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РОЛЬ МИО-ИНОЗИТОЛА В ЛЕЧЕНИИ БЕСПЛОДИЯ У ПАЦИЕНТОК С СИНДРОМОМ ПОЛИКИСТОЗНЫХ ЯИЧНИКОВ

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АННОТАЦИЯ

Синдром поликистозных яичников (СПКЯ) является широко распространенным заболеванием, представляющим собой единственное наиболее распространенное эндокринно-метаболическое расстройство у женщин репродуктивного возраста. СПКЯ отмечается в 30-40% случаев резистентностью к инсулину и гиперандрогенией. Было отмечено, что мио-инозитол повышает чувствительность к инсулину, снижает гиперандрогению и улучшает менструальный цикл. Мы провели исследование влияния мио-инозитола на СПКЯ, и он показал высокую эффективность в нормализации функции яичников и созревании ооцита и впоследствии возникновении беременности.

Ключевые слова: синдром поликистозных яичников, мио-инозитол, чувствительность к инсулину, гиперандрогения, дисменорея.

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THE ROLE OF MYO-INOSITOL IN TREATING INFERTILITY AMONG PATIENTS WITH POLYCYSTIC OVARY SYNDROME

ABSTRACT

Polycystic ovary syndrome (PCOS) is a highly prevalent disorder, representing the single most common endocrine-metabolic disorder in reproductive-aged women. PCOS is marked in 30 to 40% by insulin resistance and hyperandrogenism. Myo-inositol was noted to increase insulin sensitivity,decrease hyperandrogenism and improve the menstrual cycle.We conducted a research on the impact of Myo-inositol administration in PCOS and it marked a high effectiveness in normalizing ovarian function and maturing the oocyte and leading to pregnancy.

Key words: polycystic ovarian syndrome, myo-inositol, insulin sensitivity,hyperandrogenism, dysmenorrhea

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TUXUMODON POLIKISTOZ SINDROMI BO'LGAN BEMORLARDA BEPUSHTLIKNI DAVOLASHDA MYO-INOSITOLNING O'RNI

ANNOTATSIYA

Tuxumdon Polikistoz sindromi juda keng tarqalgan kasallik bo'lib, reproduktiv yoshdagi ayollarda eng ko'p uchraydigan endokrin-metabolik kasallikdir. Polikistik tuxumdon sindromi 30-40% insulin sezuvchanlik va giperandrogenizm bilan belgilanadi. Miyo-inositol insulinga sezgirlikni oshiradi, giperandrogenizmni kamaytiradi va hayz davrini yaxshilaydi. Biz Myo-inositolni polikistik tuxumdon sindromiga ta'siri bo'yicha tadqiqot o'tkazdik va bu tuxumdonlar faoliyatini normallashtirish va oositni yetishtirishda yuqori samaradorlikni ko'rsatdi va keyinchalik homiladorlikka olib kelishi kuzatildi.

Kalit so'zlar: polikistik tuxumdon sindromi, mio-inositol, insulin sezuvchanlik, giperandrogenizm, dismenoreya.

Introduction. Polycystic ovary syndrome is a highly inherited complex polygenic, multifactorial disorder. Pathophysiologically abnormalities in gonadotropin secretion or action, ovarian folliculogenesis, steroidogenesis, insulin secretion or action, and adipose tissue function, among others, have been described in PCOS currently there are four recognized phenotypes of PCOS:

1) hyperandrogenism+oligo-anovulation+polycystic ovarian morphology;

2) hyperandrogenism+oligo-anovulation;
3) hyperandrogenism+polycystic ovarian morphology; and
4) oligo-anovulation+polycystic ovarian morphology, each with different long-term health and metabolic implications. [2]

Polycystic ovary syndrome (PCOS) is the most common cause of ovulation disorders, hyperandrogenism and infertility due to ovulatory dysfunction, affecting more than 7% of childbearing age women. as previously mentioned, PCOS is associated with insulin resistance in 30–40% of cases.Hyperglycemia inhibits hepatic production of Sex Hormone Binding Globulin (SHBG), which leads to an increase of free androgens in the blood circulation, and insulin resistance increases the production of androgens by the theca cells. The management of this insulin resistance is therefore essential in the treatment of PCOS, and is based on nutritional rules, physical activity and other molecules including myo-inositol (MI).

The purpose of research: To check the efficiency of myo-inositol(Inotir in our case) in treating infertility among women with polycystic ovarian syndrome.

Materials and methods of research:

Complex observation of a group of women (the age within 20-30 years) were positively tested on having the polycystic ovarian syndrome. The group included 52 patients of Andijan Regional Perinatal Centre with hyperandrogenism(detected via clinical and biochemical analysis), oligo-anovulation and polycystic ovarian morphology(detected via ultrasound diagnostics or tests of functional diagnostics) suffering from infertility.

