

Method of Improving the Training Treatment Program in Polycystic Ovary Syndrome

Kh. B. Sattaraliyeva

Andijan State Medical Institute, Fergana Institute of Public Health, Uzbekistan

Abstract In the recent past, the term “polycystic ovary syndrome” (PCOS) was understood as a pathological condition of the reproductive system, characterized by a certain change in the structure and function of the ovaries, which is based on a violation of the hypothalamic regulation of the secretion of gonadotropic hormones. Polycystic ovary syndrome is one of the main causes of infertility and is a modern of obstetrics and gynecology current from problems It is considered. In treatment pathogenetic approach we this in the article illuminating we gave. This on purpose wide comprehensive literature comment transferred and hormonal and metabolic status correction through to treat this pathology directed research quoted.

Keywords Polycystic ovaries syndrome, Metabolic syndrome, Insulin resistance, Hyperandrogenism, Lipids exchange, Treatment

1. Introduction

Currently, the main cause of the development of polycystic ovary syndrome (PCOS) is unknown, so therapeutic approaches are symptomatic. The number of potentially effective drugs proposed is sufficient. Our knowledge of the true causes of PCOS is as limited as it is extensive. The success of any form of PCOS therapy depends on the treatment used, and sometimes on the characteristics of the population in which the therapy is administered [7,12].

The goal of PCOS therapy: Reducing the level of circulating androgens and reducing the clinical manifestations of hyperandrogenism (HA); improving reproductive function and fertility; reducing body weight in the presence of obesity; eliminating complications associated with PCOS and hyperinsulinemic insulin resistance (IR), such as glucose intolerance, dyslipidemia, arterial hypertension, atherosclerosis, and, most importantly, IUGR [3,12].

First Goal: to reduce circulating androgen levels and reduce the clinical manifestations of HA. In PCOS, HA is caused by hyperinsulinemic IR, so reducing circulating insulin leads to a reduction in the clinical manifestations of HA [11]. To achieve this goal, drugs are used that are directed at:

- 1) reducing the production of androgens (combined oral contraceptives containing drospirenone, cyproterone acetate; gonadotropin - releasing hormone agonists including drugs such as triptorelin, goserelin, buserelin, leuprorelin; ketoconazole),

- 2) peripheral blockade of the effect of androgens (cyproterone Acetate and spironolactone; flutamide, finasteride - directly antiandrogens, not licensed for use by women) [1,8,10].

Second goal: reproduction v function and improve fertility.

Hyperinsulinemia affects the IR at many levels - the pituitary gland, ovaries, peripheral metabolism of androgens - and causes chronic anovulation and infertility in women with PCOS. Pharmacological treatment aimed at improving insulin sensitivity helps to reduce its level and leads to spontaneous ovulation or improves standard ovulation induction schemes. To achieve this goal, antiandrogen± estrogen - progestin drugs are used for women with a normal body mass index and without GI. For patients with excess body weight and GI, - Insulin sensitizers are used in conjunction with weight management measures [4].

Biochemical evaluation before and after treatment showed positive changes.

Hormone levels in blood samples taken during the follicular phase, before and after treatment with Flutamide and at 6 months of treatment are shown. Significant decreases were observed in LH, androstenedione, testosterone and free testosterone levels, as well as in the LH/FSH ratio ($P < 0.01$). The patterns of LH and FSH levels over several days of the menstrual cycle before and after treatment for 6 months are shown. Before flutamide infusion, basal LH levels were high and without peaks, with a mean value of 14.2 ± 2.3 mIU/ml for all cycles. The mean LH/FSH ratio was 2.8 ± 0.3 . During therapy, a decrease in basal LH levels and the LH/FSH ratio was noted.

