS, clinical and metabolic implications, quality of life and psychological burden, and therapeutic strategies.

3. Results

Current evidence consistently identifies insulin resistance as one of the central metabolic features of polycystic ovary syndrome. Available evidence indicates that insulin resistance contributes to both the endocrine and metabolic manifestations of PCOS and is closely associated with impaired glucose metabolism, obesity, reproductive dysfunction, and reduced quality of life. The findings of the reviewed publications were grouped into six main thematic areas.

3.1. Insulin resistance as a central feature of PCOS

Insulin resistance is a defining metabolic characteristic of PCOS and contributes significantly to its clinical heterogeneity (Azziz et al., 2016; Diamanti-Kandarakis & Dunaif, 2012; Escobar-Morreale, 2018). Although the severity of insulin resistance varies across phenotypes, numerous studies suggest that it is present in a substantial proportion of affected women, including some who are not overweight (Cassar et al., 2016; Diamanti-Kandarakis & Dunaif, 2012; Moghetti & Tosi, 2021). Evidence from clamp studies demonstrates impaired insulin sensitivity even among lean women with PCOS, suggesting intrinsic defects in insulin signaling (Cassar et al., 2016). This is particularly important clinically, because it argues against the assumption that metabolically relevant PCOS exists only in women with obesity. Mechanistic research identifies abnormalities in skeletal muscle and adipose tissue insulin signaling pathways, including post-receptor disturbances (Diamanti-Kandarakis & Dunaif, 2012; Spritzer et al., 2015). There are abnormalities in insulin signaling in skeletal muscle of women with PCOS, supporting the concept of both intrinsic and acquired defects (Corbould et al., 2005). These mechanisms interact with adiposity, inflammation, and genetic predisposition, producing varying degrees of metabolic dysfunction (Diamanti-Kandarakis & Dunaif, 2012; Moghetti & Tosi, 2021). The major mechanisms linking insulin resistance with endocrine and metabolic manifestations of PCOS are summarized in Table 1.

Table 1. Key mechanisms linking insulin resistance to clinical manifestations of PCOS

Mechanism	Clinical Consequence
Hyperinsulinemia	Increased ovarian androgen production
Reduced SHBG synthesis	Increased free androgen levels
Chronic low-grade inflammation	Worsened insulin signaling
Obesity and visceral adiposity	Aggravation of metabolic dysfunction
Insulin signaling defects	Reduced glucose utilization

Summary of selected mechanisms contributing to endocrine and metabolic abnormalities in women with PCOS.

Abbreviations: SHBG — sex hormone-binding globulin; HOMA-IR — homeostatic model assessment for insulin resistance.

Obesity remains an important modifier of insulin resistance rather than its sole cause. Overweight and central obesity are more common among women with PCOS than in the general female population, and excess adiposity strongly aggravates insulin resistance, hyperinsulinemia, and metabolic risk (Barber & Franks, 2021; Lim et al., 2012). Adipose tissue dysfunction, altered adipokines, and chronic low-grade inflammation also appear to contribute to worsening insulin sensitivity in this syndrome (Spritzer et al., 2015).

3.2. Pathophysiological links between insulin resistance and hyperandrogenism

Studies consistently demonstrate that insulin resistance has direct endocrine consequences in PCOS. Compensatory hyperinsulinemia stimulates ovarian androgen production and acts synergistically with luteinizing hormone to increase theca cell steroidogenesis (Barber et al., 2015; Diamanti-Kandarakis & Dunaif, 2012). This results in increased circulating androgen concentrations and contributes to the characteristic reproductive phenotype of the syndrome.

Insulin also suppresses hepatic synthesis of sex hormone-binding globulin, leading to higher levels of free biologically active androgens. This mechanism worsens manifestations such as hirsutism, acne, and ovulatory dysfunction. As a result, insulin resistance does not only coexist with hyperandrogenism but actively contributes to its maintenance and clinical expression (Azziz et al., 2016; Goodarzi et al., 2011).

