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Dolna 17, Warsaw,
Poland 00-773
+48 226 0 227 03
editorial_office@rsglobal.pl

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SEMAGLUTIDE IN THE MANAGEMENT OF POLYCYSTIC OVARY SYNDROME: CURRENT EVIDENCE AND REPRODUCTIVE BENEFITS

Diana Wisz *(Corresponding Author, Email: dianawisz@vp.pl)*
Clinical Provincial Hospital No. 2 in Rzeszów, Rzeszów, Poland
ORCID ID: 0009-0007-7929-1937

Filip Maciej Huzarski
Clinical Provincial Hospital No. 2 in Rzeszów, Rzeszów, Poland
ORCID ID: 0009-0002-3773-5388

Katarzyna Adrianna Tryniecka
Clinical Provincial Hospital No. 2 in Rzeszów, Rzeszów, Poland
ORCID ID: 0009-0001-7425-5861

Katarzyna Maria Turek
Clinical Provincial Hospital No. 2 in Rzeszów, Rzeszów, Poland
ORCID ID: 0009-0003-9615-6906

Weronika Worosz
Clinical Provincial Hospital No. 2 in Rzeszów, Rzeszów, Poland
ORCID ID: 0009-0008-5284-1633

Alicja Zań
Clinical Provincial Hospital No. 2 in Rzeszów, Rzeszów, Poland
ORCID ID: 0009-0008-9686-1759

ABSTRACT

Introduction: Polycystic ovarian syndrome (PCOS) is a common endocrine and metabolic disorder affecting 6–20% of women of reproductive age [1,2]. Characterized by hyperandrogenism, oligo- or anovulation, and polycystic ovarian morphology. PCOS presents heterogeneous clinical manifestations, including obesity, metabolic dysfunction, and reproductive irregularities. New therapies, particularly glucagon-like peptide-1 receptor agonists (GLP-1 RAs) such as semaglutide, show promise in addressing these challenges.
Methodology: This review analyzed articles published from 2019 to 2025 in the PubMed database using the keywords: semaglutide, PCOS, pregnancy and treatment. Studies were selected to evaluate the effects of semaglutide on body weight, metabolic parameters, reproductive outcomes, and safety profiles in women with PCOS.
Results: Semaglutide was associated with significant weight reduction, improvements in BMI, insulin resistance, fasting glucose, and lipid profile, as well as reduced androgen levels and improved menstrual regularity [11–13]. Low-dose semaglutide was effective even in patients unresponsive to lifestyle interventions, although long-term weight maintenance after discontinuation remains challenging [12]. While weight loss may enhance fertility, semaglutide is contraindicated during pregnancy due to limited safety data and potential risks [5,9,17,18].
Conclusion: Semaglutide represents a promising therapy for obese women with PCOS, offering metabolic and reproductive benefits. Careful consideration of reproductive planning, dosing strategies, and long-term safety is essential. Further research is needed to optimize treatment protocols and assess efficacy and safety outcomes.

KEYWORDS

Polycystic Ovarian Syndrome, PCOS, Semaglutide, GLP-1 Receptor Agonist, Obesity, Fertility

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1. Introduction and purpose

Polycystic ovarian syndrome (PCOS) is a general endocrine and metabolic disorder occurring in about 6–20% of females in reproductive age. Most symptoms of PCOS appear early during puberty. Because PCOS is associated with a wide variety of symptoms, it is considered a heterogeneous disease. The most accepted diagnostic criteria is Rotterdam criteria which involves two of the latter three features: hyperandrogenism, oligo- or anovulation, and polycystic ovaries [1].

Table 1. Rotterdam Criteria of PCOS

Hyperandrogenism	Clinical and/or biochemical signs of hyperandrogenism
Ovulation	Oligo/anovulation
Ovarian morphology	Polycystic ovarian morphology

Initially, PCOS was regarded mainly as a disorder of the ovaries. Today, it is understood to result from dysfunction of the hypothalamic–pituitary–ovarian axis, driven by abnormal secretion of FSH and LH from the hypothalamus and pituitary. The condition is associated with increased LH pulse frequency and amplitude, along with ovarian abnormalities that trigger excessive androgen production [2].

