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CHANGES IN HORMONAL SPECTRUM IN OVARIAN POLYCYSTOSIS WITH CLOMIPHENREZISTENCE-BINDING INFERTILITY

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Resume: This study studied the risk factors for the development of recurrent polycystic ovaries, the role of obesity in its development. For the study, 92 women with recurrent polycystic ovaries aged 19-35 years with and without obesity were studied in the control group of 20 healthy women living in the Bukhara region. The study concluded that hyperinsulinemia plays an important role in the development of the disease in patients with obesity-related OC, while dysfunction associated with the hypothalamic-pituitary system (induced by stress), on the other hand, plays an important role in the development of OCJ independently. from obesity in patients of the second group.

Key words: recurrent polycystic lipid spectra,obesity,adrenocorticotropic hormones, thyroid hormones, prolactin, gonadotropins, autoimmune thyroiditis, hormonal contraceptives, thyroid hormones, prolactin, gonadotropins, steroid hormones.

Introduction. Obesity is one of the factors that induces ovarian polycystosis syndrome in women. It is known that there is a positive correlation between obesity and insulin resistance and hyperinsulinemia. In particular, adipose tissue has the ability to produce androgens, being involved in maintaining the balance of androgens and estrogens under the action of various activating and thinning factors [5,7]. It is known that in the case of hyperandrogenemia, the folliculogenesis process is captured at the known stage and undergoes atresia without moving on to further development [4,9,12]. According to the results of some studies, hyperandrogenemia has been found to cause stress disorder in the endoplasmic reticulum in Granulose cells and induce apoptosis in them by expression of autophageal-related genes [8,10]. Estrogens are important in the development of folliculogenesis through anti-apoptotic action [6,11,13]. Thus, decreased levels of estrogens caused by obesity in women result in indusiratory apoptosis in Granulose cells (including oocytes), and increased LG expression and increased tecocyte proliferation in the case of obesity-induced hyperinsulinemia cause the development of polycystosis.

The purpose of the study: to study the features of hormonal changes according to the degree of obesity in patients with ovarian polycystosis syndrome and conduct their analysis.

Research materials and methods. Patients in the main group were divided into two groups, depending on the TMI indicator in them, in order to more deeply determine the pathogenetic acuity of obesity, one of the factors that inoculate polycystosis, and in order to identify new molecular markers of practical importance in the prognosis of Polycystosis in the case of obesity. That is, patients with Polycystosis with obesity prevalence were 47 (the main group - 55.6%), and patients with non – obesity TPKS accounted for 45 (comparative guru-44.4%), and hormone changes were analyzed, which were considered markers of pathophysiological changes characteristic of obesity.

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Results and discussion. During the study, the results of in-depth biochemical tests were carried out in comparative and basic groups in patients with control, Polycystosis. The goal of this was to understand more deeply the pathogenesis of the development of polycystosis in women, precisely in the case of obesity and independently of obesity (see Table 1). As shown in Table 2, patients with ovarian polycystosis who do not have obesity and who have been diagnosed with obesity (comparative group) were found to have an increase in insulin concentration of 1.53 times ($p<0.01$) compared to control group indicators, typical of women with ovarian polycystosis and obesity. This group observed that patients ' serum LG increased 4.1 times ($p<0.001$) compared to control group indicators, decreased FSG by 1.69 ($p<0.01$) times, and increased their ratio (LG/FSG) by 2.42 ($p<0.001$) times, respectively. In the case of a comparative group of patients with increased serum AMG levels, we can see that the total and free testosterone levels decreased by 2.3 ($p<0.001$) and 1.85 ($p<0.001$) times, respectively, and estradiol levels by 1.29 ($p>0.05$) times. Such changes caused the estradiol testosterone ratio (E2/T) to drop by almost 3 ($p<0.001$) times, and consequently observed a decrease in aromatase activity by 66.4% ($p<0.001$), respectively, compared to control group indicators. Chen J. and according to the data cited by his cockroaches, the E2/T ratio is one of the reliable methods in the elimination of aromatase activity [1,4].

Table 1.

Women with polycystosis change the amount of hormones in the blood serum, M±m

Specification	Control group (TMI<24,9),n=20	Comparative group (TMI<24,9), n=45	Main group (TMI>29,9), n=47
Insulin, μU/mL	12,1±3,15	18,52±2,09	27,96±1,84[ab]
LG, Med/l	2,9±0,8	11,9±1,81[a]	12,07±1,66[a]
FSG, Med / l	4,9±0,42	2,9±0,31[a]	3,70±0,52[b]
LG/FSG	1,69±0,26	4,1±0,21[a]	3,43±0,14[ab]
AMG, nmol/l	3,3±0,68	3,9±0,33	4,8±0,29[ab]
Common Testosteron, ng / dl	31,93±12,4	73,34±15,7[a]	96,88±11,8[ab]
Free Testosteron, PG/ml	2,37±0,11	4,4±0,17[a]	5,16±0,22[ab]
Esterodiol, PG/ml	139±21,6	107,3±5,4	58,6±5,1[ab]
E2/T	0,435±0,037	0,146±0,034[a]	0,06±0,031[ab]
SHBG, мг/л	10,65±0,23	13,82±0,22[a]	15,9±0,31[ab]
DGEA, mkmol/l	11,2±0,85	14,8±1,4[a]	15,9±0,744[a]
Progesterone, nmol/l	1,6±0.3	1,85±0,24	1,45±0,9

