Pharmacological body-wide and immunotropic effects of galavit in the treatment of patients with purulent-inflammatory diseases of various origins and allergization

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Abstract

Introduction: Patients with purulent-inflammatory diseases (PID), especially those with allergization, manifest chronicity, low treatment efficiency, and the risk of relapses; the above actualizes the search for innovative treatment technologies. The aim of the study: The aim of the study was to specify pharmacological (body-wide) and specific (immunotropic) effects of the immunomodulator galavit additionally prescribed in the context of basic treatment for patients with deep pyoderma, chronic pyelonephritis, adnexitis, purulent infection of soft tissues, complicated by atopic dermatitis.

Materials and Methods: The study included 331 patients with PID and 30 healthy volunteers of the same age and gender who were examined prior and following the basic treatment of each nosoform. The diverse clinical and laboratory examination options were applied to investigate 17 clinical, 7 hematological, 16 immunological, 13 metabolic parameters that were grouped into clinical, hematological, immunological and metabolic syndromes, and divided into links – cellular, humoral, phagocytic, cytokine, metabolic and antioxidant. The results were statistically processed using variational parametric and nonparametric techniques; this allowed calculating the coefficient of diagnostic value, diagnostic formulas of disorders and targets of galavit under various conditions.

Results and Discussion: Targeted pharmacological therapy with galavit reliably eliminates clinical, hemato-immuno-metabolic disorders in patients with PID, though dependently on the nosoform of the disease.

Conclusion: It was demonstrated that the correction of clinical and laboratory disorders depend on PID pathogenesis and allergization; the laboratory mechanism of therapeutic effects was specified.
Introduction

Treatment of patients with purulent-inflammatory diseases (PID) of soft tissues is critical due to increased frequency of complicated pathology, developing patients’ disability, insufficient knowledge of the immuno-metabolic genesis of the infection, and a limited set of approved basic therapeutic options.

The available data allow reporting about differentiated pharmacological (body-wide) and profile (immunotropic and metabolic) properties of immunomodulators (Calogero et al. 2017; Bhargava et al. 2017; Bollestad et al. 2018; Dellis and Papatsoris 2018; Zhang et al. 2018; Desai and Brinton 2019; Falconi-McCahill 2019; Huang et al. 2019; Zemskov et al. 2019; Bratchikov et al. 2020; Pokrovskaya et al. 2020). The laboratory and clinical examination findings of patients with various types of purulent-inflammatory diseases were used to detect these properties; PID included deep pyoderma, exacerbation of chronic pyelonephritis, chronic adnexitis, purulent infection of soft tissues, chronic cystitis; part of nosoforms was aggravated by atopic dermatitis. All patients were examined prior and following the basic treatment, prior and following the basic treatment combined with the immunomodulator galavit. Clinical, bacteriological, hematological, immunological and metabolic parameters were evaluated according to generally accepted principles (Genest 2017; Ditkoff et al. 2018; Daniels et al. 2019; Faught and Reyes 2019; Forti et al. 2019; Germano et al. 2019; Goktas et al. 2019).

The number of patients with PID of soft tissues does not decrease but tends to increase. There is an advancing growth in the resistance of strains of gram-negative and gram-positive flora to many antibacterial drugs. A number of new highly effective antibacterial drugs have appeared. There is a concept of choice of therapy for PID of soft tissues considering the risk factors of multi-drug-resistant flora. The proportion of working-age patients with diseases of this profile is 75%. The average period of incapacity for work is from 13.6 to 23.8 days (Tokmakov 2009). Unambiguously applied pharmacokinetic properties, i.e. creation and maintenance of sufficient antimicrobial concentrations in the focus of infection in the dosing interval, are typical of the vast majority of antibiotics recommended for therapy. The occurrence and development of PID to a large extent depend on the body reactivity and its mechanisms to resist microbial aggression. Despite complex measures in which rational antibiotic therapy has the key role, it is necessary to search for a systematic approach to evaluate immunological disorders in the context of PID, which can significantly improve patients’ clinical outcomes.

