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SPECIFIC IMMUNE PROTECTION TO *KLEBSIELLA PNEUMONIAE* AND *ENTEROCOCCUS FAECALIS* IN THE CHRONIC PROSTATITIS SUFFERERS

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Microbiological investigation of samples in the chronic prostatitis (CP) sufferers showed that isolation opportunistic bacteria: *Enterococcus faecalis* and *Klebsiella pneumoniae* from tested samples of expressed prostatic secretions (EPS) are the cause of inflammation and aggravation of prostate in men. Except microbiological culture isolation it was investigated condition immune system. Such parameters of cellular mediated immunity as CD3+; CD4+; CD8+; CD16+; CD19+; PAN (phagocytic activity of neutrophils). Humoral mediated immunity by concentration of immunoglobulin classes: A, G, M in serum as well as determined. Such complex bacteriological and immunological investigation enables us to gain a better understanding of the reason and mechanism of inflammation. It was indicated that parameters of cellular mediated immunity during inflammation decrease, however, IgA concentration increases and concentration of IgM decrease. After specific immune protection parameters of immue system including cellular mediated immunity and humoral immunity normalized. Such detailed investigation of immune system gives an ability to specify total condition the immune system and it`s reserves for specific immune protection of mucosal from such opportunistic bacteria as: *Enterococcus faecalis* and *Klebsiella pneumoniae*.

Key words: phagocytic activity of neutrophils, immunoglobulin classes, Klebsiella pneumoniae, Enterococcus faecalis, immune system.

The deterioration of the ecological situation and environment in the industrial and technological regions significantly affects the cellular and humoral mediated immunity. It decreases the parameters of the immune system, making the body sensitive to bacterial infections and inflammation with bacteria, which is normally opportunistic and living in the body. Such opportunistic bacteria could frequently be a reason for inflammation including chronic bacterial prostatitis (CP). Frequently, the reason for inflammation could be such opportunistic bacteria as: *Staphylococcus aureus, Staphylococcus epidermidis, Staphylococcus saprophyticus, Escherichia coli, Streptococcus pyogenes, Streptococcus agalactiae,Enterococcus faecalis, Enterococcus faecium, Pseudomonas aeruginosa, Klebsiella pneumoniae, Proteus vulgaris, Proteus mirabilis, Proteus morganii, anaerobic bacteria and others* (Arias, 2010; Cai,2011). In this case, it is most important to isolate live bacteria from samples (EPS, urine, sperm) and then after its identification, specify the exact reason for inflammation or aggravation of chronic process. It is very easy to specify the condition of the body's protection against bacterial and viral infection. This is due to the tested immune parameters of the cellular and humoral links of the immune system.

It is of utmost importance to increase the parameters in the immune system, because the phagocytic activity of neutrophils directly capture and digest bacteria

cells, which cause inflammation and aggravation. In order to increase the parameters of the immune system (including the phagocytic activity of neutrophils), recover and normalize the protective functions of body, it`s necessary to use specific immune protection against those bacteria which cause of inflammation or aggravation in the prostate. Without specific immune correction against bacteria (which are reason of inflammation) it will be impossible to recover normal protection of mucosa and the cellular mediated immune system will react only with incomplete phagocytosis. In this case, neutrophil will capture bacteria, but will not be able to digest them and as a result the neutrophils will burste. It is incomplete phagocytosis and it can continue during years and decades. Immunophenotyping of lymphocytes to (CD3+, CD4+ CD8+, CD16+, CD19+, PAN (Phagocytic activity of neutrophils and others) assess the various subpopulations of lymphocytes according to clusters differentiation.

Klebsiella pneumoniae, is Gram-negative facultative-anaerobic opportunistic bacteria from Enterobacteriaceae family, that is normally present in the human colon. At the same time, *Klebsiella pneumoniae*, and *Klebsiella oxytoca* can infect urinary tract and could be reason of inflammation. They have a form of short and thick elliptical bacillus with a size 0,6-6,0 to 0,3-1,0 micrometers. Klebsiella is fixed and does not form spores. Is has an expressed capsule which it resistant to the environment.