This interaction creates a vicious cycle. Hyperinsulinemia aggravates androgen excess, while androgen excess may in turn worsen abdominal adiposity and metabolic dysfunction (Diamanti-Kandarakis & Dunaif, 2012; Spritzer et al., 2015). The reciprocal relationship between endocrine and metabolic disturbance is one reason why PCOS presents with such broad heterogeneity and why treatment strategies focused only on reproductive symptoms may fail to address the full burden of disease (Azziz et al., 2016; Escobar-Morreale, 2018; Teede et al., 2018).

The literature also suggests that inflammation and adipose tissue dysfunction may act as mediators connecting insulin resistance and androgen-related abnormalities. Chronic low-grade inflammation, commonly observed in women with obesity and PCOS, may intensify insulin signaling defects and further contribute to ovarian dysfunction (Spritzer et al., 2015). Thus, the endocrine phenotype of PCOS should be interpreted within a broader metabolic context.

3.3. Clinical implications: metabolic and reproductive consequences

The metabolic consequences of insulin resistance in PCOS are substantial. Multiple studies have shown increased prevalence of impaired glucose tolerance, dysglycemia, type 2 diabetes, and metabolic syndrome among women with PCOS compared with women without the syndrome (Legro et al., 1999; Lim et al., 2019). These findings support the view that PCOS is a clinically relevant marker of long-term metabolic vulnerability.

There is increased risk of impaired glucose tolerance and type 2 diabetes in women with PCOS, especially in the presence of obesity and other metabolic risk factors (Legro et al., 1999). More recent syntheses have reinforced these findings, indicating that metabolic syndrome is significantly more common in this patient population (Lim et al., 2019). Given the chronic nature of these risks, metabolic screening should form an integral part of long-term management rather than being limited to initial diagnosis.

Insulin resistance also has major reproductive consequences. Hyperinsulinemia promotes ovarian androgen excess, disrupts follicular maturation, and contributes to chronic anovulation, irregular menstrual cycles, and subfertility (Azziz et al., 2016; Diamanti-Kandarakis & Dunaif, 2012). These mechanisms explain

why improving insulin sensitivity may positively affect ovulatory function and menstrual regularity in at least some women with PCOS.

Available data additionally suggest an increased cardiovascular risk burden in PCOS, even though the exact magnitude of long-term cardiovascular event risk may vary between studies. Consensus recommendations emphasize the importance of assessing blood pressure, lipid abnormalities, glucose metabolism, adiposity, and family history to identify women requiring closer follow-up and preventive intervention (Teede et al., 2023; Wild et al., 2010).

Taken together, these findings indicate that insulin resistance in PCOS should not be understood as an isolated biochemical abnormality. Instead, it functions as a clinical bridge connecting reproductive symptoms with future metabolic and cardiovascular morbidity.

3.4. Assessment of insulin resistance in women with PCOS

Current clinical evidence shows that assessment of insulin resistance in PCOS remains methodologically challenging. The euglycemic hyperinsulinemic clamp is widely regarded as the gold standard for direct measurement of insulin sensitivity and has been used extensively in research investigating metabolic abnormalities in PCOS (Cassar et al., 2016; DeFronzo et al., 1979). However, because it is time-consuming, technically demanding, and expensive, it is not suitable for routine clinical practice.

For this reason, most clinical settings rely on indirect or surrogate measures. Commonly used approaches include fasting insulin concentration, the homeostasis model assessment of insulin resistance (HOMA-IR), and oral glucose tolerance test-derived indices such as the Matsuda index (Matsuda & DeFronzo, 1999; Matthews et al., 1985). Additional markers, including the triglyceride-glucose product, have also been proposed as practical surrogates of insulin resistance in metabolic medicine (Simental-Mendía et al., 2008).