The aim of research was to study pharmacological (body-wide) and profile (immunotropic) effects of complex treatment (basic treatment+galavit) for 8 nosoforms of PID (patients of group 1), and compare it to the effect provided by conventional drugs (patients of group 2) 2-3 weeks after complex therapy.
Materials and Methods

Group

The study included 331 patients, aged 27-63, receiving basic treatment (BTr) or basic treatment+galavit (BTr+G). Of these, there were 20 and 21 patients suffering from deep pyoderma, receiving basic treatment or basic treatment+galavit, respectively, 22 and 20 patients suffering from chronic pyelonephritis, receiving various treatment options; 20 and 23 patients suffering from chronic adenitis; 20 and 21 patients suffering from the purulent infection of soft tissues; 20 and 21 patients suffering from chronic cystitis; 21 and 24 patients suffering from the purulent infection of soft tissues+atopic dermatitis; 19 and 19 patients suffering from exacerbation of chronic pyelonephritis+atopic dermatitis; 21 and 19 patients suffering from exacerbation of chronic cystitis+atopic dermatitis. The immunomodulator galavit having anti-inflammatory, antioxidant-metabolic, hepatoprotective effects was used for immunomodulation; it corrected secondary immunological deficiency in T-B-links in acute and chronic bacterial infections, autoimmune complications (Zemskov et al. 2013). The drug was administered intramuscularly (5 injections of 100 mg daily).

Patients in the experimental and control groups received identical treatment within the same nosology; the only difference was the presence or absence of the study drug.

The study was approved by the Ethics Committee (Minutes No. 2a dated 07.09.2022).

Study design

General principles for PID treatment consisted of the following: antibacterial drugs administered considering sensitivity of the isolated pathogenic flora – ceftriaxone, doxycycline, hydrochloride, gentamicin, cefotetan, nolinc, nitrofurans (furagin), cefoxitin, clindamycin, quinolone; anti-inflammatory drugs – metronidazole, salicylates, pyrazolinedines; antibiosis – cetirizine, erius, loratadine, sodium thiosulfate intravenously; antifungal drugs – mycoflucan; eubiotics to prevent candidiasis and dysbiosis (lactobacillus acidophilus, bifidobacteriabifidum+lysozyme); and multivitamin complexes (B, C, E). Crystalloid and colloid solutions, protein-enriched nutrition, and physiotherapy were applied to correct disturbed homeostasis and water-electrolyte imbalance in patients. Local therapy included solutions of aniline dyes 2 times a day and ointments containing antibiotics; it was administered for patients individually considering sensitivity of pathogenic microflora in the lesions. In patients with PID complicated by atopic dermatitis, the treatment complex was additionally supplemented by combinations of the first-second-generation antihistamines, infusion therapy, enteral sorbents, antispasmodics, choleretic, hepatoprotective drugs, and eubiotics.

Common clinical investigation techniques suggested presence of inflammatory foci, fever, intoxication, lymphadenopathy, and bacterial contamination of diagnostic material in patients. Specific manifestations included edema, hyperemia, soreness, itching, oozing lesions and crusts, renal colic, pelvic pain, leucorrhoea, adnexal hypertrophy, leukocyte-, erythrocyturia, etc. Using conventional bacteriological techniques, pathognomonic microflora was cultured and identified out of the diagnostic material (urine, secretions from skin foci, genitals), with the following detection of antibiotic sensitivity of pathogens.