Enterococcus faecalis is a Gram-positive facultatively anaerobic, cocci, opportunistic bacteria of the family Streptococcaceae formerly classified in the genus *Streptococcus*. *Enterococcus faecalis* and *Enterococcus faecium* are normal inhabitants of the human intestinal tract that occasionally cause urinary tract infection (Fisher, 2009; Gerald, 1998).

Both bacteria *Enterococcus faecalis*, and *Klebsiella pneumoniae* frequently infect the urinary tract in men. It therefore causes the problem of chronic inflammation in prostate, with periodic aggravations which makes life for sufferers inconvenient (Motrich, 2007; Pavone-Macaluso, 2007).

The aim of the investigation is to determine the connection of chronic inflammation in the prostate with cellular and humoral link of immunity, and to show the advantage from specific immune - protection against both bacteria.

Enterococci are responsible for 12% of all acute prostatitis. Among enterococci - agents of urinary tract inflammation is the dominant fecal enterococci (*Enterococcus faecalis*) and it is cause of problem, because has high resistance to most antibiotics (Skerk, 2009).

Two species of Enterococcus are basic for human intestines and are intestinal symbiotic organisms: *Enterococcus faecalis* (90-95%) and *Enterococcus faecium* (5-10%) which are both opportunistic. Therefore, strong specific immunity against *Enterococcus faecalis* and *Klebsiella pneumoniae*, includes a necessary amount of protective antibodies against both bacteria, which able to strictly regulate their amount in the body (Patterson, 1991).

MATERIALS AND METHODS

Samples of EPS, urine and sperm were taken from sufferers with chronic prostatitis. Bacterial isolation was provided by known microbiological methods that sample cultivation on non selective and selective nutrient medias for bacteria isolation. The research tested samples of EPS, urine, sperm from chronic prostatitis sufferers. To isolate the reason of inflammation or aggravation, complex measurements were used, including microbiological and immunological methods.

Thioglycollate broth for multi-purpose enriched differentiating media was used primarily to differentiate oxygen requirement levels of various organisms. The use of thioglycolate broth permits the growth of anaerobic bacteria. Oxygen levels throughout the media are reduced via reactions with sodium thioglycolate. CLED agar (cystine lactose electrolyte deficient medium) is a valuable non-inhibitory growth medium used in the isolation and differentiation of urinary organisms. Being electrolyte deficient, it prevents the swarming of Proteus species. Cystine promotes the formation of cystine-dependent dwarf colonies. Lactose fermenters produce yellow colonies on CLED agar. Non-lactose fermenters appear blue (Becton Dickinson Company, MD, USA.

To isolate the bacteria, a classical microbiological method was used, where microorganisms on Petri dishes with different solid nutrient medias were cultivated. Differential nutrient media: Pseudosel agar for isolation *Pseudomomas aeruginosa*, Enterococcosel agar for isolation *Enterococcus faecalis*, Endo Agar, is a slightly selective and differential medium for the isolation, cultivation and differentiation of gram-negative microorganisms of *Escherichia coli* and coliform bacteria. Chocolate Agar is an improved medium for use in qualitative procedures for the isolation and cultivation of fastidious microorganisms (Becton Dickinson Compamy, MD, USA) (Murray, 1990; Nieuwkoop, 2008; Taylor, 1998). All cultures of *Enterococcus faecalis* after isolation were deposited in the NRRL Culture Collection, Peoria, IL, USA.