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TTG, mme/l	1,8±0,156	3,4±0,56[a]	2,96±0,32[a]
T4, nmol/l	17,3±1,44	13,9±1,34[a]	14,8±1,8[a]
Prolactin, mme/l	369,8±13,9	715,4±21,4[a]	649,1±24,8[ab]
AKTG, pmol/l	13,8±2,9	43,7±3,7[a]	23,7±4,5[b]
Cortisol, nmol/l	492±21,2	774,8±26,0[a]	660±33,5[ab]

Indication: p<0.05 compared to a – control group, B - compared to the result shown by non – obese patients-p<0.05.

Women with polycystosis and no obesity were found to have increased serum levels of SHBG and DGEA by 1.3 (p<0.05) and 1.32 (p<0.05) respectively, while there was a tendency to increase progesterone levels. Prolactin levels were found to increase 1.93 (p<0.001) times. Analysis of thyroid indicators showed that TTG levels were increased by 1.89 (p<0.001) in comparative group Women's blood serum, while T4 levels decreased by 1.25 (p<0.05) times. This is evidenced by the presence of hypothyroidism in women. ACTG and cortisol levels have been found to increase by 3.4 (p<0.001) and 1.57 (p<0.01) times, and indicate the presence of hypercortisolemia (Itsenko-Cushing syndrome). As shown in Table 1, the determination of serum hormone levels in women with TPKS and Obesity showed an increase in insulin concentration of 2.32 (p<0.001) and 1.51 times (p<0.01) compared to the indicators of control and comparative groups, and indicated the presence of changes characteristic of obesity and polycystosis. The main group did not differ from the comparative group indicators, in which the amount of LG in women's blood serum increased by 4.2 times (p<0.001) compared to the indicators of the control group. While the amount of FSG decreased by 1.32 (p<0.05) times compared to the control group indicators, it was 1.28 (p<0.05) times higher than the comparative group indicators, keeping the LG/FSG indicator at 2.03 (p<0.001) times lower than the comparative group indicators at 1.2 (p<0.05) times. The amount of AMG increased convincingly compared to control and comparative groups of 1.45 (p<0.05) and 1.26 (p<0.05) times. We observed an increase in total and free testosterone levels of 3.03 (p<0.001) and 2.18 (p<0.001) times compared to control group indicators, 1.32 (p<0.05) and 1.17 (p<0.05) times compared to comparative group indicators. We observed a corresponding decrease in Estradiol levels of 2.37 (p<0.001) and 1.83 (p<0.001) times compared to the indicators of control and comparative groups. Such changes were found to reduce estradiol testosterone ratio (E2/T) by almost 7.25 (p<0.001) and 2.43 (p<0.001) times compared to the group indicators noted above, and consequently decreased aromatase activity by 86.2% (p<0.001). When the amount of SHBG increased by 1.5 (p<0.05) times compared to the control group indicators, it was found that there is a tendency to increase compared to the comparative group indicators. Similar changes were also observed in the amount of dgea: that is, it was found that the motility increased by 1.42 (p<0.05) times compared to the control group indicators, compared to the comparative group. it has been found that gannicity, and progesterone levels have not changed in static reliability compared to control and comparative group indicators. Patients were found to be prone to decreased serum prolactin levels compared to comparative group indicators if they increased by 2.41 (p<0.001) times compared to the control group. Testing of other hormones in patients showed a 1.64 (p<0.01) increase in TTG compared to the control group, a 1.24 (p<0.05) 1.17 (p<0.05) decrease in T4, compared to comparative group indicators we showed a decrease in

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TTG and an increase in T4. The amounts of ACTG and cortisol increased by 2.72 ($p<0.001$) and 1.34 ($p<0.05$) times compared to the control group, while showing a decrease of 1.84 ($p<0.01$) and 1.17 times compared to the comparative group indicators.

