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Proceedings of the International Conference on Sustainable Development:
Problems, Analysis And Prospects

Genetic Study of the Risk of Volumetric Prostheses

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Abstract: *A chronic disease caused by weakening of the musculoskeletal system of the pelvic organs, which leads to a decrease in the walls of the vagina and prolapse of the uterus. A key role in the pathogenesis of genital prolapse is played by the expression of genes encoding connective tissue, a1 type I COL1A1 collagen, a1 type III Col a1III proteins. Type I collagen is a component of the vaginal walls, sacro-uterine ligaments, and pelvic parietal fascia. Type III collagen in the pelvis predominates in the walls of large blood vessels and in the connective tissues that surround the vagina and other internal organs. These fibrillation types of collagen provide elasticity and mechanical strength and affect cell growth and differentiation.*

Keywords: *genital prolapse, collagen, connective tissue.*

Introduction

In past few years, researchers have done everything they can to figure out the molecular genetic nature of connective tissue diseases, genes linked to mutations, the formation of the primary structure of collagen, extracellular matrix components, and many enzymes involved in intracellular and extracellular collagen maturation, fibrilogenesis, and collagen fiber formation. [1]. Forms I and III are the primary components of epithelial tissue, and there are 19 different types of collagen. They are huge interstitial fibrils of collagen. The stroma of the human body, as well as ligaments, tendons, blood vessels, skin, and internal organs, include this form of collagen [2].

The gene encoding the a1 chain of collagen type COL1A1-I. The product of collagen denaturation is gelatin. The temperature of collagen macromolecule denaturation is close to the temperature of fibril formation. This property of the collagen molecule makes it most sensitive to mutational substitutions.

Fibriogenesis is the formation of collagen fibers in the cells and tissues of the human body by attaching to bundles of fibrillar-thin protein filamentous structures or in connective tissue. The stronger the collagen fibers formed during fibrogenesis, the stronger the connective tissue. A component of type I collagen called the pro-a1(I) chain is made from the COL1A1 gene. Collagen begins as rope molecules of tropocollagen, each consisting of three chains.

Two pro-a1 (I) chains and one pro-a2 (I) chain make up type I collagen (produced from the COL1A2 gene). Enzymes process three types of tropocollagen molecules in stages: mature collagen is generated both inside and outside the cell. Collagen molecules then split into long, thin fibrils that create stable cross-links in the gaps between cells. Cross-linking causes type I collagen fibers to become extremely strong. At the TTA obstetrics and gynecology complex, 61 women with a diagnosis of genital prolapse (main group) were examined on a volunteer basis. The individuals studied ranged in age from 18 to 68 years old (median - 39.0 years).

For a more accurate assessment of the significance of the polymorphic marker of genetic polymorphism COL1A1 (rs1107946) and COL4A1 (rs605143) in the formation of genital

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prolapse, the main group was divided into 1 and 2 (comparison groups). Group 1 consisted of patients with pelvic organ prolapse (n = 31) with conventional surgical treatment of COL1A1 (rs1107946) and COL4A1 (rs605143) genetic polymorphisms;

Group 2 - patients with prolapse (n = 30) who have been on a pelvic floor muscle stimulator for 6 months. As a control group, the DNA chain of the Department of Molecular Medicine and Cellular Technologies of the Research and Development Institute of the Ministry of Health of the Republic of Uzbekistan and DNA were studied. 63 apparently healthy patients with PTO and other asymptomatic gynecological diseases selected from PCa. All of them corresponded to the age of the study group.

When studying the predictive effectiveness of the genes of polymorphism of the COL1A1 and COL4A1 genes rs1107946 and rs605143, a significant increase in the AUC in patients (group 2) with genital prolapse at the level of 0.515 ($p<0.05$) was studied. The prognostic efficiency of the rs1107946 polymorphism in the group of women with pelvic organ prolapse (group 1) according to the auction values was 0.50 ($p>0.05$), and rs605143 was 0.5 ($p>0.05$). This showed sufficient predictive value as an independent marker for predicting the development of pelvic organ prolapse.

In the main group of patients, genotypes G/G, G/A, A/A had empirically observed and theoretically expected levels of 0.66/0.63 0.28/0.33, 0.07/0.04.

In control group samples, the level of these genotypes was 0.73 / 0.72; 0.24 / 0.26; 0.03 / 0.02. From the above results, it can be seen that the comparative difference between the observed and expected prevalence rates of genotypes in both groups was statistically insignificant (ch2 <1.28; R> 0.251).

Conclusion

In conclusion, the COL1A1 and COL4A1 locus genotypes are in Hardy-Weinberg equilibrium, the AUC is the prognosis efficiency, and * p is Fisher's exact test. The polymorphism of the COL1A1 and COL4A1 genes is crucial in the development of pelvic insufficiency, according to the Association's findings, and may enhance the chance of acquiring this condition. Choosing the proper approaches requires using all genetic diagnostic methods, enhancing the quality of surgery, therapy, and prevention of genital prolapse in all age groups.

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