Laboratory tests

- Routine hematological tests to detect leukocytes, lymphocytes, neutrophils, eosinophils, basophils, monocytes, ESR.
- Immunological tests to detect T-B cells (T, B), T-helpers (Th), T-cytotoxic lymphocytes (Tcylt), natural killers (NK), immune globulins (Ig), absorption and metabolic ability of phagocytes – phagocytic index and number (Phi, PhN), spontaneous and activated NBT-tests (NBTrsp, NBTac), anti- and pro-inflammatory cytokines (IL4, IL6, IL-8, TNF).
- Metabolic tests include tests of free-radical oxidation of high molecular weight substrates (FRO) - Schiff base (SchB), malondialdehyde (MDA), bityroine crosslinks (BC), diene conjugates (DC), ketodies (KD); tests to detect parameters of the antioxidant system – vitamin E (VE), superoxide dismutase (SOD), protein thiols (PT), total thiols (TT), non-protein thiols (nonPT), blood antioxidant activity (AOA).
- Hematological parameters of inflammation were determined by conventional tests using HAVIOS Beckman Coulter flow cytometry, CYTO-STAT tetra CHROM monoclonal antibodies, Chospitec biochemical analyzer, spectrophotometric, turbidimetric, enzyme immunoassay techniques, and the kits of the Protein Contour company; clones and subclones of lymphocytes; pro- and anti-inflammatory cytokines; absorption and oxygen-producing activity of peripheral phagocytes; the content of circulating immune complexes (CIC), middle molecules (MM), serum immune globulins of the main three classes. Ultraviolet spectrophotometry, fluorescence, reactions with 2-thiobarbituric acid, etc., allowed characterising FRO and antioxidant defence (AOD) parameters.

Statistical analysis

Clinical and laboratory findings of patients were statistically processed using parametric (Student’s) and nonparametric (Wilcoxon-Mann-Whitney) criteria, depending on the normal distribution of values. The coefficient of diagnostic value was used to determine typical formulas of disorders and targets of treatment options (Jung et al. 2018; Keagy 2018; Falconi-McCahill 2019; Germano 2019; Huang et al. 2019; Berezhnova et al. 2020).

Body-wide effects of galavit were evaluated by their impact on grouped parameters: hematological (H), immunological (I), metabolic (M), bacteriological (B), clinical (C); the ranks of differences from the normal level were calculated considering the examination findings of healthy individuals (rank 1 – maximum rank, changes > 66%; rank 2 – medium, changes 33-66%; rank 3 – minimal, changes<33%).

Method of statistical analysis of grouped clinical and laboratory parameters of patients includes the following steps:

- to group, the average % of the modified parameters is calculated depending on the examination methods
Results

The results obtained are given in Tables 1–4.

The acute period of uncomplicated PID was manifested by typical clinical symptoms; general irritation of the erythroid lineage with a deficiency of lymphocytes; T-link immunity suppression, B-link immunity activation, imbalance (of pro- and anti-inflammatory cytokines), suppression of phagocytosis, stimulation of lipid peroxidation in the context of antioxidant protection inhibition.

In case of disease allergization, patients additionally manifested: clinical symptoms of atopic dermatitis; stimulation of the lymphocytes, eosinophils, basophils; excess of T-cytotoxic suppressors, imbalance in the concentration of serum immune globulins classes M and G, absorptive and metabolic capacity of neutrophils.

Discussion

The effectiveness of the body-wide effect of the basic treatment complex with the modulator galavit is demonstrated in Table 1, which analyses variations of the grouped hematological (D), immunological (I), metabolic (M), bacteriological (B), clinical (C) test findings from the basic treatment level depending on the PID nosoforms.

As presented in Table 1, galavit realized a positive body-wide final effect on the grouped clinical and laboratory parameters of patients from the level of basic treatment depending on the nosological forms of purulent-inflammatory diseases. Thus, in deep pyoderma, galavit resulted in a significant normalization of all the studied parameters (hemato-immuno-metabolic-bacteriological-clinical). The dynamics of clinical findings was ineffective in case of exacerbation of chronic adenitis, purulent infections of soft tissues; the dynamics of metabolic findings was ineffective in case of exacerbation of chronic cystitis; the dynamics of metabolic-clinical findings was ineffective in purulent infection of soft tissues+atopic dermatitis; the dynamics of hematoo-immuno-metabolic-findings was ineffective in case of exacerbation of chronic pyelonephritis+atopic dermatitis, exacerbation of chronic cystitis+atopic dermatitis. There was a decreased final activity of immunotherapy under allergic diseases. Depending on the declining rating of the overall efficacy of immunotherapy with galavit, PID