Despite their usefulness, surrogate indices have limitations. Their diagnostic performance may vary according to BMI, ethnicity, age, and PCOS phenotype. (Matsuda & DeFronzo, 1999; Matthews et al., 1985; Simental-Mendía et al., 2008). Moreover, there is no universally accepted cut-off value that applies equally well to all populations of women with PCOS (Cassar et al., 2016; Diamanti-Kandarakis & Dunaif, 2012). This heterogeneity limits the ability to use a single numerical index as a definitive diagnostic standard. (Cassar et al., 2016; Moran et al., 2011)

Current international guidelines therefore emphasize broader metabolic screening rather than narrow dependence on a single surrogate marker of insulin resistance (Teede et al., 2018, 2023). In practical terms, this means that clinicians should integrate anthropometric assessment, glucose metabolism testing, lipid profile evaluation, and clinical risk factors into a comprehensive metabolic appraisal.

Among surrogate measures, the homeostasis model assessment of insulin resistance (HOMA-IR) remains one of the most commonly used approaches in clinical and research settings (Matsuda & DeFronzo, 1999; Matthews et al., 1985). Additional metabolic indices, such as the triglyceride-glucose product, have also been proposed as practical surrogate markers of insulin resistance, although they are not specific to PCOS (Simental-Mendía et al., 2008). From a healthcare perspective, this issue is relevant because under-assessment of insulin resistance may lead to delayed identification of women at increased long-term metabolic risk. Conversely, excessive reliance on imperfect surrogate markers without adequate clinical interpretation may contribute to patient misclassification and inconsistent management strategies (Ehrmann, 2005). Therefore, assessment of insulin resistance in women with PCOS should be individualized and interpreted within a broader metabolic and clinical context.

3.5. Impact on quality of life, emotional wellbeing, and everyday functioning

One of the most important themes emerging from the reviewed literature is that PCOS substantially impairs quality of life. This reduction is multidimensional and includes physical, emotional, social, and sexual aspects of wellbeing (Bazarganipour et al., 2014). Although not all quality-of-life impairment can be directly attributed to insulin resistance, metabolic dysfunction appears to intensify many of the factors that lower subjective wellbeing.

Women with PCOS frequently experience distress related to body image, weight gain, menstrual irregularity, infertility, hirsutism, and acne. These symptoms often interact rather than occur in isolation, producing cumulative psychosocial burden (Bazarganipour et al., 2014; Cooney et al., 2017). When insulin resistance contributes to progressive weight gain, metabolic instability, or perceived difficulty losing weight, frustration and feelings of helplessness may become more pronounced (Harrison et al., 2011).

The association between PCOS and mood disorders is also well documented. There is an increased risk of depressive disorders in women with PCOS and high prevalence of moderate or severe depressive and anxiety symptoms (Cooney et al., 2017; Hollinrake et al., 2007). These findings indicate that psychological assessment should not be treated as optional in women with significant PCOS-related symptom burden.

Evidence also suggests that metabolic burden can influence everyday functioning. Chronic concern about fertility, body appearance, long-term diabetes risk, and unsuccessful attempts at symptom control may impair self-esteem, interpersonal relationships, social participation, and sexual wellbeing. Health-related quality of life in PCOS is shaped by several predictive factors, underlining the multidimensional nature of patient experience (Bazarganipour et al., 2014).

From an interdisciplinary perspective, this domain is particularly relevant. If PCOS is managed only through a narrow reproductive lens, substantial components of patient suffering may remain unrecognized. The literature strongly supports a model of care in which endocrine, metabolic, nutritional, and psychological needs are addressed together.

3.6. Therapeutic implications and multidisciplinary management

Lifestyle intervention is consistently recognized as the foundation of treatment for women with PCOS and insulin resistance. Diet, physical activity, and behavioral strategies may improve insulin sensitivity, body composition, menstrual cyclicity, and broader metabolic health (Harrison et al., 2011; Moran et al., 2011). Lifestyle treatment is especially important in women with overweight or obesity, but it may also benefit lean women by improving metabolic health and cardiovascular risk profile.