In recent years, substantial progress has been made in understanding PCOS. Treatments such as oral contraceptives, metformin, and hormone therapy have been developed to counteract its effects. Nevertheless, long-term management appears to rely most effectively on lifestyle modifications aimed at preventing abnormal immune activation and reducing exposure to inflammatory factors [3]. The aim of this review is to comprehensively synthesize the current evidence regarding the therapeutic effects of semaglutide in PCOS. By critically evaluating data on its impact on body weight, pregnancy outcomes, and other clinical manifestations, it seeks to offer evidence-based insights that can guide clinical decision-making and inform future research directions in PCOS management.

2. Material and methods

The review was based on research of articles published from 2019 to 2025 on the PubMed database using the following keywords: semaglutide, pcos, pregnancy, treatment. The analysis draws on the latest clinical trial results and scientific articles to provide a detailed overview of the current situation and future directions in this crucial area of medical research and clinical practice.

3. Results

Oral contraceptives remain a first-line therapy for management of hyperandrogenism and irregular cycles and the role for metformin, while limited, may still add benefit for metabolic dysfunction and weight management. Many challenges remain in the treatment of PCOS. The high prevalence of obesity is a significant contributing factor to its morbidity. Early attention to weight gain in childhood and adolescence in those at risk for PCOS may be an important measure of prevention. The use of obesity drugs is limited by their high price and availability, but emerging evidence suggests their efficacy, especially glucagon-like peptide-1 (GLP-1) receptor agonists, in treating obesity in women with PCOS, compared with metformin [4].

Semaglutide is a glucagon-like peptide-1 (GLP-1) receptor agonist indicated as an adjunct to a low-calorie diet and increased physical activity for long-term weight management in adult patients with an initial body mass index (BMI) of 30 kg/m² or greater and 27 kg/m² or greater with at least one weight-related comorbidity (e.g., hypertension, type 2 diabetes, or dyslipidemia) [5]. Additionally, its potential utility in

various other indications is under investigation, including cardiovascular risk reduction in type 2 diabetes, treatment of nonalcoholic steatohepatitis (NASH), and Alzheimer's disease [6]. No major safety concerns have emerged to date, although definitive conclusions regarding pancreatic cancer, thyroid cancer, and complications of diabetes and reproductive disorders cannot be drawn at this stage [7].

3. 1 The expression of GLP-1 receptors in the hypothalamic–pituitary–gonadal system

GLP-1 receptor agonists (GLP-1 RAs) act on multiple brain regions, particularly the hypothalamus, where they stimulate proopiomelanocortin (POMC) neurons, increasing protein kinase A activity and calcium influx into L-type calcium channels, thereby suppressing appetite. In animal studies, GLP-1 RAs reduce food intake, food reward, and ingestive behavior and can also activate thermogenesis in brown adipose tissue via the hypothalamic AMPK kinase. In humans, weight loss is primarily due to decreased appetite rather than increased energy expenditure.

GLP-1 also influences reproductive hormones by stimulating GnRH release, increasing luteinizing hormone levels, and influencing ovarian steroidogenesis. In women with PCOS, liraglutide lowels androgen levels and increases SHBG levels [8]. Metabolically, GLP-1 improves insulin sensitivity through anti-inflammatory effects and weight loss. It also slows gastric emptying and reduces postprandial glucose excursions. In the pancreas, GLP-1 enhances insulin secretion via cAMP- and calcium-dependent pathways, promotes β-cell proliferation, and protects against apoptosis. These actions make GLP-1 RAs valuable in the treatment of type 2 diabetes, obesity, and the metabolic disorders of PCOS [8].