Table 1. Body-wide effect of galavit in patients with PID

<table>
<thead>
<tr>
<th>Disorovers</th>
<th>Differences of the basic treatment+galavit (BTr+G) complex from the basic treatment (BTr) complex; grouped parameters, %</th>
<th>Level of differences, ranks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep pyoderma</td>
<td>H: 36/2* I: 49/2* M: 28/3* B: 37/2* C: 33/2* Mean difference value %//2 ranked</td>
<td>II</td>
</tr>
<tr>
<td>Exacerbation of chronic pyelonephritis</td>
<td>H: 19/3 I: 55/2* M: 16/3 B: 33/2* C: 5/3 Mean difference value %//2 ranked</td>
<td>IV</td>
</tr>
<tr>
<td>Exacerbation of chronic adenitis</td>
<td>H: 22/3* I: 35/2* M: 20/2* B: 42/2* C: 7/3 Mean difference value %//2 ranked</td>
<td>III</td>
</tr>
<tr>
<td>Purulent infection of soft tissues</td>
<td>H: 40/2* I: 45/2* M: 25/2* B: 79/1* C: 10/3 Mean difference value %//2 ranked</td>
<td>I</td>
</tr>
<tr>
<td>Exacerbation of chronic cystitis</td>
<td>H: 33/2* I: 23/3* M: 10/3 B: 38/2* C: 37/2* Mean difference value %//2 ranked</td>
<td>III</td>
</tr>
<tr>
<td>Purulent infection of soft tissues+atopic dermatitis</td>
<td>H: 20/3* I: 20/3* M: 13/3 B: 38/2* C: 9/3 Mean difference value %//2 ranked</td>
<td>V</td>
</tr>
<tr>
<td>Exacerbation of chronic pyelonephritis+atopic dermatitis</td>
<td>H: 11/3 I: 16/3 M: 10/3 B: 36/2* C: 34/2* Mean difference value %//2 ranked</td>
<td>IV</td>
</tr>
<tr>
<td>Exacerbation of chronic cystitis+atopic dermatitis</td>
<td>H: 15/3 I: 19/3 M: 8/3 B: 30/3* C: 27/3* Mean difference value %//2 ranked</td>
<td>VI</td>
</tr>
</tbody>
</table>

Note: BTr – basic treatment, G – galavit, Σ – overall value, * – significant differences from BTr at P<0.05; 1, 2, 3 – value differences from BTr, ranks; I-VI – decreasing overall levels of BTr+G efficiency.
variants were distributed according to the following ranking: purulent infections of soft tissues – deep pyoderma – (exacerbation of chronic adenitis, exacerbation of chronic cystitis) – (exacerbation of chronic pyelonephritis, exacerbation of chronic pyelonephritis + atopic dermatitis) – purulent infection of soft tissues + atopic dermatitis – exacerbation of chronic cystitis.

Detailed distribution of the galavit effect in ranks on grouped hematological, immunological, metabolic, bacteriological and clinical parameters in patients was as follows: deep pyoderma, exacerbation of chronic pyelonephritis, exacerbation of chronic adenitis, purulent infections of soft tissues, purulent infection of soft tissues + atopic dermatitis, exacerbation of chronic pyelonephritis + atopic dermatitis, exacerbation of chronic cystitis, exacerbation of chronic cystitis + atopic dermatitis – 2:2:3:2:2, 3:2:3:2:3, 3:2:2:2:1:3, 2:2:2:1:3, 2:3:3:2:2, 3:3:3:2:3, 3:3:3:2:2, 3:3:3:3:3; it seemed to be almost exclusive for each nosological form of PID. For example, in patients with purulent infections of soft tissues, the "proper" – independent on conventional treatment – effect of galavit on hematological, immunological, and metabolic parameters was moderately pronounced (33-66%), on bacteriological – extreme (>66%), and on clinical – low (<33%). Notably, in exacerbation of chronic cystitis + atopic dermatitis, the variations of all clinical and laboratory parameters were of the minimum rank 3 under the influence of the same modulator.