Evidence regarding insulin-sensitizing drugs beyond metformin remains heterogeneous, although systematic reviews suggest that selected agents may improve ovulatory and reproductive outcomes in some women with PCOS (Morley et al., 2017).

Exercise appears to have independent benefits beyond weight reduction alone. There is important value of exercise therapy in PCOS, particularly in relation to metabolic and reproductive outcomes (Harrison et al., 2011). This is clinically relevant because it supports framing physical activity not simply as a weight-loss tool, but as a therapeutic intervention in its own right.

Among pharmacologic options, metformin remains the most established insulin-sensitizing drug used in PCOS. Systematic reviews have shown that metformin may improve insulin sensitivity, menstrual regularity, and some reproductive and metabolic outcomes, although the magnitude of effect differs across patient groups and treatment aims (Johnson, 2014; Lord et al., 2003; Morley et al., 2017). It is particularly relevant in women with impaired glucose tolerance, elevated metabolic risk, or insufficient response to lifestyle intervention alone.

Other therapeutic strategies have also attracted attention. Inositol compounds, especially myo-inositol, have been studied for their potential to improve ovarian function and oocyte quality, and some evidence suggests possible benefit in selected women with PCOS (Garg & Tal, 2016; Unfer et al., 2011). Newer weight-management approaches such as liraglutide have shown promising effects on eating behavior and obesity-related outcomes in selected populations (Jensterle et al., 2015). These approaches are of interest but should be interpreted within the limits of available evidence.

Importantly, the literature and current guidelines emphasize that management of PCOS should be individualized and multidisciplinary (Teede et al., 2018, 2023). Effective care may require cooperation between gynecologists, endocrinologists, primary care physicians, dietitians, psychologists, and fertility specialists. Such a model is particularly important when metabolic risk coexists with infertility, obesity, or significant emotional distress. The main therapeutic approaches targeting insulin resistance and broader clinical consequences of PCOS are summarized in Table 2.

Table 2. Selected therapeutic approaches targeting insulin resistance and clinical manifestations of PCOS

Intervention	Main therapeutic target	Potential clinical benefits
Lifestyle modification	Insulin resistance and obesity	Improved metabolic profile, weight reduction, improved ovulatory function
Regular physical activity	Metabolic dysfunction	Improved insulin sensitivity and cardiovascular health
Metformin therapy	Hyperinsulinemia and dysglycemia	Improved insulin sensitivity, menstrual regularity, and metabolic outcomes
Inositol supplementation	Ovarian dysfunction and insulin signaling	Potential improvement in oocyte quality and reproductive outcomes
Weight-management therapies	Obesity-related metabolic burden	Reduction in adiposity and improvement in metabolic parameters
Psychological support and counseling	Emotional distress and reduced quality of life	Improved emotional wellbeing, treatment adherence, and patient support
Multidisciplinary care	Complex metabolic and psychosocial burden	More comprehensive and individualized long-term management

Overview of selected therapeutic strategies used in women with PCOS to address insulin resistance, metabolic dysfunction, reproductive symptoms, and quality-of-life impairment.

Abbreviations: PCOS — polycystic ovary syndrome.

4. Discussion

The literature reviewed in this paper supports the concept that insulin resistance is one of the most clinically meaningful components of PCOS. It links metabolic dysfunction with endocrine abnormalities and provides a mechanistic explanation for many of the syndrome's reproductive, metabolic, and psychosocial consequences (Barber & Franks, 2021). Although the degree of insulin resistance varies between phenotypes, the available evidence suggests that it should be regarded as a major determinant of disease burden rather than a secondary or incidental finding (Moghetti & Tosi, 2021).