For testing immune system condition, samples of peripheral blood were used. To determine cellular mediated immunity the following parameters were tested: CD3+, CD4+, CD8+, CD16+, CD19+, PAN (Phagocytic activity of neutrophils), E-RFL, E-RFN. To determine humoral mediated immunity, the concentration of immune globuln classes A,G,M were tested. It was tested using the Manchini method of radial immune diffusion in agar gel. For this purpose the test-kits of Microgen Company (Nijniy Novgorod, Russian Federation) were used. The assessment for the level of basic subpopulations of lymphocytes in blood was done by method of direct immunefluorescence to monoclonal antibodies for differentiation of antigens CD3+ (T-lymphocytes), CD4+ (T-helpers), CD8+ (T-supressors), CD16+ (NK-killers), CD19+ (B-lymphocytes). All tests for CD was done using test kits produced in ("Sorbent" Company, Russian Federation). PAN (Phagocytic activity of neutrophils) tested using load with boiled yeast culture *Saccharomyces cerevisiae*. Lymphocytes loaded with bacterial and viral antigens and immunomodulating remedy were tested for

using phenomena of rosette forming lymphocytes E-RFL and rosette forming neutrophils E-RFN (Frank, 2011; Rodney, 2002).

The parameters of immune system in chronic prostatitis sufferers were tested before and after specific immune protection with immunogen conjugate were used for desensitization of the body against *Enterococcus faecalis* and *Klebsiella pneumoniae*. During this time, there were three times the controlled bacterial growth of *Enterococcus faecalis* and *Klebsiella pneumoniae*.

Statistical deviation and significance were evaluated by the Student`s t-test with P – value: P<0.1; P; <0.05; P; <0.01. The Spearman rank correlation coefficient was also calculated for the tested parameters of cellular and humoral mediated immunity in the group without specific immune protection and after specific immune protection. Each test was repeated three times to confirm the exact result. For both tested groups of chronic prostatitis sufferers dispersion analysis (ANOVA) done as well as. The dispersion analysis (ANOVA) based on Fisher`s test (unifactorial model) was applied, where the F-criterion determined whether relevant samples belong to one from general aggregate and then possible to pool them or not.

RESULTS AND DISCUSSION

The primary growth of the bacterial culture of *Enterococcus faecium* and *Klebsiella pneumoniae* was totally full on Petri dishes with nutrient media. Bacterial cultures isolated from samples of chronic prostatitis sufferers were deposited in the NRRL Culture Collection. *Enterococcus faecalis* NRRL B-59255; NRRL B-59256; NRRL B-59257; NRRL B-59258; NRRL B-59259; NRRL B-59262; NRRL B-59265; *Enterococcus faecium* NRRL B-59223; NRRL B-59369. *Klebsiella pneumoniae subsp. pneumoniae* NRRL B-59261.

Bacterial culture were primary cultivated in Thioglycollate broth during 24-48 hours, then samples were inoculated in differential selective media such as Endo Agar, Cled Agar, Preudosel Agar, Staphylococco Agar, Streptococco Agar, Corynebac Agar and others. Fig. 1 presented primary growth from samples. Fig.2 presented growth from Enterococcosel agar which is selective and differential for the *Enterococcus faecalis*. Fig.3 presented growth on Endo Agar which is selective for group of bacteria Enterobacteriaceae. Fig.4 presented primary isolation from EPS samples the *Klebsiella pneumoniae* colonies, grows in Endo Agar.