Of particular interest is the evaluation of detailed changes in parameters grouped by immunity links with a quantitative measurement of the ranks of these variations from the norm before treatment, after the basic treatment and combined basic treatment + galavit in patients with monososforms of purulent-inflammatory diseases without complications (see Table 2).

Quantitative rank analysis of the differences in the values of the key clinical and laboratory parameters from the normative values demonstrated a developing unified pattern in patients suffering from deep pyoderma, chronic pyelonephritis, purulent infections of soft tissues, chronic cystitis – a certain positive effect of the baseline treatment from the baseline values and even more pronounced when it was combined with the modulator galavit. The proof of this regularity was the total sum of ranks – prior and following BTr and BTr+G (see Table 2). For example, the sum of ranks in deep pyoderma before treatment was 10, after BTr – 11, and after BTr+G – 11. In exacerbation of chronic pyelonephritis, there were 11-13 ranks, respectively, etc.

The specific laboratory effect of BTr + G distributed over the links of immunity in deep pyoderma was expressed in complete normalization of the T-Ph-links, in average normalization of the Cyt- and C- and in unsatisfactory normalization of the B- and A-links. According to the above gradation, the same combination of treatment for exacerbation of chronic pyelonephritis led to a different effect: complete normalization of the B- and Cyt-links, average normalization of the T-, Ph- and A-links, and unsatisfactory normalization – of the C-link. In case of exacerbation of chronic adenitis, there was complete normalization of the B-, Ph- and Cyt-links, average normalization of the T-link and unsatisfactory normalization of the C- and A-links. In case of purulent infections of soft tissues, there was complete normalization of the T-, B-, Ph-links, average normalization of the Cyt-links and unsatisfactory normalization of the C- and A-links. In case of exacerbation of chronic cystitis, we observed complete normalization of the T-, B-, Ph- and Cyt-links, and unsatisfactory normalization of the A-links.

Attention was drawn to the fact that there was rather high variability in the nature of reactions of cellular, humoral, phagocytic immunity to galavit, which

Table 2. Galavit immunotherapy effect on the immunity of patients with PID of diverse genesis

<table>
<thead>
<tr>
<th>Disorders</th>
<th>Terms/treatment</th>
<th>Formulas for detailed disorders of immuno-metabolic links of immunity</th>
<th>Σ/level of differences from the norm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Deep pyoderma</td>
<td>Baseline</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>10/I</td>
</tr>
<tr>
<td></td>
<td>BTr</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>11/II</td>
</tr>
<tr>
<td></td>
<td>BTr+G</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>12/III</td>
</tr>
<tr>
<td>Exacerbation of chronic pyelonephritis</td>
<td>Baseline</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>8/I</td>
</tr>
<tr>
<td></td>
<td>BTr</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>11/II</td>
</tr>
<tr>
<td></td>
<td>BTr+G</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>13/II</td>
</tr>
<tr>
<td>Exacerbation of chronicadnexitis</td>
<td>Baseline</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>8/I</td>
</tr>
<tr>
<td></td>
<td>BTr</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>13/II</td>
</tr>
<tr>
<td></td>
<td>BTr+G</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>9/I</td>
</tr>
<tr>
<td>Purulent infection of soft tissues</td>
<td>Baseline</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>12/II</td>
</tr>
<tr>
<td></td>
<td>BTr</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>13/III</td>
</tr>
<tr>
<td></td>
<td>BTr+G</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>9/I</td>
</tr>
<tr>
<td>Exacerbation of chronic cystitis</td>
<td>Baseline</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>12/II</td>
</tr>
<tr>
<td></td>
<td>BTr</td>
<td>Т₂Б₂Н₂Ф₂Ф₂С₂Т₂А₂</td>
<td>14/II</td>
</tr>
</tbody>
</table>

Note: T, B, Ph, Cyt, C, A – cellular, humoral, phagocytic, cytokine, FRO and AOD dependent links of immunity; (•) – normalization of 1, 2, 3 – ranks of differences, numerator Σ ranks, denominator (I, II, III) decreasing level of differences from the norm; all the rest notes are given in the text earlier.
depended on the pathogenesis of diseases, and its rather low correction effectiveness of metabolic and oxidative reactions.