3.2 GLP-1 Analogs in the Context of Obesity

The 2023 international evidence-based guidelines for the evaluation and treatment of polycystic ovary syndrome (PCOS) recommend that anti-obesity medications, including liraglutide, semaglutide, glucagon-like peptide-1 receptor agonists (GLP-1 RAs), and orlistat, may be considered in addition to active lifestyle intervention for the treatment of overweight in adults with PCOS. The decision to initiate GLP-1 RA therapy should take into account potential side effects and the likely need for long-term use, given the high risk of weight regain after treatment discontinuation and the current lack of long-term safety data [9].

Semaglutide has demonstrated greater efficacy than liraglutide in head-to-head clinical comparisons. In a randomized trial involving adults with overweight or obesity without diabetes, once-weekly subcutaneous semaglutide, when combined with counseling on diet and physical activity, resulted in significantly greater weight loss over 68 weeks than once-daily liraglutide. At week 68, mean body weight reduction was –15.8% in the semaglutide group compared with –6.4% in the liraglutide group, with concomitant improvements in several cardiovascular risk factors [10].

In a prospective study by Carmina et al., the effects of low-dose semaglutide [0.5 mg/week] were evaluated in 27 obese women with classic phenotype A PCOS (chronic anovulation, hyperandrogenism, and polycystic ovarian morphology) who had failed to achieve meaningful weight loss following a lifestyle intervention program (diet, physical exercise, and psychological support). After 3 months of semaglutide treatment, mean weight loss was 7.6 kg, with a mean BMI reduction of 3.1 kg/m², and nearly 80% of participants achieved ≥5% weight loss. Non-responders (22%), defined as those losing <5% of their baseline body weight, tended to be more severely obese. Importantly, even non-responders showed improvements in fasting insulin, HOMA-IR, and fasting glucose. In women with impaired fasting glucose, 80% achieved normoglycemia after 3 months, and the remainder showed marked improvement.

In the second phase of the study, patients who responded to treatment experienced an additional 3 months of treatment, resulting in a total mean weight loss of 11.5 kg, with a reduction in BMI from 34.4 to 29.4 kg/m². Menstrual regularity improved in 80% of these patients, while hirsutism scores (Ferriman–Gallwey–Lorenzo) remained unchanged. Treatment was generally well tolerated, with mild gastrointestinal side effects, including nausea (33%) and occasional vomiting (7%), and no treatment discontinuations due to adverse events. The authors concluded that low-dose semaglutide is effective in reducing body weight and improving metabolic and reproductive parameters in obese PCOS patients unresponsive to lifestyle modification, with the greatest benefit in women with milder obesity. For those with severe obesity, higher doses may be required to achieve comparable effects. Notably, semaglutide improved insulin resistance and fasting glucose independently of weight loss, suggesting potential as an alternative to metformin for metabolic management in PCOS [11].

Long-term weight maintenance following semaglutide withdrawal remains a clinical challenge. In a 2-year observational study by Jensterle et al., 25 women with PCOS and obesity receiving stable metformin therapy [2000 mg/day] and lifestyle counseling underwent semaglutide treatment [1.0 mg/week] for 16 weeks, followed by discontinuation of semaglutide while continuing metformin. Initial therapy produced significant weight loss, along with improvements in lipid profile, fasting glucose, oral glucose tolerance test results, and reduced free testosterone. Two years after semaglutide cessation, participants regained approximately one-third of their weight loss—less than the two-thirds typically reported in the general population. However, cardiometabolic benefits largely reverted to pre-treatment levels, while reductions in free testosterone were sustained. Side effects were mostly mild and gastrointestinal in nature. The findings suggest that metformin may attenuate post-semaglutide weight regain in insulin-resistant PCOS patients, though larger randomized studies are needed to confirm these observations and guide long-term treatment strategies [12].