Table 1 showed parameters of cellular mediated immunity in the chronic prostatitis sufferers in two groups, before and after specific immune protection. Table 2 showed parameters of cellular mediated immunity in the CP sufferers including load tests with antigens and remedies without and after specific immune protection. Table 3, showed humoral mediated immunity in the CP sufferers before and after specific immune protection. Table 4 showed calculated results of dispersion analysis (ANOVA) changes of some parameters of cellular mediated immunity in CP sufferers before and after specific immune protection against *Klebsiella pneumoniae* and *Enterococcus faecalis*.
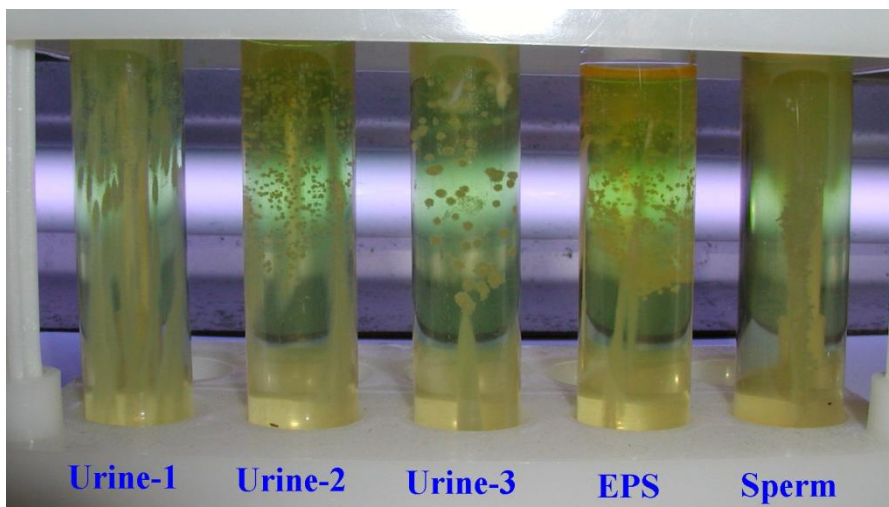
Fig.1 Primary bacterial growth in Thioglycollate broth.
1 – first portion of urine; 2- middle portion of urine; 3- end portion of urine;
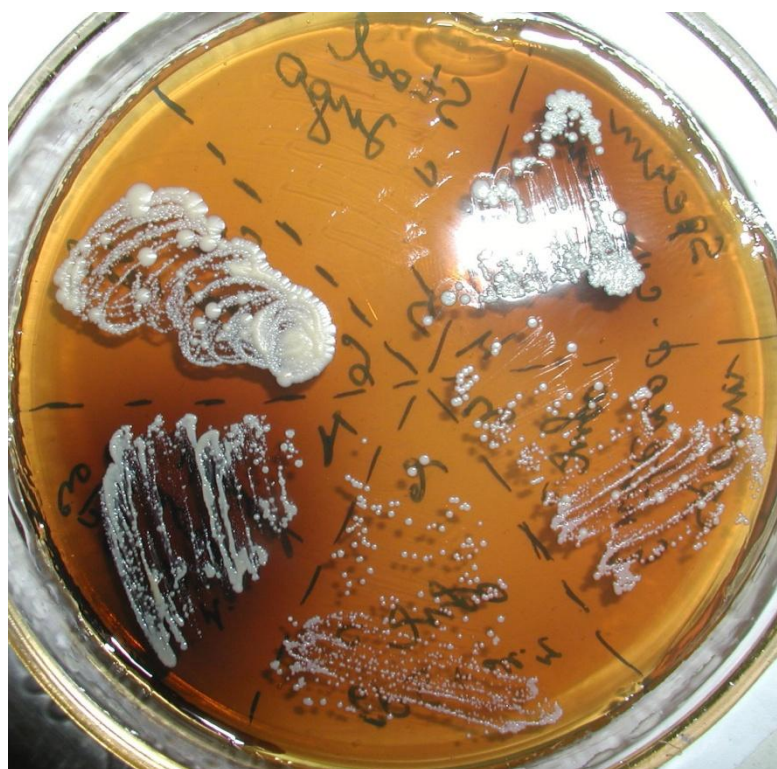4 – EPS (expressed psrostatic secretions); 5 – sperm.

Fig.2 Primary isolation from EPS samples the *Enterococcus faecalis* on selective
Enterococcosel Agar.

Fig.3 Bacterial growth from EPS samples on selective Endo Agar the specimens inoculated from Thioglycollate broth.
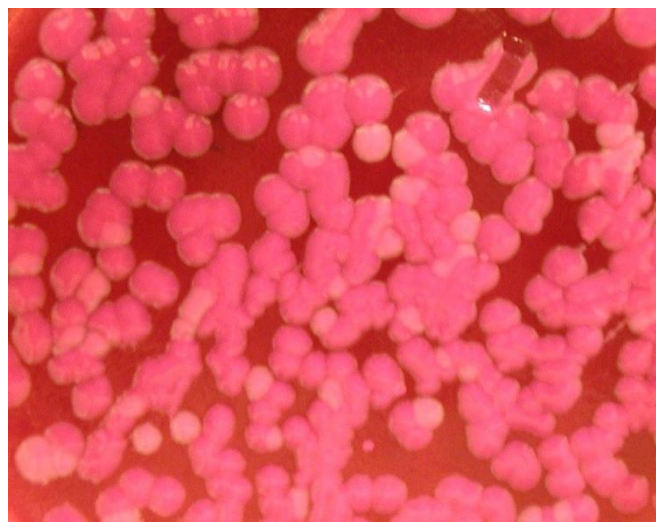