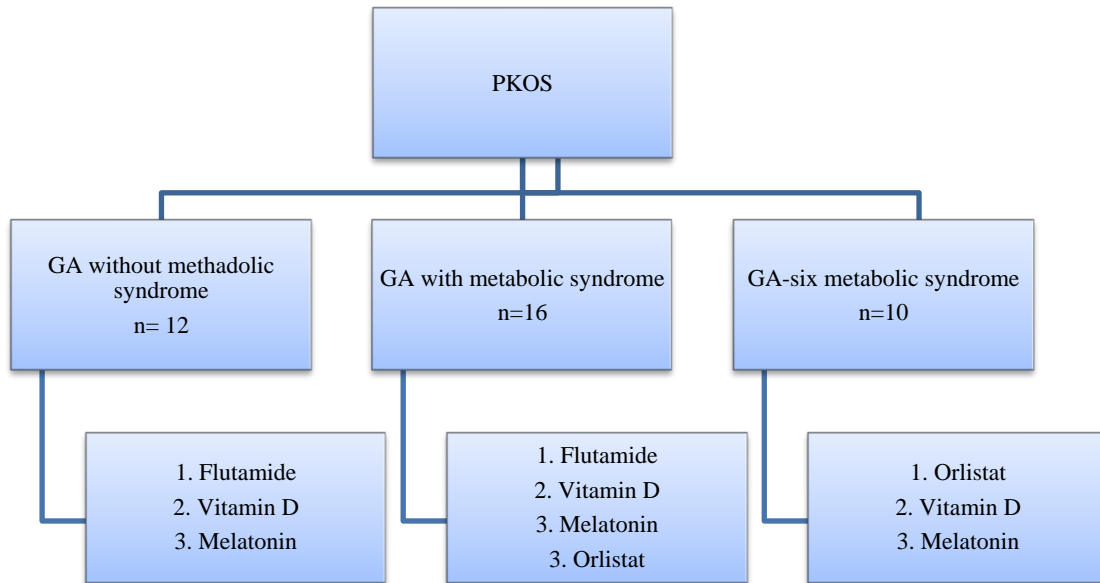


Figure 1. Algorithm of the preparatory stage for the main group before the EK program