A meta-analysis encompassing 176 overweight women with PCOS further supports the metabolic benefits of GLP-1 RAs. Treatment was associated with significant reductions in BMI, waist circumference, total testosterone [by approximately 33%], and triglycerides. However, no significant improvement in HOMA-IR was observed, indicating that insulin sensitivity outcomes may be variable across patient populations [13].

The available evidence suggests that GLP-1 RAs, and semaglutide in particular, are an effective treatment option for weight loss and metabolic improvement in women with PCOS who have not responded to lifestyle changes alone. However, the optimal dosing strategy, duration of therapy, and long-term safety profile remain areas for further investigation, especially given the high propensity for weight regain after treatment discontinuation.

Table 2. Comparison of semaglutide in studies

Study	Semaglutide Dose	Population	Treatment Duration	Weight Change	BMI Change	Glycemia / Insulin Resistance
Head-to-head vs. liraglutide	Not specified in text	Adults with overweight/obesity, without diabetes	68 weeks	–15.8% vs –6.4% [liraglutide]	—	Improvement in cardiometabolic risk factors
Carmina et al. – Phase I	0.5 mg/week	27 women with PCOS, phenotype A, obesity, non-responders to lifestyle change	3 months	–7.6 kg	–3.1	Improved fasting insulin, HOMA-IR, fasting glucose; in IFG: 80% achieved normoglycemia
Carmina et al. – Phase II [responders]	0.5 mg/week	Subgroup from above [responders]	Additional 3 months [6 months total]	–11.5 kg [from baseline]	34.4 → 29.4	—
Jensterle et al.	1.0 mg/week for 16 weeks, then discontinued [metformin continued]	25 women with PCOS and obesity on metformin	16 weeks treatment + 2-year follow-up	Significant weight loss; after 2 years regained ~1/3 of lost weight	—	Improved lipids, fasting glucose, OGTT during treatment; partial reversal after discontinuation
Meta-analysis [semaglutide subset]	Various	Women with PCOS and overweight	—	Weight loss	BMI reduction	No significant change in HOMA-IR

3.3 Effects on fertility

While weight loss is known to improve certain fertility-related outcomes, evidence for its effect on live birth rates (LBR) remains mixed. However, newer anti-obesity drugs, particularly GLP-1 receptor agonists such as semaglutide [Wegovy], can induce far greater weight losso an average of 14.9–17.9% of body weight within 68 weeks which may contribute to improving fertility in patients with PCOS [14].

Obesity-related infertility can improve through weight loss, with benefits including higher spontaneous conception rates, better ovulation induction responses, and fewer pregnancy complications. Professional

guidelines (ACOG, British Fertility Society) recommend achieving a BMI <25 kg/m² before fertility treatment and deferring treatment if BMI ≥35 kg/m² [14, 15].

GLP-1 RAs, including semaglutide, not only facilitate significant weight loss but may also directly enhance fertility via anti-inflammatory and hormonal effects on the reproductive system. In women with obesity and PCOS, GLP-1 RA treatment has been associated with improved menstrual regularity, lower androgen levels, reversal of polycystic ovarian morphology, and higher conception rates—sometimes even when other fertility treatments have failed. [14,16]

3.4 Semaglutid during pregnacy

Spontaneous ovulation may resume during weight loss with semaglutide, creating a potential risk of fetal exposure. Accordingly, the FDA recommends discontinuing semaglutide immediately once pregnancy is confirmed. For patients planning pregnancy, WEGOVY should be stopped at least two months before conception due to the drug's long half-life [5]. Healthcare professionals should ensure effective contraception in women of childbearing potential receiving GLP-1 receptor agonists, as safety data during pregnancy are limited [9].