Fig.4 Primary isolation from EPS samples the *Klebsiella pneumoniae* colonies, grows in Endo Agar.

Table 1. **Cellular mediated immunity in the CP sufferers before and after specific Immune protection**

Tested parameter of immune system	Measurement units	Without Immune protection	After specific Immune protection	Normal range
WBT (white blood cells)	10^9/L	3.1±0.2	5.1±0.2	4.0-8.0
Lymphocytes	%	33±2.25	45±2.7	19-37
Lymphocytes	10^9/L	1.7±0.2	2.3±0.3	1.5-3.0
CD3+(T- Lymphocytes)	%	60±5.57	62±2.05	55-70
CD3+(T- Lymphocytes)	10^9/L	*0.83± 0.05	*1.42± 0.03	0.8-2.0
CD4+(T- Herpes)	%	34±2.0	50±5.03	40-60
CD4+(T- Herpes)	10^9/L	*0.47± 0.02	*1.15± 0.05	0.4-1.2
CD8+(T- Supressors)	%	26±2.71	12±2.05	10-20
CD8+(T- Supressors	10^9/L	*0.36± 0.05	*0.27± 0.03	0.1-0.7
Ratio CD4+/ CD8+	-	1.3±0.27	4.2±0.20	2.0-4.0
CD19+(B- Lymphocytes)	%	5±0.17	16±0.27	6-15
CD19+(B- Lymphocytes)	10^9/L	**0.07± 0.01	*0.36± 0.05	0.1-0.38
CD16+ Cytotoxic activity (T-killer EK cells)	%	9±0.47	8±0.41	10-20
CD16+ Cytotoxic activity (T-killer EK cells)	10^9/L	**0.12± 0.008	**0.18± 0.01	0.15-0.6
Phagocytic activity of neutrophils	%	76±2.71	86±2.05	40-80
Phagocytic activity of neutrophils	10^9/L	**1.3±0.05	2.4±0.08	1.6-4.0

Standard deviation was calculated, statistical significance of difference was evaluated by the Student's t-test. Note: P-value * P≤0.05; ** P≤0.01.

Table 2. **Cellular mediated immunity in the CP sufferers including load tests with antigens and remedies without and after specific immune protection**

Tested parameter of immune system	Measurement units	Without Immune protection	After specific Immune eprotection	Normal range
*E-RFN (E- rosette forming neutrophils)	%	66±2.68	68±2.71	50-70
**E-RFL (T- active Lymphocites)	%	80±4.21	82±1.46	65-85
E-RFL/E-RFN (Index of load)	ratio	*1.2±0.03	*1.2±0.05	1-2
***E-RFC load with antigens:				
E-RFL (T- lymphocites "CONTROL")	%	78±2.71	82±2.05	65-85
Herpes virus antigen	%	64±3.01	66±4.11	65-85
Inhibition of Herpes virus antigen	%	16±0.87	16±0.74	0-8
Antigen from retina	%	68±4.77	72±7.05	65-85
Inhibition of antigen from retina	%	12±1.25	10±0.5	0-8
Tuberculine antigen	%	76±8.22	80±4.11	65-85
Inhibition of Tuberculine antigen	%	4±0.55	2±0.35	0-8
E-RFC load with remedies:				
Interferon (immunestimulator)	%	67±6.16	82±6.80	65-85
Inhibition of Interferon	%	8±0.87	3±0.74	0-8
Amixin (immunestimulator)	%	43±8.9	76±5,43	65-85
Inhibition of amixine	%	10±1.12	6±0.85	0-8

Standard deviation was calculated, statistical significance of difference was evaluated by the Student's t-test. P-value * P≤0.05. *E-RFN – Rosette forming Neutrophils. **E-RFL – Rosette forming Lymphocites. ***E-RFC – Rosette forming cells (same EAC - rosette forming cell or erythrocyte- amboceptor-complement rosettes.