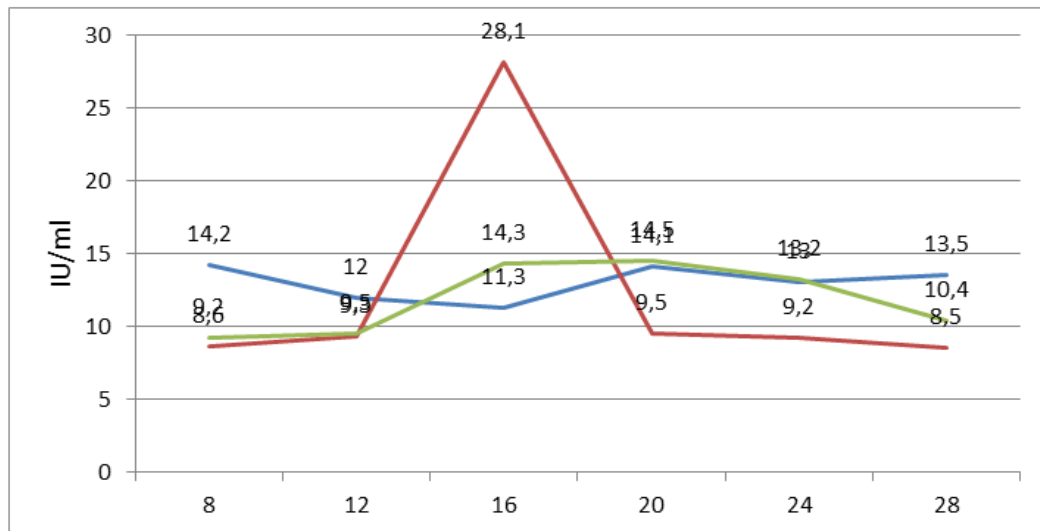


Figure 2. Changes in the level of LG during the menstrual cycle

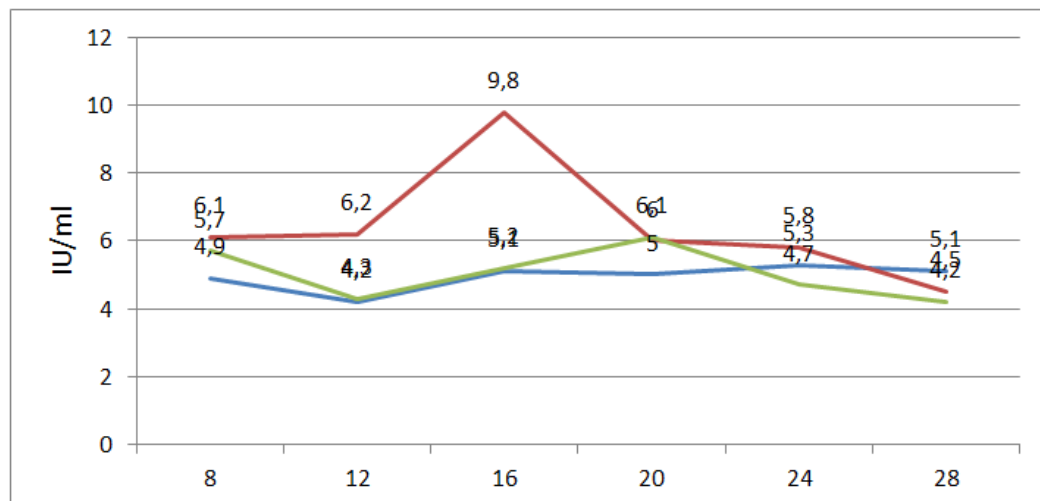


Figure 3. Changes in FSG levels during the menstrual cycle

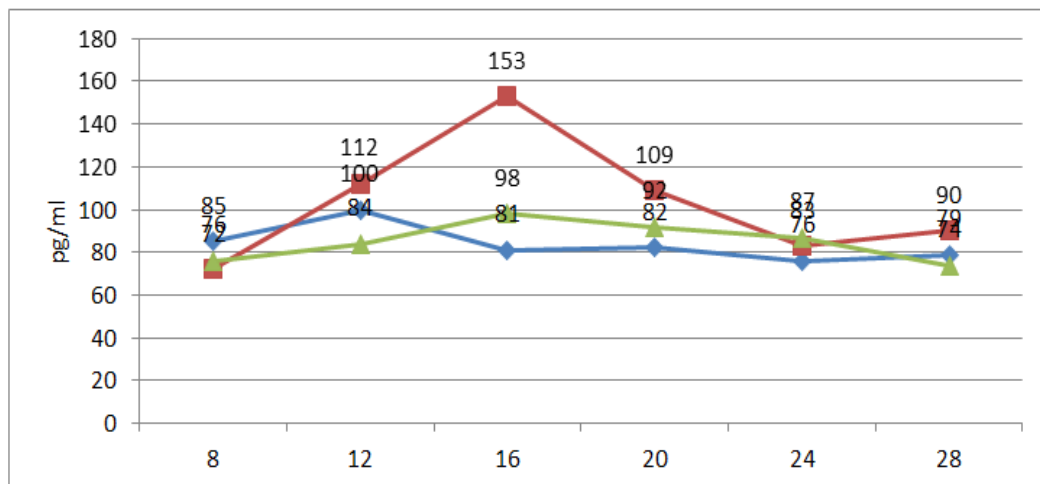


Figure 4. Changes in estradiol levels during the menstrual cycle

Our study shows that the inclusion of Flutamide in the treatment of PCOS with hyperandrogenism improves hormonal parameters, restores the ovulatory cycle, effectively reduces the level of androgens in the blood, and also reduces hirsutism and It treats acne. The results show that, in addition to blocking androgen receptors, Flutamide significantly reduces plasma androgen levels by inhibiting the atretic effects of LH on theca and granulosa cells.

2. Conclusions

1. As a result of the treatment during the preparatory phase, women in the main group tended to have a higher number of oocytes retrieved (15.0 vs. 13.0; $P < 0.001$) and a higher number of fertilizations (12.0 vs. 10.0; $P < 0.001$), as well as a higher number of embryos transferred (2.2 vs. 2.4; $P < 0.001$).
2. A higher IVF rate (80.5% vs. 75.3%; $P = 0.003$) and a higher number of cycles with quality embryos (97.6% vs. 95.0%; $P = 0.002$) were observed. Embryo type (cleavage stage embryo, or blastocyst) was not significantly different in both groups ($R = 0.430$). The frequency of implantation, the frequency of clinical pregnancy, and the frequency of childbirth were significantly higher in the main group than in the control group (respectively, 49.3% vs. 38.1%, 70.9% vs. 59.8%, and 58.3% vs. 52.1%; $R < 0.001$).
3. The developed improved program, unlike traditional measures that are long and phased, ensures the restoration of social and physical activity, increases the effectiveness of treatment and rehabilitation by 14%, and socio-economic efficiency by 65%.

