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GLP-1 RECEPTOR AGONISTS VERSUS METFORMIN IN POLYCYSTIC OVARY SYNDROME: METABOLIC BENEFITS, REPRODUCTIVE OUTCOMES AND THE EVIDENCE GAP IN LEAN PCOS

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ABSTRACT

Polycystic ovary syndrome (PCOS) is one of the most frequently diagnosed disorders that affects many women of reproductive age and is linked to both metabolic and reproductive problems. Metformin has been used for many years as a standard treatment for insulin resistance in PCOS, while glucagon-like peptide-1 (GLP-1) receptor agonists have recently gained interest because of their stronger effects on body weight and metabolism. The aim of this review was to compare the effects of GLP-1 receptor agonists and metformin in women with PCOS, with special attention to the lack of data in lean PCOS.

Most studies show that GLP-1 receptor agonists lead to greater weight loss and larger reductions in body mass index, waist circumference, and insulin resistance compared with metformin, especially in women with overweight or obesity. However, when it comes to reproductive outcomes such as ovulation, menstrual regularity, or androgen levels, neither treatment clearly shows better results than the other. An important limitation of current research is that most studies include mainly overweight patients, while data on lean PCOS are still limited.

Overall, GLP-1 receptor agonists seem to offer stronger metabolic benefits, while metformin remains a practical and commonly used first-line treatment, particularly in lean patients. More studies focusing on different PCOS phenotypes, especially lean PCOS, are needed to better guide treatment decisions.

KEYWORDS

PCOS, Metformin, BMI, GLP-1, Female Fertility, Physical Activity, Reproductive Health, Lifestyle Factors, Exercise, Anti-obesity pharmacotherapy, Lean PCOS, Adolescent Obesity

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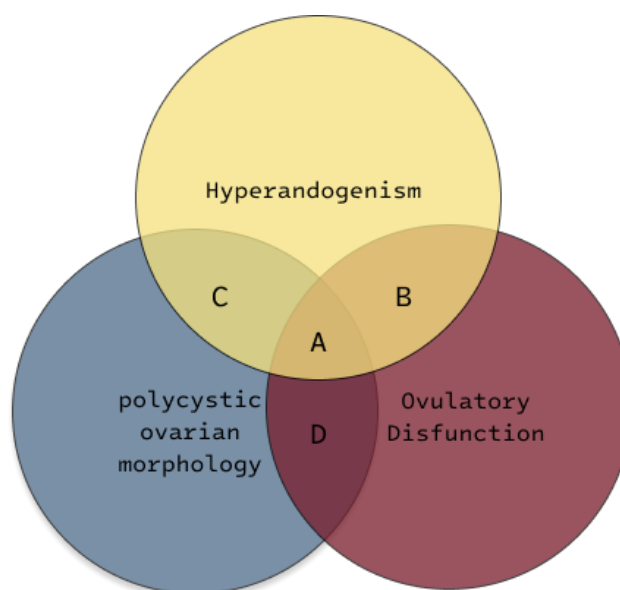
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1. Introduction

Polycystic Ovary Syndrome (PCOS) is one of the most common endocrine disorders affecting women of reproductive age. The prevalence is estimated at 5-18% in adults and 3-11% in adolescents [1]. It is a heterogeneous condition with several clinical phenotypes, shaped by both hormonal and metabolic disturbances.

PCOS is characterized by the presence of hyperandrogenism (HA), ovulatory dysfunction (OD), and polycystic ovarian morphology (PCOM). According to the Rotterdam criteria, the diagnosis is established when at least two of these features are present, allowing classification into four phenotypes (A-D) (graph 1) depending on their combination [14]. These phenotypes differ not only in reproductive manifestations but also in their associated metabolic risk.



Phenotypes associated with hyperandrogenism, particularly A and B, are more often linked to insulin resistance, obesity, an increased risk of metabolic syndrome, type 2 diabetes mellitus and cardiovascular disease. By comparison, phenotype D is generally considered to have a more favorable metabolic profile, while phenotype C appears to represent an intermediate form. Importantly, body mass index (BMI) acts as an independent modifier across all phenotypes, and metabolic abnormalities may also occur in individuals with normal body weight [13].

Management of PCOS includes both non-pharmacological and pharmacological approaches. Lifestyle interventions - including dietary modification, increased physical activity, and weight reduction - are the first-line strategy, particularly in individuals with overweight or obesity. Pharmacological treatment is tailored to the clinical presentation and may include oral contraceptives, antiandrogens, ovulation induction agents, and insulin-sensitizing therapies [16].