The ongoing question of whether insulin resistance is a primary driver or a consequence of other metabolic abnormalities in PCOS continues to be debated, although current evidence suggests a bidirectional and self-reinforcing relationship (Moghetti & Tosi, 2021). PCOS should be understood not only as an endocrine and reproductive disorder, but also as a chronic condition associated with substantial public health and healthcare-system burden. The syndrome affects long-term healthcare utilization, work productivity, emotional wellbeing, and social functioning, while frequently requiring prolonged multidisciplinary management. In everyday practice, patients are often managed primarily because of infertility, irregular menstruation, acne, or hirsutism. However, the reviewed evidence indicates that metabolic risk and quality-of-life impairment deserve equally serious attention. If clinical care focuses only on visible reproductive symptoms, opportunities for prevention of long-term cardiometabolic complications may be missed (Goodarzi et al., 2011; Wild et al., 2010).

The reviewed literature also highlights the close relationship between insulin resistance and obesity, while at the same time showing that PCOS-related insulin resistance cannot be reduced to obesity alone. This distinction is clinically important. It prevents underestimation of risk in lean women and reminds clinicians that BMI by itself is an incomplete measure of metabolic health. It also underscores the need to address central adiposity, lifestyle factors, and chronic inflammation when designing therapeutic strategies (Barber & Franks, 2021).

Another major finding is the substantial effect of PCOS on mental health and daily functioning. Reduced quality of life, increased depressive and anxiety symptoms, and persistent concerns related to body image and fertility all appear repeatedly in the literature. These domains are not peripheral to the disease; they are part of its lived clinical reality. From this perspective, the relevance of PCOS extends beyond endocrinology and gynecology and enters the broader field of social functioning, healthcare access, and long-term patient support (Jensterle et al., 2015).

From a broader healthcare perspective, PCOS illustrates how a chronic metabolic condition can affect psychological wellbeing, lifestyle behaviors, social participation, healthcare access, and long-term treatment adherence. In this sense, insulin resistance is not only a metabolic disturbance but also a factor influencing

patient experience, adherence, and long-term engagement with healthcare systems (Azziz et al., 2016; Bazarganipour et al., 2014). The relationship between insulin resistance and the major reproductive and metabolic manifestations of PCOS is presented in Figure 1.