Table 3. **Humoral mediated immunity in the CP sufferers before and after specific immune protection**

Tested parameters	Measurement units	Without Immune protection	After specific Immune protection	Normal range
Immuneglobuline A	IgA (g/L)	0.87±0.22	*2.66±0.1	1.2-2.0
Immuneglobuline G	IgG (g/L)	8.82±3.08	11.43±2.19	9.0-18.0
Immuneglobuline M	IgM (g/L)	*0.88±0.1	*0.81±0.1	0.9-1.2

Standard deviation was calculated, statistical significance of difference was evaluated by the Student's t-test. P-value * P≤0.1.

Table 4. **Dispersion analysis (ANOVA) represents changes of some parameters of cellular mediated immunity in CP sufferers before and after specific immune protection against *Klebsiella pneumoniae* and *Enterococcus faecalis*.**

Tested parameter	The group before specific immune protection			The group after specific immune protection		
	F	P	R_s	F	P	R_s
WBT/ Lymphocytes	15.89	0.004	0.51	903.08	< 0.0001	0.75
$CD3^+$ / $CD4^+$	61.47	< 0.0001	**0.84**	272.35	0.001	**0.98**
$CD3^+$ / $CD8^+$	160.79	< 0.0001	**0.47**	6150.4	< 0.0001	-0.27
$CD3^+$ / $CD16^+$	386.65	< 0.0001	**0.55**	10414.2	< 0.0001	-0.38
$CD3^+$ / $CD19^+$	443.7	< 0.0001	**0.87**	4485.33	< 0.0001	**0.37**
$CD4^+$ / $CD8^+$	43.75	< 0.0001	-0.65	945.11	< 0.0001	-0.47
$CD4^+$ / $CD16^+$	444.97	< 0.0001	-0.38	1158.08	< 0.0001	**0.95**
$CD4^+$ / $CD19^+$	696.96	< 0.0001	-0.65	753.76	< 0.0001	**0.76**
$CD8^+$ / $CD16^+$	410.89	< 0.0001	**0.94**	23.53	0.001	**0.73**
$CD8^+$ / $CD19^+$	980.1	< 0.0001	-0.54	23.53	0.001	**0.96**
$CD16^+$/$CD19^+$	54.23	< 0.0001	**0.59**	94.12	< 0.0001	-0.47
*PAN /$CD3^+$	106.91	< 0.0001	**0.47**	690.78	< 0.0001	**0.94**

PAN / CD4+	1023.47	< 0.0001	**0.85**	2112.27	< 0.0001	-0.27
PAN / CD8+	1880.63	< 0.0001	**0.84**	13395.6	< 0.0001	-0.06
PAN / CD16+	3605.37	< 0.0001	**0.41**	10036.2	< 0.0001	0.03
PAN / CD19+	4273.72	< 0.0001	-0.60	8073.6	< 0.0001	**0.94**
Immuno globulines A/G	432.27	< 0.0001	-0.09	1503.17	< 0.0001	**0.88**
Immuno globulines A/M	2.71	0.138	-0.44	422.73	< 0.0001	0.18
Immuno globulines G/M	616.45	< 0.0001	**0.48**	2235.26	< 0.0001	-0.33

*PAN - Phagocytic activity of neutrophils.

IgA antibodies not only function in external secretions, but also exert their antimicrobial properties within the epithelial cell during transport across the epithelium. Passive mucosal delivery of monoclonal IgA molecules neutralizes pathogens responsible for aggravation and/or the prostate infections. Mucosal and systemic immunity can be achieved by protection with administered specific immune conjugate which protect the prostate from infection attacks and aggravations (Corthésy, 1999). IgA is the primary immunoglobulin responsible for protecting these mucosal surfaces from infections. While those who have IgA deficiency lack IgA, they do produce the other immunoglobulins found in the body. Both IgA and IgM are produced at normal levels in the mucosa. IgM is a basic antibody that is produced by B-cells. Two biological properties of IgM make it useful in the diagnosis of infectious diseases. Demonstrating IgM antibodies in a patient's serum indicates recent infection. IgM is not versatile as IgG, but it is of vital importance in complement activation and agglutination.

