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# Changes in Serum Lipid Indicators during the Development of Ovarian Polycystosis Syndrome

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## ABSTRACT

Obesity is one of the tpks inducing factors in women. It is known that there is a positive correlation between obesity and insulin resistance and hyperinsulinemia. In particular, insulin and IGF 17-al'fa can induce hydroxylase and LG receptor ecpression. Perhaps, it is through this pathogenetic mechanism that hyperinsulinemia can cause the development of TPKS in many cases. After all, pathological conditions such as hyperinsulinemia and insulin resistance increase the LG sensitivity of thecocytes and disrupt its repression by re-negative binding . For this reason, as a result of increased LG secretion in tpks pathogenesis, and as a result of tecocyte hyperplasia and LG indusiratory hypeandrogenemia, the capture of follicles in antral state (non-ovulation) causes specific cystic structures to originate after a certain period of time.

**Purpose of the study:** changes in serum lipid indicators in the development of ovarian polycystosis syndrome

**Materials and methods:** in order to achieve our goal, 304 patient women who live permanently in the Bukhara region, have obesity and diagnosis of polycystosis were examined.

For this reason, in order to more deeply determine the pathogenetic acuity of obesity, one of the tpks inactivating factors, and in order to identify new molecular markers of practical importance in the prognosis of tpks in the case of obesity, patients in the main group are guided by the TMI indicator in them it was divided into two gurukhs. That is, tpks li patients with obesity were 50 (the main group - 55.6%), and tpks patients with obesity were 40 (comparative guru – 44.4%). First of all, the marker of pathophysiological changes characteristic of obesity was examined for biochemical changes (3.1.- look at the table).

As presented in Table 3.1, when the comparative group observed a decrease in total cholesterol levels in blood serum compared to the results of the control group, a decrease in the amount of ZPLP-XS by 1.17 ($p<0.05$) times was observed, the amount of ZYULP-XS increased by a statistically convincing 1.27 ($p<0.05$) times. This caused the atherogenicity coefficient to decrease by 1.44 ($p<0.05$) times.

1.33 ($p<0.05$) 1.34 ($p<0.05$) 1.34 ($p < 0.05$). While zplp-XS has been increased by 1.73 ($p<0.01$) and 2.02 ($p<0.001$) times, ZYULP-XS has been shown to decrease by 1.93 ($p<0.001$) and 2.45 ($p<0.001$) times. 3.31 ($p < 0.001$) 4.78 ($p<0.001$).

Interestingly, in all observed results, the rates of patients with tpks with obesity and non-obesity conditions are opposite, which also confirms the reliability of patient guruhinization on the clinical side, and it was found that patients with obesity have a high risk of developing atherosclerosis.

**Conclusion**

In the course of the investigation, it became known that in the case of lipid metabolism markers, obesity was found to have a statistically reliable decrease in total XS, ZPLP and ar indicators, compared to the indicators of non-fat and conditionally healthy examiners, and ZYULP. As such, according to the results of in-depth biochemical tests, statistical reliable balance of LG/FSG, total and free testosterone levels, E2/T ratio, SHBG, DGEA, TTG, T4, prolactin and cortisol indicators compared to the results of the control group, when compared with patient groups, patients who do not have obesity have a reliable balance of LG/FSG ratio, estradiol, prolactin, ACTG and cortisol indicators, on the other hand, patients who have obesity, insulin, AMG, general and free testosterone, It was found that the E2 / T ratio was statistically reliably reduced.When diagnostic efficacy was independently tested in patient groups on SHBG balandigi and so on LG/FSG, E2/T, insulin, and AKTG, its quality was 0.91, 0.96, 1.0, and 0.96 in obese patients, respectively, and 0.94, 0.94, 0.88, and 1.0 in non-obese patients, respectively.

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