REFERENCES

- [1] Alesi S, Ee C, Moran LJ, Rao V, Mousa A. Nutritional Supplements and Complementary Therapies in Polycystic Ovary Syndrome. *Adv Nutrition*. 2022 Aug 1; 13 (4): 1243-1266. doi: 10.1093/advances/nmab141.
- [2] Brutocao C, Zaiem F, Alsawas M, Morrow AS, Murad MH, Javed A. Psychiatric disorders in women with polycystic ovary syndrome: a systematic review and meta-analysis. *Endocrine*. 2018 Nov; 62 (2): 318-325. doi: 10.1007/s12020-018-1692-3.
- [3] Dabadghao P. Polycystic ovary syndrome in adolescents. *Best Pract Res Clin Endocrinology Metab*. 2019 Jun; 33(3): 101272. doi: 10.1016/j.beem.2019.04.006.
- [4] Dumesic DA, Hoyos LR, Chazenbalk GD, Naik R, Padmanabhan V, Abbott DH. Mechanisms of intergenerational transmission of polycystic ovary syndrome. *Reproduction*. 2020 Jan; 159(1): R1-R13.
- [5] Ganie MA, Vasudevan V, Wani IA, Baba MS, Arif T, Rashid A. Epidemiology, pathogenesis, genetics & management of polycystic ovary syndrome in India. *Indian J Med Res*. 2019 Oct; 150 (4): 333-344. doi: 10.4103/ijmr.IJMR_1037
- [6] Haddad- Filho H, Tosatti JAG, Vale FM, Gomez KB, Reis FM. Updates in diagnosing polycystic ovary syndrome-related infertility. *Expert Rev. Cattle Diagn*. 2023 Feb; 23(2): 123-132. doi: 10.1080/14737159.2023.2177536.
- [7] Karam M, Najjar H, El Sabban M, Hamade A, Najjar F. Regenerative Medicine for Polycystic Ovary Syndrome: Stem Cell-Based Therapies and Brown Adipose Tissue Activation. *Stem Cell Rev Rep*. 2023 May; 19 (4): 853-865. doi: 10.1007/s12015-023-10505-5.
- [8] Kunicki M, Smolarczyk R. Polycystic Ovary Syndrome and Fibrocystic Breast Disease: An Updated Review. *Horm Metab Res*. 2021 Apr; 53 (4): 219-224. doi: 10.1055/a-1392-0938.
- [9] Osibogun O, Ogunmoroti O, Michos ED. Polycystic ovary syndrome oath cardiometabolic risk: Opportunities for cardiovascular disease prevention. *Trends Cardiovascular Med*. 2020 Oct; 30(7): 399-404. doi: 10.1016/j.tcm.2019.08.010. Epub 2019 Sep 4. PMID: 31519403.
- [10] Ozgen Saydam B, Yildiz BO. Polycystic Ovary Syndrome oath Brain: An Update moment Structural oath Functional Studies. *J Clin Endocrinology Metab*. 2021 Jan 23; 106(2): e430-e441. doi: 10.1210/clinem/dgaa843. PMID: 33205212.
- [11] Palomba S, Colombo C, Busnelli A, Caserta D, Vitale G.

Polycystic ovary syndrome oath thyroid disorder: a comprehensive narrative review of the literature. *Front Endocrinol (Lausanne)*. 2023 Aug 11; 14: 1251866. doi: 10.3389/fendo.2023.1251866. PMID: 37635968; PMCID: PMC10453810.

[12] Palomba S, Falbo AI, Marci R, Caserta D. Is endometrial receptivity impaired in woman with polycystic ovary syndrome ? *Minerva Obstetrician Gynecol*. 2023 Apr; 75(2): 172-180. doi: 10.23736/S2724-606X.22.05144-2. Epub 2022 Aug 1. PMID: 35912467.

Copyright © 2025 The Author(s). Published by Scientific & Academic Publishing

This work is licensed under the Creative Commons Attribution International License (CC BY). <http://creativecommons.org/licenses/by/4.0/>