Current evidence regarding semaglutide exposure during pregnancy remains scarce. Data from 32 documented pregnancies do not indicate an increased risk of major congenital malformations, although statistical power is limited [17]. Compared with unexposed pregnancies, semaglutide use was associated with higher risks of preterm birth, large-for-gestational-age (LGA) infants, neonatal hypoglycemia, and neonatal jaundice. Notably, these associations were not observed when semaglutide-exposed pregnancies were compared with those exposed to insulin alone, likely reflecting that most semaglutide-exposed women were also receiving insulin, were older, and had a higher baseline BMI. The small number of exposed pregnancies represents a major limitation, precluding adequate adjustment for potential confounders. The dataset included 27 pregnancies exposed to Ozempic® (2019–2023) and 5 recent cases involving Wegovy® in women without diabetes (2023) [17]. With the increasing use of semaglutide for weight loss—particularly among women without diabetes—incidental early pregnancy exposure is expected to rise, especially in women with overweight or obesity, as weight loss may improve fertility. Maternal weight regain after treatment discontinuation may independently contribute to adverse obstetric outcomes [17].

A larger prospective multicentre study assessed first-trimester GLP1-RA exposure in 168 women, compared with pregnant women with diabetes or overweight/obesity. No specific pattern of birth defects was observed, and the risk of major congenital anomalies (2.6%) was similar to both reference groups and general population rates. Pregnancy losses were not increased, although elective terminations were more frequent, possibly reflecting unplanned pregnancies and concerns about fetal risks [18].

A case report further illustrates potential concerns. A 31-year-old woman with PCOS used semaglutide (0.5–1.0 mg/week) for six months prior to conception, discontinuing at gestational week 3 + 4. She lost 27 kg before pregnancy but subsequently gained 35 kg during gestation. Delivery at week 41 + 5 resulted in a healthy female infant weighing 5.23 kg (+38% above expected birth weight), with labor complicated by shoulder dystocia and transient neonatal hypoglycemia [18]. This first documented pregnancy with early semaglutide exposure highlights the potential role of post-discontinuation rebound weight gain in maternal obesity and fetal macrosomia, emphasizing the need to balance preconception weight loss with the risk of excessive gestational weight gain in women of reproductive age [19].

In summary, pregnancies exposed to both semaglutide and insulin showed similar risks of congenital malformations, preterm birth, LGA, neonatal hypoglycemia, and neonatal jaundice compared with pregnancies exposed to insulin alone. Nevertheless, substantial knowledge gaps remain. Given the paucity of both human and animal data, current recommendations against the use of semaglutide during pregnancy should not be revised. Anticipated increases in unintended early pregnancy exposure underscore the urgent need for larger, well-controlled studies to clarify safety and inform clinical guidance [17].

4. Conclusions

Polycystic ovarian syndrome (PCOS) is a complex endocrine and metabolic disorder with heterogeneous manifestations, affecting a significant proportion of women of reproductive age [1,2]. Current management strategies combine lifestyle modification, pharmacologic interventions such as oral contraceptives and metformin, and emerging therapies targeting obesity and metabolic dysfunction, including GLP-1 receptor agonists [3,4]. Semaglutide, a GLP-1 RA, has demonstrated significant efficacy in reducing

body weight, improving metabolic parameters, and partially restoring reproductive function in women with PCOS, especially those unresponsive to lifestyle interventions [11–13]. However, long-term weight maintenance after discontinuation remains challenging, and optimal dosing strategies and safety profiles require further investigation [12,13]. While semaglutide-induced weight loss may enhance fertility, its use during pregnancy is contraindicated due to limited safety data and potential risks to the fetus, underscoring the need for effective contraception in women of reproductive age [5,9,17,19]. Overall, semaglutide represents a promising adjunctive therapy in PCOS management, particularly for obese patients, but careful consideration of reproductive planning and long-term outcomes is essential.

Disclosure

Author's contribution:

Conceptualization: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Methodology: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Check: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Formal analysis: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Investigation: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Resources: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Data curation: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Writing rough preparation: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Writing-review and editing: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Supervision: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

Project administration: Diana Wisz, Filip Maciej Huzarski, Katarzyna Tryniecka, Katarzyna Maria Turek, Weronika Worosz, Alicja Zań

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