Conclusion. From the changes cited, it can be concluded that patients who do not have obesity, especially high LG/FSG ratio and consequently decreased aromatase activity (low E2/T ratio), have the highest levels of AKTG, TTG, cortisol and prolactin in the same group, develop tpks mutagily into obesity, and the development pathogenesis of chronic stress induced dysfunction related to the hypothalamo-pituitary system muxim pathological change in the ratio of indusirated LG/FSG, it can be assumed that there is a strong decrease in aromatase activity in the ham – the development of TPKS in these patients, insulin resistance has acquired the main Achaemenid. After all, chronic stress can induce hypothalamic-pituitary-adrenal linkage disruption and thus inducing many ACTG diversions from the pituitary [2,7,13]. High amounts of ACTG, in turn, along with stimulating the production of many gluglucocorticoids from the adrenal gland, cause ham to increase the production of androgens from the retina [2,4,8]. As presented in Table 2, the hypothalamo-hypophysar disorder not only caused ACTG abberant production, but also caused LG/FSG ratio Ham strong disorder (higher than the result shown by obese patients). As mentioned above, the violation of the LG/FSG ratio, the normative development of thecocytes and Granulose cells in follicles, incapacitates the process of normal folliculogenesis by causing a violation of the balance of estrogen and androgens [4; 11,13].

Literature used:

1. Alieva, F. N. Izuchenie osnovnix etiopathogeneticheskix michanov formirovaniya syndroma polycystoznix yaichnikov / F. N. Alieva, N. V. Samburova / / molodezhny nauchny forum: estestvennie i medisinskie nauki. – 2017. – № 2(41). - S. 16-25. 2
2. Blesmanovich, A. E. Syndrome polikistoznich yaichnikov: classics I Sovremennie nyuansi / A. E. Blesmanovich, Yu. A. Petrov, A. G. Alexina / / magazine nauchnix statey Zdorove I obrazovanie v XXI veke. – 2018. - T. 20. – № 4. - S. 33-37.
3. 3. Vedzijeva E. R. K voprosu o pathogeneze reproductivnix narusheniy u jentshin s ojireniem / E. R. Vedzijeva, I. V. Kuznesova, Yu. B. Uspenskaya [I dr.] / / Akusherstvo I gynecology. – 2017. – № 6. - S. 18-25.
4. Glukhova M. V. Osobennosti klinicheskix, hormonalnix I metabolicheskix narusheniy u bolnix s syndromom polikistoznix yaichnikov s raznimi tipami ojireniya I normalnoy Massoy tela / M. V. Glukhova, T. V. Draznina, T. V. Karpov [I dr.] / / Upravlenie kachestvom medisinskoy pomotshi. – 2016. – № 1-2. - S. 33-37.
5. Grigoryan, O. R. Syndrome polycystaznix yaichnikov-atdalennie risk / o. R. Grigoryan, E. N. Andreeva / / Effektivnaya pharmacotherapy. – 2015. – № 5. - S. 20-25.
6. Dubrovina S.O., Berlim Yu.D., Krasilnikova L.V. I dr. Prognozirovanie residiva endometrioidnix cyst yaichnika vo vremya pervichnogo operativnogo vmeshatelstva. // Tavrichesky mediko-biologichesky vestnik. – 2018. - № 21(2). - S.26-30.
7. Zaydieva, Z. S. Syndrome polycystoznix yaichnikov: Sovremennie predstavleniya pathogenesis, diagnostic I lechenia / Z. S. Zaydieva, A. T. Uruymagova / / medisinsky Soviet. – 2021. – № 13. - S. 102-111.
8. Karimova N.N., Naimova N.S., Halimova E.M., Karimov D.N. Metodi korrektsii hormonalnogo dysbalansa U jentshin s syndromom prejdevremennoy nedostatochnosti yaichnikov / / New Day in medicine. - Tashkent, 2021. -№3(35/1). - S.203-206.

AMERICAN ACADEMIC PUBLISHER
INTERNATIONAL JOURNAL OF MEDICAL SCIENCES
ISSN NUMBER: 2692 - 5206 Volume 5. No 11. November ,2025

9. Levine S, Muneyyirci-Delale O. Stress-Induced Hyperprolactinemia: Pathophysiology and Clinical Approach. Obstet Gynecol Int. 2018 Dec 3;2018:9253083. doi: 10.1155/2018/9253083. PMID: 30627169; PMCID: PMC6304861.
10. Nayimova N.S., Karimova N.N. Disharmony and its correction in premature ovarian insufficiency //Journal of Pharmaceutical Negative Results, Vol. 13 special ISSUE 08 (2022) Scopus. P 3328-3332
11. Horm Behav. 2011 Aug;60(3):284-91. doi: 10.1016/j.yhbeh.2011.06.003. Epub 2011 Jun 12. PMID: 21689656; PMCID: PMC3148770.
12. 26. Gao Y , Chen F , Kong QQ , Ning SF , Yuan HJ , Lian HY , Luo MJ , Tan JH . Stresses on female mice impair oocyte developmental potential: effects of stress severity and duration on oocytes at the growing follicle stage. Reprod Sci. 2016;23(9):1148–1157.
13. Prasad S, Tiwari M, Pandey AN, Shrivastav TG, Chaube SK. Impact of stress on oocyte quality and reproductive outcome. J Biomed Sci. 2016 Mar 29;23:36. doi: 10.1186/s12929-016-0253-4. PMID: 27026099; PMCID: PMC4812655.