This review focuses on insulin-sensitizing treatment strategies in PCOS, with particular emphasis on comparing the established role of metformin with newer incretin-based therapies, such as glucagon-like peptide-1 (GLP-1) receptor agonists.

Although PCOS is commonly associated with overweight and obesity, a proportion of patients present with normal body mass index while still fulfilling diagnostic criteria. This subgroup, often referred to as lean PCOS, may involve distinct underlying pathophysiological mechanisms. Because most therapeutic evidence has been derived from studies conducted in overweight populations, the applicability of these findings to lean individuals remains uncertain. For this reason, the present review also examines the role of metformin and GLP-1 receptor agonists in lean PCOS, with particular attention to the existing gap in evidence for this subgroup [12].

2. Insulin resistance management strategy - metformin

Metformin is a synthetic biguanide, commonly used in the treatment of type 2 diabetes mellitus. It is the most widely studied insulin-sensitizing agent in polycystic ovary syndrome (PCOS). It is mainly prescribed in patients presenting with insulin resistance, impaired glucose tolerance, or other metabolic abnormalities. Available clinical data indicate that metformin improves insulin sensitivity and lowers circulating androgen levels. Metformin may also contribute to better menstrual regularity, particularly in women with obesity or more pronounced metabolic disturbances [5,16].

The effects of metformin are largely related to its ability to reduce hepatic gluconeogenesis and enhance peripheral glucose uptake. At the molecular level, these actions are mediated in part through activation of AMP-activated protein kinase (AMPK). As circulating insulin levels decrease, ovarian androgen production may also decline, which in turn can support improved follicular development and ovulatory function [5].

Overall, the metabolic effects of metformin are considered modest but clinically meaningful. Reported weight reduction usually ranges from 1 to 3 kg, accompanied by decreases in body mass index of approximately 0.5-1.5 kg/m² over 12-24 weeks of therapy. Improvements in insulin resistance, most often

assessed using the HOMA-IR index, have been observed in the range of about 10-25%, although the magnitude of change varies depending on baseline metabolic status [5].

In addition to metabolic effects, metformin demonstrates benefits in reproductive outcomes. Ovulation rates of approximately 30-50% have been reported with monotherapy, with improved outcomes when combined with ovulation induction agents. Menstrual regularity improves in approximately 40-70% of patients, and reductions in circulating androgen levels of approximately 10-25% have been observed, although clinical manifestations such as hirsutism may respond more slowly [5,16].

Despite these benefits, the clinical response to metformin is not uniform across all patient groups. Its effects tend to be more noticeable in individuals with clear metabolic disturbances, particularly those with overweight or obesity. In contrast, the benefits may be less evident in lean PCOS, where metabolic abnormalities are often less pronounced. This variability underscores the importance of individualized treatment strategies and highlights the need to consider alternative or adjunctive therapies when the response to metformin is limited [4].

3. Limitations of metformin in PCOS

Despite its long-standing role as an insulin-sensitizing agent in polycystic ovary syndrome (PCOS), metformin is associated with several limitations that have led researchers to explore alternative pharmacological options [5,16].

One of the main concerns is its relatively modest effect on body weight and central fat accumulation. Although metformin can lead to mild weight loss, its overall impact on body mass index (BMI) and visceral adiposity tends to be limited. This is particularly relevant in patients with overweight or obesity, in whom excess adipose tissue plays a central role in metabolic dysfunction [5,18].

Another important consideration is the variability in therapeutic response. Although metformin improves insulin sensitivity, the magnitude of this effect differs across individuals and may depend on phenotype, baseline metabolic status, and body composition, indicating that not all patients achieve clinically meaningful benefit [5,16].

Metformin also demonstrates inconsistent effects on reproductive and hormonal outcomes. Improvements in ovulation and menstrual regularity are reported, but not uniformly across all populations, and reductions in androgen levels may be modest in certain clinical contexts [4,5].

Tolerability represents another practical limitation. Gastrointestinal side effects, such as nausea, diarrhea, and abdominal discomfort, occur in approximately 10-25% of patients. In some cases, these symptoms lead to treatment discontinuation, particularly during the early phase of therapy when the dose is being introduced [5].

It should also be noted that the available evidence base has its own limitations. Many studies differ substantially in terms of design, patient characteristics, and outcome definitions, and several trials include relatively small sample sizes. As a result, drawing firm conclusions about long-term efficacy and generalizability remains challenging [4,5].

4. Introduction of GLP-1 receptor agonists

Glucagon-like peptide-1 (GLP-1) receptor agonists represent a class of pharmacological agents that were originally developed for the treatment of type 2 diabetes mellitus. Their effects on glucose homeostasis involve glucose-dependent insulin secretion, suppression of glucagon release, delayed gastric emptying, and regulation of appetite. These processes are largely mediated through central hypothalamic pathways and enhanced satiety signaling [8,10].

