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Breif Commentries

Recent Advances in Pharmacologic Management of PCOS: Targeting Androgens, Metabolism and Ovulation

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ABSTRACT

Polycystic ovary syndrome (PCOS) is one of the most common endocrine metabolic disorders in women of reproductive age, characterized by hyperandrogenemia, chronic anovulation and insulin resistance. Traditionally, the treatment of PCOS is mainly aimed at symptomatic relief and most of them are over-the-counter medications and there is still no therapeutic drug specifically approved for PCOS1. However, in recent years, important advances have been made in drug treatment strategies targeting the three core features of PCOS. This editorial describes and summarizes established treatments as well as novel therapeutic agents.

Keywords: PCOS; Pharmacologic management; Androgens; Metabolism; Ovulation

1. Treatment for Hyperandrogenemia

Combination oral contraceptives (COCs) remain the treatment of choice. COCs are effective in reducing free testosterone by suppressing luteinizing hormone (LH) and increasing sex hormone binding globulin (SHBG) levels[1]. Low-dose COCs containing anti-androgenic progestins such as drospirenone have been shown to be efficacious in improving hirsutism, acne and menstrual disorders and have a positive effect on metabolic markers[2].

Spironolactone is widely used as an androgen receptor antagonist in patients with poor symptom control or who cannot tolerate COCs. Studies have shown that spironolactone (100 mg/day for 6-12 months of treatment) reduces hirsutism scores by more than 50% and has no negative effect on body weight, lipids or blood glucose levels[3].Other antiandrogenic drugs such as finasteride (5α-reductase inhibitor) and flutamide (androgen receptor blocker) have also been shown to be efficacious[2]. but the latter is restricted due to higher risk of hepatotoxicity. In recent years a topical androgen receptor inhibitor (clascoterone cream) has been approved for the treatment of acne, giving a new option for topical treatment of androgenic skin symptoms in patients with PCOS.

2. Treatments Targeting Metabolic Dysfunction

Insulin resistance is one of the central mechanisms in the pathogenesis of PCOS. Metformin improves insulin sensitivity and reduces hepatic glucose output through activation of the AMPK pathway, which also indirectly reduces ovarian androgen secretion and helps to restore a more regular ovulation cycle[2]. Recent studies have shown that metformin plays a positive role in modestly improves ovulation rates, menstrual regularity

and reduces serum androgen levels in women with PCOS[2]. Thiazolidinediones (e.g., pioglitazone), on the other hand, improve insulin sensitivity by activating PPAR-γ receptors-increase ovulation rates and improve metabolic markers[2].

Inositols (e.g., myo-inositol, D-chiral inositol) have received much attention in recent years as natural insulin signaling molecules. Systematic evaluations have noted that inositol improves ovulation rate, insulin sensitivity and menstrual cycle regularity[4], but the quality of the evidence is still somewhat controversial and needs to be selected in the context of patient individualization.

GLP-1 receptor agonists (e.g., liraglutide, simethicone) have shown superior weight management and metabolic improvement in PCOS by promoting satiety, suppressing appetite and improving insulin sensitivity[5]. Recent studies have shown that GLP-1 receptor agonists are not only superior to metformin in weight management[2,5], but also help to improve the menstrual cycle with lower androgen levels[2].

Sodium-glucose co-transporter protein 2 (SGLT2) inhibitors, such as dalgogliflozin, improve blood glucose levels and promote weight loss and preliminary studies suggest that it may also improve hormonal profiles and metabolic parameters in PCOS patient[1].

3. Treatment for Ovulation Disorders

Letrozole, an aromatase inhibitor, has replaced clomiphene as the first-line drug for ovulation induction in PCOS. By inhibiting estrogen synthesis, letrozole relieves negative feedback stimulation of FSH secretion and promotes follicular development. A recent meta-analysis showed that letrozole showed higher ovulation, pregnancy and live birth rate[6].

Weight loss by GLP-1 receptor agonists or SGLT2 inhibitors followed by ovulation induction has also emerged as an emerging integrative treatment strategy in overweight or obese PCOS patients.