The technique for determining the signaling targets of the modulator (FIT) allows selecting key tests from the total set of laboratory findings significantly changed from the initial level, and representing them in the form of diagnostic formulas. Notably, it is necessary to take into account that the mechanism of action of the complex patients’ treatment is composed of all the treatment options applied: conventional drugs, their combination with a modulator and the “proper” effect of the latter independently on conventional therapy. These data are illustrated by Table 3.

As follows from the data in Table 3, an action of a new quality is developed in 5 mono-PID variants under use of standard immunocorrection with galavit due to the additional immunotrophic effects of all treatment options. For example, FIT of the basic treatment for deep pyoderma includes B+Tcyt+3NBTS+3 (stimulation of the level of B-cells, T- cytophilic lymphocytes, spontaneous NBTest); FIT of the treatment for deep pyoderma using BTr+G includes IL4+3TNF-3Ma+3 (accumulation of interleukin 4 in the context of suppression of tumor necrosis factor and an increased number of carriers of the adhesion marker); FIT of the treatment for deep pyoderma under galavit effect includes TNF+3Ma+3lgM+2 (potentiation of TNF formation, the number of Ma-cells and class M immune globulins). Since three factors are informationally essential – the place of the reference test in the formula, the vector (+, -) and the rank of changes (1, 2, 3), - the originality of the composition of three diagnostic formulas is 100%.

Slightly different results were obtained when studying metabolic tests in patients with purulent-inflammatory diseases. First, in the acute PID, patients were diagnosed with pronounced initial metabolic disorders: in pyoderma, there was a decreased level of vitamin E and the accumulation of FRO-dependent malonic dialdehyde in the context of the suppressed antioxidant activity of the antioxidant properties of blood serum. In patients with exacerbation of chronic pyelonephritis there was an excess concentration of Schiff bases with the suppressed antioxidant enzymes – superoxide dismutase and activated MDA of 2-3 ranks. Chronic adenitis in sick women was accompanied by the stimulated level of antioxidant vitamin E, the suppressed formation of AOA with the stimulated formation of Schiff bases of the 2nd rank. Purulent infection of soft tissues induced the activation of two factors of lipid and protein free-radical oxidation – bityrose crosslinks and ketodienes combined with activated catalysts of 2-3 ranks. Chronic cystitis was accompanied by the inhibited mechanisms of antioxidant activity – non-protein thiols and AOA and superoxide dismutase.

Conventional and complex treatment with the modulator galavit for each nosoform of PID in all five cases did not result in a significant correction of metabolic disorders (see Table 3). After patients being discharged from the hospital, the initial laboratory findings aggravated in all patients, or were normal, but with an unreliably minimal expressiveness.

Under these conditions, the determination of the “proper” effect of galavit, independently on the basic treatment, did not provide a positive effect. And indeed, the obtained formulas FMCTprop supported minimal changes.

To increase the information content of assessment regarding the state of immuno-metabolic reactivity in patients suffering from purulent-inflammatory diseases of various genesis, mathematically selected basic tests of the initial and final formulas of disorders of the immune and metabolic systems were discussed instead of a frontal analysis of variations in dozens of studied laboratory findings. Notably, there were recorded not only entire volumetric variations and grouped parameters of individual immunity links, but also detailed, point-specific diagnostically significant parameters, and their distribution over the main links – T-, B- and phagocytic-dependent (see Table 4).