Results and discussion:

Inositol is a polyalcohol of which there are nine stereoisomers (cyclohexane-1,2,3,4,5,6-hexol). Two of them have been shown to mediate the post-receptor effects of insulin: myo-inositol (MI-cis-1,2,3,5-trans-4,6-cyclohexanehexol) and D-chiro-inositol (DCI-cis-1,2,4-trans-3,5,6-cyclohexanehexol) (DCI). The food categories that contain the highest concentration of inositols are fruits, beans, corn and nuts. At the intestinal level, DCI negatively interferes with MI absorption. Uptake of free inositol by tissues occurs by a membrane dependent sodium inositol cotransporter.. MI and DCI are controlled by some inositol phosphoglycans (IPGs), known as second messengers. These mediators are then internalized and modify enzymatic activity and intracellular metabolism, mimicking the action of insulin. When insulin binds to its receptor, these IPGs are generated by hydrolysis of glycosylphosphatidylinositol (GPI) lipids and/or specific proteins located on the outer part of the cell membrane. Two IPGs are formed: IPG-DCI (or IPG-P) and IPG-MI (or IPG-A). IPG-P will directly activate the glycogen synthase but will also indirectly activate it via the activation of phosphoprotein phosphatase 1 (PP1). IPG-A causes direct glucose uptake and inhibits cAMP protein kinase A and adenylate cyclase, thereby activating PP1. These effects allow a decrease in blood glucose levels (insulin-like effect), regardless of the signal passing through the insulin receptor [3]. In women with PCOS, impaired inositol and/or GPI metabolism contributes to insulin resistance, but obesity plays a specific role in abnormal IPG-P production independently of PCOS [3]. MI decreases body weight, leptin secretion and increases HDL cholesterol [4] Thanks to its antioxidant action (SOD, catalase and GSH increase), MI improves cell morphology and growth, as well as the synthesis of lipids participating in cell membranes. Figure 1 summarizes the different actions of MI in the ovary.

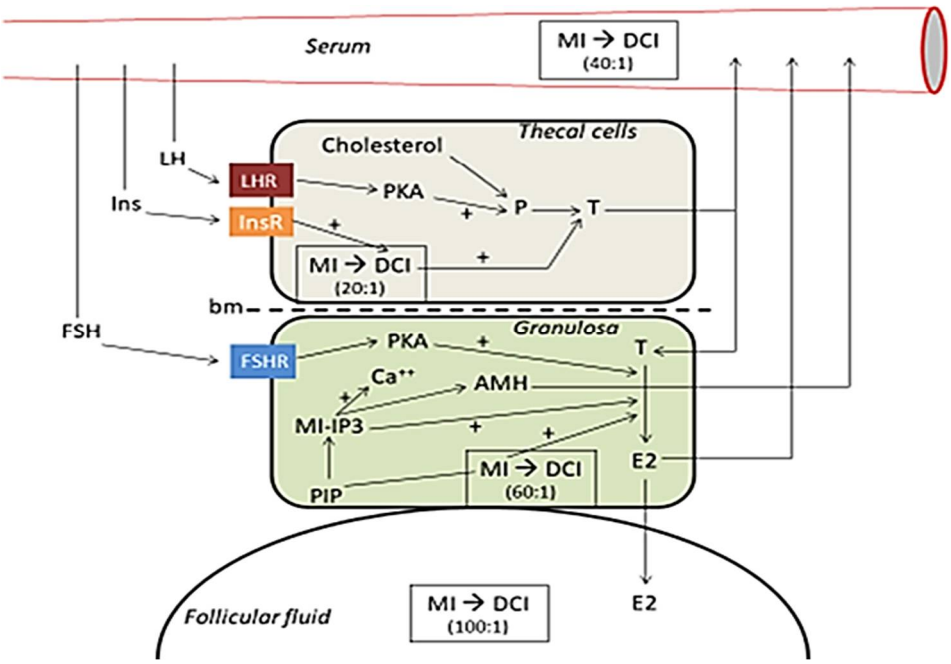

Fig.1 Roles of myo-inositol (MI) in the ovary (original figure from the author, after review of patho-physiologic effects of MI, DCI and others hormones on ovarian cells). MI: myo-inositol; DCI: D-chiro-inositol; (40:1): MI/DCI ratio; LHR: LH receptor; PKA: protein kinase A; P: progesterone; T: testosterone; INs: insulin; InsR: insulin receptor; bm: basalis membrane; PIP: phospho inositide phosphate; IP3: inositide triphosphate; AMH: anti-Müllerian hormone; E2: estradiol; + : stimulating effect

In PCOS, early follicular growth is excessive, but subsequent progression to a dominant follicle is interrupted (follicular arrest). Intraovarian androgens have been implicated in the excess of follicles and the elevated serum estradiol levels. This increased production of androgens is an inherent property of thecal cells, but it is increased by the surplus of LH and by hyperinsulinism. In women with PCOS, treatment with metformin (MET) ameliorated the insulin sensitivity and decreased the androgens levels, but the limitations to MET use are its gastrointestinal side effects. In this case of PCOS, the place of MI was evaluated. Studies show that MI leads to a decrease in LH and androgen levels, as well as a decrease in insulin resistance. Thus, MI is believed to be able to re-establish ovulatory menstrual cycles (especially in obese women with PCOS).

As a result, in our research with PCOS patients, after administering drug INOTIR 3g twice a day these changes were noted:

1. Relative lowering of blood insulin levels
2. Decreased testosterone concentration
3. Increase in number of globulins binding sex hormones after administering the drug during 24 weeks and more.
   And occurrence of pregnancy due to symptomatic refinement.

Conclusion.

Polycystic ovarian syndrome is a highly inherited complex polygenic, multifactorial disorder. Pathophysiological abnormalities in gonadotropin secretion or action, ovarian folliculogenesis, steroidogenesis, insulin secretion or action, and adipose tissue function, among others, have been described in PCOS. Insulin resistance increases the production of androgens by the theca cells .Elevated androgen levels play a significant role in menstrual cycle disrturbance and anovulation . These factors then become obstacles in occurrence of pregnancy. Myo-inositol (MI) increases insulin sensitivity, decreases hyperandrogenism and improves the menstrual cycle as a result leading to pregnancy.

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