IgM is predominantly found in the lymph fluid and the blood and is a very effective neutralizing agent in the early stages of disease. Elevated levels can be a sign of recent infection or exposure to antigen.

When an antigen makes contact for the first time with cells of the humoral immune system, B-lymphocytes that are producers of specific immunoglobulins against that antigen multiply and in days synthesize their antibodies. This is called primary response. Some of these specific B-lymphocytes remain in the circulation for a long time, sometimes during the entire life of the individual, and they become the memory cells of the immune system. When the body is exposed in the future to the same antigen the production of antibodies will be faster and more intense since the immune system is already prepared to react against that antigen. This is immune memory, called the secondary response for antigen. In case of bacterial and viral infection the immune attack is made by the cellular

immune system, mediated by T and NK (natural killers) lymphocytes and neutrophyls that destroy specific cells (bacteria, fungi, viruses). The lymphocytes and neutophils that participate in the cellular immune response are the T lymphocytes. T lymphocytes differentiate into three main types: cytotoxic T lymphocytes (cytotoxic T-cell), helper T-lymphocytes (helper cell) and suppressor T- lymphocytes. The cytotoxic cells are the effectors of the system, i.e., they directly attack other cells recognized as foreign (for example, fungi cells, cells infected by virus, neoplastic cells, graft cells, etc.). The T-helper cells and the suppressor T-lymphocytes act as regulators of the system releasing substances that respectively stimulate and inhibit the immune action of T- and B-lymphocytes. After the primary immune response memory T- lymphocytes also remain in the circulation to provide faster and more effective reaction in case of future infections.

Phagocytosis and destruction of engulfed bacteria involves the following sequence of events: delivery of phagocytic cells to the site of infection; phagocytic adherence to the target; ingestion or engulfment of the target particle; phagolysosome formation; intracellular killing; intracellular digestion (Kenneth, 2007).

The inflammatory process in prostate begins with a decrease in cellular and humoral mediated immunity, and leads phagocytes to work in an idle and unproductive mode. Phagocytes are swallowing pathogens, but cannot digest them. Therefore, the inflammatory process in prostate continues indefinitely up to the moment when will start to work well phagocytic activity of neutrophils, which is achieved by the specific immune correction.

In the phase of aggravation (the control group without specific immune correction)
indicated low concentration of immunolobuline A and G, beween CD3$^+$ and CD8$^+$ correlation is direct, weak, but higly reliable connections. There is moderate correlation between CD3$^+$ and CD16$^+$, a high correlation between CD3$^+$ and CD19$^+$, and a very high correlation for CD8$^+$ and CD16$^+$. There is moderate correlation between CD16$^+$ and CD19$^+$, a low correlation between PAN and CD3$^+$, a high correlation between PAN, CD4$^+$ and CD8$^+$ and a direct, week, but higly reliable connection between PAN and CD16$^+$. For humoral mediated immunity there is weak correlation, but with higly reliable connection.

It was determined that in the phase of remission the inflammatory process in the prostate increased T-helpers and phagocytic activity of neutrophils. Beween CD3$^+$ and CD4$^+$ correlation is very high. Between CD3$^+$ and CD19$^+$ there is a direct, weak, but higly reliable connection and a very high correlation between CD4$^+$ and CD16$^+$. The correlation between CD4$^+$ and CD19$^+$ is high, while the correlation between CD8$^+$ and CD16$^+$ is also high. CD8$^+$ and CD19$^+$ have a very high correlation, and PAN and CD3$^+$ also have a very high correlation. In addition,

there is a very high correlation between PAN and CD19[+]. Immino globulin A and G also have a high correlation.