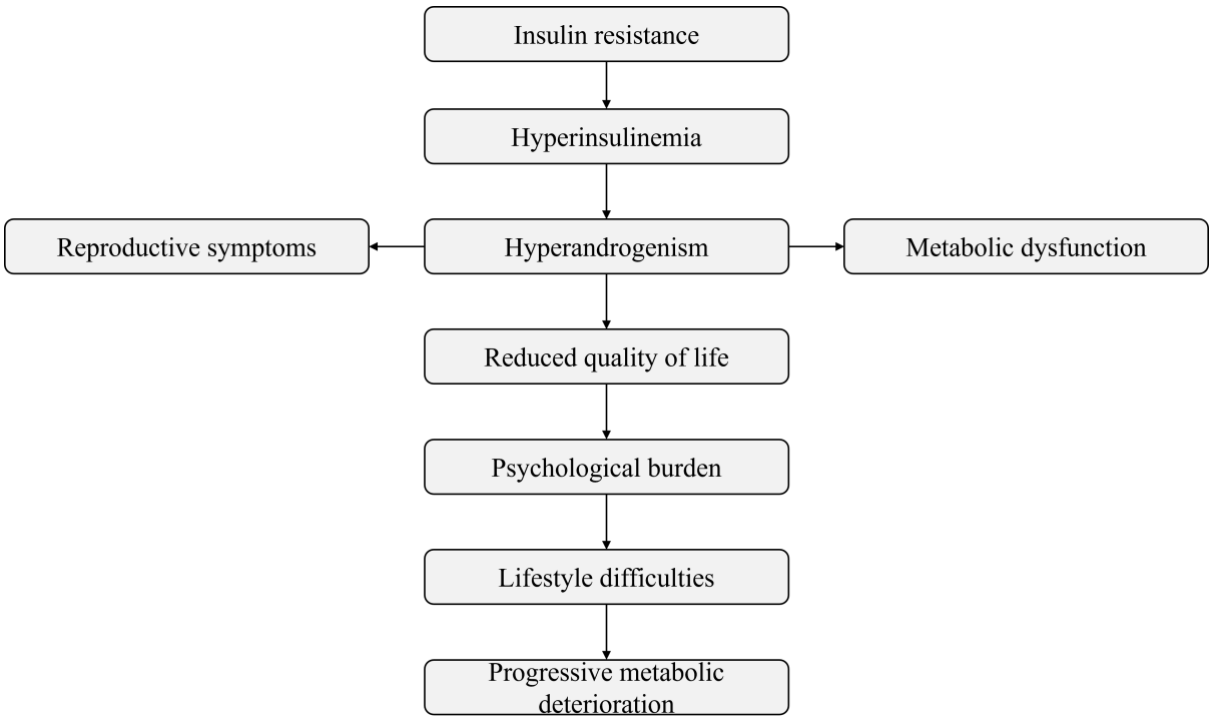

Fig. 1. *Simplified overview of the interactions between insulin resistance, endocrine abnormalities, metabolic dysfunction, and quality-of-life impairment in women with polycystic ovary syndrome. Note: Figure created by the author.*

The therapeutic implications of these findings are clear. Women with PCOS benefit most from care models that combine early metabolic screening, individualized treatment, sustained lifestyle support, and attention to psychological wellbeing. Guideline-based management increasingly supports this integrated approach, but implementation in routine care remains uneven. Fragmentation between specialties may delay diagnosis, reduce continuity of care, increase long-term healthcare burden, and leave important psychosocial aspects of the syndrome insufficiently addressed.

The increasing prevalence of obesity, metabolic syndrome, and insulin resistance among women of reproductive age may further increase the long-term healthcare burden associated with PCOS. Delayed diagnosis, fragmented care pathways, and insufficient integration of psychological and metabolic support may contribute to higher healthcare utilization and reduced effectiveness of long-term management strategies. Therefore, improving interdisciplinary coordination and preventive care may benefit both individual outcomes and broader public health systems.

This review also has limitations. As a narrative review, it does not provide a formal systematic search strategy or quantitative meta-analytic synthesis. The included literature is heterogeneous in design, diagnostic criteria, study populations, and methods used to assess insulin resistance, which limits direct comparison between studies. Some controversies remain regarding the best surrogate markers of insulin resistance and the relative contribution of obesity versus intrinsic cellular defects. Nevertheless, across these differences, the general picture is consistent: insulin resistance occupies a central role in the pathophysiology and clinical consequences of PCOS.

Future research should focus on more precise phenotyping of women with PCOS, better clinically applicable methods for metabolic risk stratification, and interventions that combine endocrine, metabolic, and psychological outcomes. More work is also needed on patient-centered care models and on how integrated management affects long-term quality of life and chronic disease prevention.

5. Conclusions

Insulin resistance is a key element of the pathophysiology of polycystic ovary syndrome and has wide-ranging consequences for reproductive, metabolic, and psychological health. Its impact extends from hyperandrogenism, ovulatory dysfunction, and subfertility to dysglycemia, metabolic syndrome, and increased long-term cardiometabolic risk. At the same time, women with PCOS frequently experience reduced quality of life, emotional distress, and social burden, which may be intensified by metabolic dysfunction and chronic symptom persistence.

These findings support the view that PCOS should be understood as a multidimensional disorder requiring more than symptom-based treatment. Early identification of metabolic risk, individualized therapeutic planning, and interdisciplinary care are essential for improving both objective clinical outcomes and subjective wellbeing. A broader and more integrated approach to insulin resistance in PCOS may therefore contribute to better long-term management and a more patient-centered standard of care. Improved interdisciplinary management and earlier recognition of metabolic risk may contribute to better individual outcomes and more effective long-term healthcare strategies for women with PCOS. Given the rising prevalence of metabolic disorders among women of reproductive age, strengthening long-term interdisciplinary care may also have meaningful public health implications.

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