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CHARACTERISTICS OF IMMUNE DISORDERS IN CHRONIC PROSTATITIS DEPENDING ON THE PRESENCE OF METABOLIC SYNDROME IN YOUNG MEN

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Annotation. The use of panavir in the complex of etiopathogenetic therapy of patients with chronic bacterial prostatitis in combina tion with metabolic syndrome statistically significantly improves the results of treatment of chronic prostatitis, indicators of lipid metab olism, increases the fertility of the ejaculate. There were no adverse reactions to the drug. Should include panavir in the standard of therapy for patients with chronic bacterial prostatitis.

Keywords: prostate, metabolic syndrome, infertility, sperm, ejaculate, immunoactive substances.

The incidence of chronic prostatitis (CP) in Russia is unknown according to official statistics from the Ministry of Health, but there is data on the frequency of this disease among outpatient urological patients: the proportion of prostatitis ranges from 4.5 to 42.5% depending on the affiliation of the medical institution (municipal or private) [1]. The cohort of patients with CP is heterogeneous; there is no indisputable data that the type of constitution, race, type of activity (physical or mental labor), the presence of bad habits, etc. contribute to the development of CP.

The standard definition of metabolic syndrome (MS) implies a combination of abdominal obesity, carbohydrate and lipid metabolism disorders, and arterial hypertension [2], although there is also a broader understanding of MS: a metabolic disorder in general [3-4]. MS correlates with prostate volume and the risk of developing urinary disorders [1,5], has a positive correlation with the level of prostate-specific antigen [6] and increases the risk of prostate cancer [2,7]. Moreover, MS is a strong risk factor for the development of urinary incontinence and erectile dysfunction after prostatectomy [1,8]. MS also has a strong correlation with the development of benign prostatic hyperplasia [2,9] and is one of the factors of its progression [3,10]. Many studies confirm the negative impact of MS on ejaculate fertility [1,2,17]; comorbidity with MS leads to a statistically significantly more frequent violation of sperm

morphology [12]. It has been established that MS contributes to the maintenance of chronic inflammation. In patients with MS, the level of C-reactive protein as an inflammation marker should be determined [13]. MS provokes and aggravates a chronic systemic inflammatory response [14]. The degree of metabolic disorders in MS positively correlated with the degree of prostate inflammation, which was confirmed by changes detected during ultrasound examination and the level of interleukin-8 (IL-8) [15-16]. It should be noted that calcification foci in the prostate parenchyma detected during ultrasound examination are usually interpreted as prostatolithiasis, although in reality these may be calcified foci of undiagnosed tuberculosis, proceeding under the guise of CP [17]. Patients who had 3 or more components of MS significantly more often had CP [12]. Thus, MS is a risk factor for the occurrence and more severe course of CP.

Purpose of the study: The levels of pro- and anti-inflammatory cytokines and vascular growth factor in the blood serum and ejaculate of men with chronic prostatitis were studied depending on the presence of metabolic syndrome at a young age.

Materials and methods of research: To carry out this research work, 45 young men aged 27 to 44 years with chronic prostatitis who applied to the urology department of the Bukhara Regional Multidisciplinary Medical Center were examined in the period from 2023 to 2024. This cohort was divided into two subgroups: The main group (n = 20) - men with chronic prostatitis in combination with metabolic syndrome; the comparison group (n = 25) - men with chronic prostatitis without metabolic syndrome.

Research results and their discussion: In the first stage of our study, we examined the level of pro- and anti-inflammatory cytokines (IL-2, IL-4, TNFa) and vascular growth factor (VEGF) in the blood serum of patients with chronic prostatitis without metabolic syndrome (comparison group, n = 25).

IL-2 is a key cytokine that stimulates T-lymphocyte proliferation, which reflects the adaptive response of the body to the inflammatory process in the prostate []. In the control group, this indicator was 13.2±0.17 pg/ml, while in patients with chronic prostatitis without metabolic syndrome (CP without MS) it was slightly higher - 14.4±0.09 pg/ml. A slight increase in the IL-2 level in patients with CP without MS compared to the control group may indicate activation of the cellular component of immunity in conditions of chronic inflammation.

IL-4 plays an important role in the differentiation of T-helpers of the second type (Th2) and stimulates the production of antibodies, which may indicate an adaptive immune response in conditions of chronic inflammation []. In the control group, this indicator was 8.53±0.11 pg/ml, while in patients with chronic prostatitis without

metabolic syndrome (CP without MS) it was significantly higher - 11.7±0.11 pg/m. An increase in the IL-4 level in patients with CP without MS may indicate increased activation of the humoral component of immunity.