<table>
<thead>
<tr>
<th>Disease</th>
<th>Basic treatment</th>
<th>+ galavit</th>
<th>Immunotropic effect</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>FIT/FMCT</td>
<td>FIT/FMCT</td>
<td>FMCTprop/FMCTprop</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep pyoderma</td>
<td>B+Tcyt+3NBTS+3</td>
<td>IL4+3TNF-3Ma+3</td>
<td>TNF+3Ma+3lgM+2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exacerbation of chronic pyelonephritis</td>
<td>NBTS+3PhN+3NKp+3</td>
<td>PhI+3IL4+3TNF+3</td>
<td>PhI+3NBTS+3lgG+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exacerbation of chronic adenitis</td>
<td>Tac+3lgM+3Th+3</td>
<td>PhN+3IL4+3NKp+3</td>
<td>NK+3CIC+3lgG+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Purulent infections of soft tissues</td>
<td>lgM+3TNF+3CIC+3</td>
<td>PhI+3NBTS+3MM+3</td>
<td>PhI+3NBTS+3Tac+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exacerbation of chronic cystitis</td>
<td>T+3IL4+3MM+2</td>
<td>PhI+3B+3Th+3</td>
<td>NK+3lgG+3T+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Effect on metabolic immunity</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Deep pyoderma</td>
<td>SchB+AOA+MDA+2</td>
<td>VE+3TT+BC+3</td>
<td>SOD+nonPT+SchB+</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exacerbation of chronic pyelonephritis</td>
<td>AOA+2PT+3DC+1</td>
<td>MDA+3SO+TT+3</td>
<td>VE+3MDA+3AOA+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exacerbation of chronic adenitis</td>
<td>SchB+AOA+K+3</td>
<td>VE+3DC+SchB+</td>
<td>BC+3SO+K+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Purulent infections of soft tissues</td>
<td>BC+VE+3KD+2</td>
<td>AOA+3BC+CP+</td>
<td>CP+nonPT+KD+3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Exacerbation of chronic cystitis</td>
<td>MDA+3SO+nonPT+</td>
<td>KD+3SchB+MDA+2</td>
<td>MDA+SchB+VE+3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Note: CP – ceruloplasmin, all the rest notes are given in the text earlier.*
It is demonstrated that, under pharmacological modulation with galavit, there was a change in the nature of immunological and metabolic parameters at the level of individual links for the entire studied systems and the key components of typical formulas. In these circumstances, the effect of complex treatment on immunological parameters was generally positive, and on metabolic tests – intact.

It is noteworthy that this demonstrated trend was regulated by clinical variants of diseases. So, in deep pyoderma the ratio of altered T: B: H links of immunity before treatment was 1:3:2, and after treatment with galavit – 3:3:1; in chronic pyelonephritis, on the contrary, the dynamics was 1:3:2 and 3:2:1, respectively; in purulent infections of soft tissues – 1:2:3 and 2:2:2; in chronic cystitis – 1:1:1 and 1:2:3. There was an evident modification of the nature of immunopathology in patients when being discharged from the hospital. Apparently, this is explained by the fact that the repeated terms of patients’ follow-up examinations – 2-3 weeks – is not optimal enough to achieve the final immunomodulation, which develops a few months later.

Analysis of the components of the initial and final FISD allows reporting on a set of diagnostically significant parameters, the vector of their change for stimulation or suppression, the degree of severity of variations in the course of treatment and, thus, evaluating the effectiveness of immunocorrection. The following regularity has been stated: in patients with PID in the initial period, the ratio of the number of suppressed and activated signal tests of the formulas was 44.4% and 55.6%, after immunotherapy with galavit – 6.6% and 94.4%, respectively. We can state the predominance of prevalent stimulation of the immune status under the effect of differentiated correction.

This regularity is supported by the analysis of variations in the parameters of metabolic immunity - the low final efficiency of the normalization of metabolic disorders and the final modification of disorders in the form of replacing signal tests of typical diagnostic formulas.

Conclusions

We examined more than 280 individuals suffering from various types of purulent-inflammatory diseases – pyoderma, pyelonephritis, adnexitis, purulent infection of soft tissues, cystitis – who underwent additional treatment with the modulator galavit. As demonstrated, the additional treatment with the modulator galavit had the following effects: the pharmacological body-wide effect (on grouped hemato-immuno-metabolic-bacteriological and clinical parameters) and the detailed two-level profile immuno-metabolic effect (on grouped individual links and on specific immuno-metabolic parameters) formalized in diagnostic formulas. Notably, all the listed patterns depended on the clinical nosological forms of purulent-inflammatory diseases.

Conflict of interests

The authors declare no conflict of interests.
References


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