In both investigated groups, there exist a high correction between of CD3[+] and CD4[+]. In the group without specific protection, the correction is high 0.84. In the group after specific protection the correction is very high 0.98.

After specific immune protection, the cellular and humoral mediated immunity parameters of the chronic prostatitis sufferers were tested. The results showed a significant increased level of protective immunoglobulins, which are responsible for antibacterial immune protection of the mucosal. There is an increased level of neutrophils phagocytic activity, indicating that phagocytes are ready for complete phagocytosis and bacteria digest.

CONCLUSIONS

Specific protection against *Enterococcus faecalis, Enterococcus faecium, Klebsiella pneumoniae*, allows for immune system of chronic prostatitis sufferers to strongly control the prostate from inflammation and to protect from bacterial attacks which is most advantageous for sufferers.

ACKNOWLEDGEMENTS

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СПЕЦИФИЧЕСКАЯ ИММУНОЗАЩИТА К *KLEBSIELLA PNEUMONIAE* И *ENTEROCOCCUS FAECALIS* У СТРАДАЮЩИХ ХРОНИЧЕСКИМ ПРОСТАТИТОМ.

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Микробиологическое исследование проб у страдающих хроническим простатитом (ХП) показало, что выделяемые *Enterococcus faecalis* и *Klebsiella pneumoniae* из секрета простаты являются причиной воспалений и обострений у мужчин. Кроме микробиологического выделения культуры было исследовано состояние и параметры иммунной системы. Исследовались такие параметры клеточного иммунитета, как: CD3+, CD4+, CD8+, CD16+, CD19+; фагоцитарная активность нейтрофилов. Исследовались концентрации таких классов иммуноглобулинов, как: A, G, M в сыворотке крови. Такой комплекс бактериологических и иммунологических исследований позволил лучше понять причины и механизмы воспаления. Было отмечено, что показатели клеточного иммунитета при этом снижаются при увеличении концентрации IgA, однако при этом концентрация IgM снижается. После специфической иммунозащиты показатели клеточного и гуморального звена иммунной системы нормализуются. Такое детальное исследование иммунной системы дает возможность определить общее состояние иммунной системы и ее резервные возможности для специфической иммунозащиты слизистых оболочек от таких оппортунистических бактарий как: *Enterococcus faecalis* и *Klebsiella pneumoniae*.

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СПЕЦІФИЧНИЙ ІМУНОЗАХИСТ ДО *KLEBSIELLA PNEUMONIAE* ТА *ENTEROCOCCUS FAECALIS* У СТРАЖДАЮЧИХ ХРОНИЧНІМ ПРОСТАТИТОМ.

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Мікробіологічне дослідження зразків у страждаючих хронічним простатитом (ХП) показали, що виділяються *Enterococcus faecalis* і *Klebsiella pneumoniae* з секрету простати є причиною запалень і загострень у чоловіків. Крім мікробіологічного виділення культури було досліджено стан та параметри імунної системи. Досліджувалися такі параметри клітинного імунітету, як: CD3 +, CD4 +, CD8 +, CD16 +, CD19 +; ФАН (фагоцитарна активність нейтрофілів). Досліджувалися концентрації таких класів імуноглобулінів, як: A, G, M у сироватці крові. Такий комплекс бактеріологічних та імунологічних досліджень дозволив краще зрозуміти причини і механізми запалення. Було відзначено, що показники клітинного імунітету при цьому знижуються при збільшенні концентрації IgA, однак при цьому концентрація IgM знижується. Після специфічного імунозахисту показники клітинного та гуморального ланки імунної системи нормалізуються. Таке детальне дослідження імунної системи дає можливість визначити загальний стан імунної системи та її резервні можливості для специфічного імунозахисту слизових оболонок від таких опортуністичних бактарій як: *Enterococcus faecalis* і *Klebsiella pneumoniae*.

Ключові слова: фагоцитарна активність нейтрофілів, класи імуноглобулінів, *Klebsiella pneumoniae*, *Enterococcus faecalis*, імунна система.