TNF-α is a key inflammatory mediator involved in maintaining chronic inflammation and activating immune cells. In the control group, this indicator was 27.4±0.09 pg/ml, while in patients with CP without metabolic syndrome it increased to 32.4±0.25 pg/ml. The difference between the groups was an increase of 1.18 times compared to the control group. This result reflects increased activation of the inflammatory response in patients with chronic prostatitis.

VEGF is a key regulator of vascular permeability and growth of new vessels, which is important for maintaining the inflammatory process and tissue regeneration. In the control group, this indicator was 163.7±0.48 pg/ml, while in patients with CP without MS it was significantly higher - 223.5±1.05 pg/ml. The difference between the groups corresponds to an increase in the VEGF level by 1.37 times compared to the control group. Increased VEGF levels in patients with CP without MS may indicate activation of angiogenesis in response to chronic inflammation in the prostate tissue.

Next, we studied the level of cytokines in a subgroup of men with chronic prostatitis in combination with metabolic syndrome (main group, n=20).

In the control group, the IL-2 level was 13.2±0.17 pg/ml, and in patients with CP with MS it increased to 15.1±0.11 pg/ml, which corresponds to a 1.14-fold increase in the level compared to the control group.

In patients with CP with MS, the level of interleukin-4 (IL-4) was 17.8±0.10 pg/ml, which is 2.09 times higher than in the control group (8.53±0.11 pg/ml) ($p < 0.05$).

The level of tumor necrosis factor-α (TNF-α) in patients with chronic prostatitis with metabolic syndrome (CP with m.s.) was 45.7±0.28 pg/ml, which is 1.67 times higher than in the control group (27.4±0.09 pg/ml) ($p < 0.05$).

When analyzing the level of vascular endothelial growth factor (VEGF), it was found that in patients with chronic prostatitis with metabolic syndrome (CP with m.s.), it was significantly increased by 1.67 times and amounted to 272.6±2.65 pg/ml than in the control group (163.7±0.48 pg/ml) ($p < 0.05$).

Thus, a significant increase in the levels of the studied markers in groups of patients with chronic prostatitis, especially in the main group, is associated with increased inflammation and metabolic disorders. Increased IL-2 indicates activation of the cellular component of immunity and chronic inflammation. Increased IL-4 reflects activation of the humoral component of immunity and cytokine imbalance, which is

more pronounced in patients with metabolic syndrome. Increased TNF-α confirms increased inflammation, since this cytokine is a key mediator of inflammation actively produced by macrophages. Increased VEGF indicates activation of angiogenesis, which is associated with hypoxia of prostate tissues and vascular dysfunction, especially pronounced in patients with metabolic syndrome.

At the next stage of our research, we examined the levels of cytokines IL-2, IL-4, TNF-α and VEGF in the ejaculate of men in the control group and in patients with chronic prostatitis without metabolic syndrome (comparison group, n=25).

The IL-2 level in the control group was 11.4±0.12 pg/ml, and in patients with CP without MS it increased to 13.8±0.11 pg/ml, which is 1.21 times higher than in the control.

The IL-4 level in the control group was 16.2±0.10 pg/ml, and in the CP group without MS it increased to 18.7±0.28 pg/ml, which is 1.15 times higher than in the control.

The level of TNF-α was 42.6±0.53 pg/ml in the control group and increased to 51.3±0.54 pg/ml in patients with CP without MS, which is 1.2 times higher than in the control.

The VEGF level in the control group was 325.8±2.85 pg/ml, and in patients with CP without MS it increased to 397.5±2.40 pg/ml, which is 1.22 times higher than in the control.

Thus, patients with chronic prostatitis without metabolic syndrome show a moderate but statistically significant increase in all studied cytokines compared to the control group. These changes reflect the activation of inflammatory and immune mechanisms, as well as the involvement of angiogenesis in the pathogenesis of chronic prostatitis.

Next, the levels of cytokines IL-2, IL-4, TNF-α and VEGF in the ejaculate of men in the control group and in patients with chronic prostatitis with metabolic syndrome (main group, n = 20) were studied.

The IL-2 level in the control group was 11.4±0.12 pg/ml, while in patients with CP with MS it increased to 17.5±0.11 pg/ml, which is 1.54 times higher than in the control ($p \leq 0.05$).

The IL-4 level in the control group was 16.2±0.10 pg/ml, and in patients with CP with MS it reached 25.4±0.30 pg/ml, which is 1.57 times higher than in the control ($p \leq 0.05$).