In recent years, GLP-1 receptor agonists have attracted growing interest as potential therapeutic agents in polycystic ovary syndrome (PCOS), particularly among patients with obesity and insulin resistance. In addition to glycemic control, these agents influence body weight, appetite, and overall metabolic function, thereby targeting several important components of PCOS pathophysiology [8,10].

Studies have reported noticeable reductions in body weight, body mass index, and waist circumference, usually observed within 12-24 weeks of treatment. Reported weight loss typically ranges from approximately 3-7 kg, with reductions in waist circumference of about 3-6 cm [3,9]. These effects appear to be mainly related to reduced caloric intake and improved insulin sensitivity. As insulin levels decrease, hyperinsulinemia-driven ovarian androgen production may also be attenuated, linking metabolic improvement with potential endocrine effects [8,10].

Recent evidence points to possible benefits in endocrine and reproductive parameters; however, the results remain inconsistent across studies. Ongoing research, including investigations of incretin-based multi-agonist therapies, continues to examine the broader therapeutic role of this class of agents in PCOS [10,11].

5. Limitations of GLP-1 in PCOS

Despite their promising metabolic effects, several limitations of GLP-1 receptor agonists need to be taken into account when considering their use in the management of PCOS.

Gastrointestinal adverse effects represent the most frequently reported limitation. Nausea is observed in approximately 20-40% of patients, while vomiting and diarrhea occur in around 5-20%.

Although these symptoms are usually transient, they may lead to treatment discontinuation in approximately 5-15% of cases [3,9].

Another limitation relates to the current focus of available evidence on metabolic outcomes. While improvements in body weight, body mass index, and insulin resistance are reported consistently, data concerning reproductive and hormonal outcomes remain limited and heterogeneous. As a result, no definitive conclusions can be drawn regarding ovulation, menstrual regularity, or androgen levels [3,10].

The generalizability of the existing evidence is also restricted. Most studies have been carried out in overweight or obese populations, with only limited representation of lean PCOS and few phenotype-specific analyses. This reduces the applicability of these findings to this subgroup [8,10].

Long-term safety data also remain insufficient. Most available trials are limited to 12-24 weeks in duration, and the long-term metabolic, reproductive, and cardiovascular effects of GLP-1 receptor agonists in PCOS have not yet been fully established [10,11].

6. Why GLP-1 Receptor Agonists Might Be Better Than Metformin

GLP-1 receptor agonists and metformin both target core metabolic disturbances in polycystic ovary syndrome (PCOS), particularly insulin resistance. However, current evidence suggests that GLP-1 receptor agonists provide greater improvements in outcomes related to adiposity and metabolic risk.

The most consistent advantage of GLP-1 receptor agonists is their effect on body weight and fat distribution. Across randomized trials and meta-analyses, these agents induce greater weight loss (approximately 3-7 kg) and larger reductions in body mass index (1.5-3.0 kg/m²) compared with metformin (approximately 1-3 kg and 0.5-1.5 kg/m², respectively) [3,9]. Similarly, reductions in waist circumference (~3-6 cm) are more pronounced, indicating a stronger effect on visceral adiposity.

This difference is clinically relevant given the central role of visceral fat in PCOS pathophysiology, where it contributes to insulin resistance, hyperinsulinemia, and ovarian androgen excess. By more effectively reducing central adiposity, GLP-1 receptor agonists may exert a greater upstream impact on metabolic dysfunction [8,10].

Both therapies improve insulin sensitivity, but the magnitude of effect tends to favor GLP-1-based treatments, with reductions in HOMA-IR of approximately 15-30% compared with 10-25% for metformin [3,4,9]. Although moderate, this difference is most relevant in patients with significant metabolic impairment.

Another distinction lies in response consistency. Metformin demonstrates variable efficacy across PCOS phenotypes, with reduced effectiveness in some groups, including lean

individuals. In contrast, GLP-1 receptor agonists show more predictable benefits in patients with excess adiposity, where weight reduction itself is a primary therapeutic target [8,10].

These benefits must be balanced against tolerability. Gastrointestinal adverse effects, particularly nausea, are more frequent with GLP-1 receptor agonists (20-40%) than with metformin (10-20%), although they are generally manageable with dose titration.

Overall, GLP-1 receptor agonists demonstrate a greater magnitude of metabolic effect, particularly in overweight and obese PCOS, supporting their role when weight reduction and insulin resistance are primary therapeutic targets [4].