In conclusion, important advances have been made in the pharmacologic management of PCOS in recent years. COCs, spironolactone, metformin and letrozole remain the cornerstone of hyperandrogenism management; GLP-1 receptor agonists along with SGLT2 inhibitors offer new options for patients with metabolic disorders. In the future, treatment will be more individualized and precise, integrating to improve the endocrine and metabolic status of PCOS patients to optimize reproductive and long-term health outcomes **(Table 1)**.

Table 1: Pharmacologic therapies in PCOS – classes, mechanisms, clinical targets and recent evidence.

Therapy Class	Mechanism of Action	Clinical Targets	Key Recent Findings
Combined Oral Contraceptives	Suppress gonadotropins (↓LH), ↑SHBG (less free T)	Hirsutism, acne; regulate menses	Remain first-line for hyperandrogenism; low-dose combos effectively reduce hirsutism and acne[2] pmc.ncbi.nlm.nih.govpmc.ncbi.nlm.nih.gov. Anti-androgenic progestins in newer COCs improve metabolic profile.
Anti-androgens (e.g. spironolactone, finasteride)	Block androgen receptor or synthesis (finasteride inhibits 5α-reductase)	Hirsutism, acne (usually adjunct to COC)	Spironolactone 50 –200 mg/day s ignificantly re duces hirsutism with mi nimal side ef fects[3] pmc.ncb i.nlm.nih.gov. Finaster ide and fluta mide al so effe ctive for hirsutism[2] pmc.nc bi.nlm.nih.g ov, tho ugh flu tamide use limited by hepatotoxicity.
Metformin (biguanide)	Improves insulin sensitivity; reduces hepatic gluconeogenesis	Insulin resistance; dysglycemia; assists ovulation	Still a sta ple in sulin sensitizer - improves menstrual regularity and ov ulation, modest weight loss[5] pmc.ncbi.nlm.nih.gov . Ofte n combined with other agents; GI tolerability can limit use.
Inositols (myo-inositol & D-chiro-inositol)	Insulin second messenger; improves insulin signaling and oocyte microenvironment	Insulin resistance; ovulatory dysfunction	Meta-analys es sho w improved insulin sensitivity and ovulation, comparable to metformin in some studies[4]rbej.biomedcentr al.com . However, latest evidence reviews deem clinical benefit inconclusive[7]pmc.ncbi.nlm.nih.gov.
GLP-1 Receptor Agonists (liraglutide, semaglutide)	Incretin mimetic: ↑insulin (glucose-dependent), ↓glucagon; slows gastric emptying; central appetite suppression	Obesity, metabolic syndrome; secondary benefits on hyperandrogenism and anovulation	Achieve significant weight loss (~5–10%) and ↓insulin resistance in P COS[5] pmc.ncbi.nlm .nih.gov. Superior to metformi n fo r BMI reduction; improves menstrual regularity and lower s androgens in many patients. Gaining acceptance as adjunct therapy in obese PCOS[5] pmc.ncbi.nlm.nih.gov.
SGLT2 Inhibitors (dapagliflozin, etc.)	Promote renal glucose excretion (↓blood glucose); osmotic diuresis and mild weight loss	O v e r w e i g h t / o b e s i t y ; metabolic dysfunction; possibly menstrual regularity	Early trial s re port ↓body weight, ↓visceral fat and androgen levels and improved cycle frequency[1]pmc.ncbi.nlm.nih.gov. Well tolerated; considered promis ing add-on for PCOS with impaired glucose tolerance.
Aromatase Inhibitors (letrozole)	Inhibit estrogen synthesis, ↑FSH release -> follicular growth	Anovulation (infertility treatment)	Now established 1st-line for ovulation induction in PCOS. Higher ovu lati on and live birth rates than clomiphene [6]mdpi.com. Rec ent studies confirm letrozole's efficacy across BMI cat egories; extended protocols can help letrozole-resistant cases.

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