The level of TNF-α in the control group was 42.6±0.53 pg/ml, and in patients with CP with MS it increased to 59.4±0.49 pg/ml, which is 1.39 times higher than in the control.

The VEGF level in the control group was 325.8±2.85 pg/ml, while in patients with CP with MS it increased to 452.7±3.03 pg/ml, which corresponds to an increase of 1.39 times compared to the control.

Conclusions: Patients with chronic prostatitis and metabolic syndrome show a significant increase in all studied cytokines in the blood serum and ejaculate compared to the control group. These changes reflect a pronounced cytokine imbalance, as well as the involvement of angiogenesis in the pathogenesis of the disease. The data obtained confirm the significant impact of metabolic syndrome on the enhancement of inflammatory and immune disorders in chronic prostatitis.

LIST OF USED LITERATURE

1. Albosale AH. Association Between Promoter Polymorphisms of IL-1B, IL-4 and IL-6 Genes and a Viral Load Infected Women with Human Papillomavirus // J. Reprod Infertil. - 2021. - No. 22 (2). - PP. 92-102.
2. Abdullaev KU, Yarmukhamedov A, S,. Gaibullaev AA Aripova TU,. Fayzullaeva N.Ya. : Diagnosis and prevalence of immune infertility in men with impaired fertility // Journal of theoretical and clinical medicine. 2024 No. 5 P.56-60
3. Baud D. Sperm Microbiota and Its Impact on Semen Parameters // FrontMicrobiol. - 2019. - No. 10. - P. 234-240.
4. Choi JB Analysis of bacterial community using pyrosequencing in semen from patients with chronic pelvic pain syndrome: a pilot study //Transl Androl Urol. - 2020. - No. 9 (2). - PP. 398-404.
5. Fayzullaeva N.Ya., Yarmukhammedov A.S., Norkulov Zh.O. Immunobiochemical factors in the development of idiopathic infertility in men // Journal of theoretical and practical medicine. 2024, No. 6. P. 80-85
6. Feng C. Correction to: Sperm parameters and anti-Müllerian hormone remain stable with Helicobacter pylori infection: a cross-sectional study // BMC Urol. - 2021. - No. 21 (1). -P. 19-27.
7. Khamdamova M.T., Akramova D. E. Genetic aspects of genital prolapse in women of reproductive age // New day in medicine. Bukhara, 2023. - No. 5 (55). - P. 638-643.
8. Khamdamova M.T., Teshaev Sh.Zh., Hikmatova M.F. Morphological changes of the thymus and spleen in renal failure in rats and correction with pomegranate seed oil // New day in medicine. Bukhara, 2024. - N. 3(65). - P. 167-187.

9. Khamdamova M.T., Khasanova M.T. Various mechanisms of pathogenesis of endometrial hyperplasia in postmenopausal women (literature review) // New day in medicine. Bukhara. 2023. - No. 8 (58). - P. 103-107.
10. Khamdamova M.T., Zhaloldinova M.M., Khamdamov I.B. The state of nitric oxide in blood serum in patients with cutaneous leishmaniasis // New day in medicine. Bukhara, 2023. - No. 5 (55). - P. 638-643.
11. Khamdamova M.T., Zhaloldinova M.M., Khamdamov I.B. The value of ceruloplasmin and copper in blood serum in women wearing copper-containing intrauterine device // New day in medicine. Bukhara, 2023. - No. 6 (56). - P. 2-7.
12. Khamdamova M.T., Akramova D. E. Immediate and long-term results of surgical treatment of genital prolapse in elderly women // New day in medicine. Bukhara, 2025. - N3 (77). - P. 201-206.
13. Khamdamova M. T., Khasanova M.T. Genetic mechanisms of development of endometrial hyperplastic processes in women in menopacteric age // New day in medicine. Bukhara, 2025. - N3 (77). - P. 207-211.
14. Khamdamova M. T., Umidova N. N. Genetic factors of genital endometriosis // New day in medicine. Bukhara, 2025. – N4 (78). - P. 82-87.
15. Ghai V., Subramanian V., Jan H., Loganathan J. et al. Evaluation of clinical practice guidelines (CPG) on the management of female chronic pelvic pain (CPP) using the AGREE II instrument // International Urogynecology Journal. - 2021. - Vol. 32, N. 1. - P. 1-14.
16. Hedger MP. Region-specific immune responses to autoimmune epididymitis in the murine reproductive tract // Cell Tissue Res. - 2020. - No. 381(2). - PP. 351-360.
17. Isaguliants M. Genetic instability and anti-HPV immune response as drivers of infertility associated with HPV infection // Infect Agent Cancer. -2021. - No. 16 (1). - P. 29-39.