7. Lean PCOS and Phenotype-Specific Therapeutic Considerations

Lean PCOS describes patients who meet the diagnostic criteria for PCOS despite having a normal body mass index (BMI < 25 kg/m²). Although these individuals may present with insulin resistance, hyperandrogenism, and ovulatory dysfunction, their metabolic profile is generally less influenced by excess adiposity compared with obese phenotypes [13,17].

This distinction carries important therapeutic implications. The clinical effects of GLP-1 receptor agonists are largely related to weight reduction and improvements in insulin resistance associated with adiposity. As a result, their effectiveness in lean PCOS remains uncertain, since most available studies have been conducted in overweight or obese populations [3,9]. This situation creates a gap between the existing evidence and its practical applicability to lean phenotypes.

Metformin, by contrast, improves insulin sensitivity independently of weight loss and has been associated with ovulation rates of approximately 30-50% and improved menstrual regularity in about 40-70% of patients [5,16]. These features support its use in lean PCOS, particularly in cases where reproductive dysfunction is the predominant concern.

For this reason, a phenotype-oriented approach appears justified. GLP-1 receptor agonists seem to be most beneficial in patients with overweight or obesity and marked metabolic disturbances, whereas metformin remains a reasonable first-line option in lean individuals. Combination therapy may provide additional metabolic benefits, especially in terms of weight reduction; however, evidence supporting its use in lean PCOS is still limited [9,10].

In summary, although GLP-1 receptor agonists represent a promising therapeutic option, their role in lean PCOS is still not clearly defined. Further studies focused on this subgroup are needed to address the existing gap and support phenotype-specific management.

8. Conclusions

Current evidence from randomized controlled trials and meta-analyses indicates that GLP-1 receptor agonists provide greater improvements in body weight, body mass index, waist circumference, and insulin resistance compared with metformin in women with polycystic

ovary syndrome (PCOS) [3,4,9]. These effects are consistent across studies primarily assessing metabolic outcomes and are particularly relevant in patients with overweight or obesity, where excess adiposity is a central driver of disease pathophysiology [8,10].

In contrast, available data do not demonstrate consistent or clinically meaningful superiority of GLP-1 receptor agonists over metformin in reproductive outcomes or androgen-related parameters. Ovulation rates, menstrual regularity, and hormonal markers show heterogeneous results across studies, with no clear advantage of one therapy over the other [4,10]. Similarly, both treatments appear to exert comparable effects on glycemic control, reinforcing the view that their primary distinction lies in metabolic rather than endocrine outcomes.

Metformin remains a well-established first-line therapeutic option in PCOS, supported by extensive clinical experience, a favorable safety profile, and demonstrated benefits in both metabolic and reproductive domains [5]. In contrast, GLP-1 receptor agonists are more frequently associated with gastrointestinal adverse effects, including nausea and vomiting, which may contribute to higher treatment discontinuation rates [5,10]. Although these effects are often transient, they remain a relevant limitation in clinical practice.

Importantly, the current evidence base is derived predominantly from studies involving overweight and obese populations. Data specific to lean PCOS are scarce, and most trials do not include phenotype-stratified analyses based on criteria such as the Rotterdam criteria for PCOS diagnosis. Given the distinct pathophysiology of lean PCOS, where metabolic dysfunction is less strongly linked to adiposity, the generalizability of existing findings to this subgroup remains uncertain [13,17].

Overall, GLP-1 receptor agonists and metformin differ primarily in the magnitude of their metabolic effects, with GLP-1 receptor agonists demonstrating superior efficacy in weight reduction and insulin resistance, particularly in non-lean PCOS. However, current evidence does not support a clear advantage in reproductive or endocrine outcomes, where metformin remains a rational and often preferred option.

Given these complementary mechanisms of action, combination therapy with a GLP-1 receptor agonist and metformin represents a promising therapeutic strategy. Such an approach may allow simultaneous targeting of metabolic dysfunction through weight reduction and improved insulin sensitivity, while preserving the beneficial effects of metformin on ovulation and menstrual regulation. Preliminary evidence suggests that combination therapy may provide additive benefits, particularly in terms of weight loss and metabolic improvement, although robust data on reproductive outcomes remain limited [4,10].

Future research should focus on well-designed randomized controlled trials stratified by PCOS phenotype to better define the role of both monotherapy and combination therapy across diverse patient populations, with particular emphasis on addressing the current evidence gap in lean PCOS. This discrepancy in lean and non-lean PCOS treatment data highlights a critical limitation in current PCOS management, where treatment strategies derived from predominantly obese populations may not fully address the pathophysiological mechanisms underlying lean PCOS.

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