Abstract
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CHANGES IN HORMONAL CONTROL IN PATIENTS WITH DEMODICOSIS AS A RESPONSE TO PARASITIC INFESTATION

The paper establishes patterns of changes in clinical and hormonal parameters which deepen our knowledge on the pathogenesis of demodicosis and create certain preconditions for the directed correction of compensatory-adaptive possibilities of the host organism and open prospects for improving pathogenetic treatment of demodicosis and its complications.

Keywords: demodicosis, sex, gonadotropins hormones, changes in hormonal control, parasitic infestation.

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Резюме
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ЗМІНИ ГОРМОНАЛЬНОЇ РЕГУЛЯЦІЇ У ХВОРИХ НА ДЕМОДЕКОЗ У ВІДПОВІДЬ НА ПАРАЗИТАРНУ ІНВАЗІЮ

У роботі виявлені закономірності змін клініко-гормональних показників, що розширюють знання про патогенез демодекозу, а також створюють певні передумови для спрямованої компенсаторно-приспосіблювальної можливостей організму господаря для удосконалення патогенетичного лікування демодекозу та його ускладнень.

Ключові слова: демодекоз, статеві гормони, гонадотропні гормони, зміни гормональної регуляції, паразитарна інвазія.

ізменения гормональной регуляции у больных демодекозом в ответ на паразитарную инвазию

В работе выявлены закономерности изменений клинико-гормональных показателей, расширяющих знания о патогенезе демодекоза, а также создающих определенные предпосылки для направленной коррекции компенсаторно-приспособительных возможностей организма хозяина для совершенствования патогенетического лечения демодекоза и его осложнений.

Ключевые слова: демодекоз, половье гормоны, гонадотропные гормоны, изменение гормональной регуляции, паразитарная инвазия.

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Вступ
A relatively small number of parasitic diseases is able to cause acute disease with severe characteristic clinical manifestations: malaria, sleeping sickness, leishmaniasis, amoebiasis, and trichinosis. The feature of most parasitic diseases is chronicity, due to the long-term presence of the pathogen in the patient (without specific treatment), defined by the lifetime or the parasite or its frequent reinfection. Such patients can only be cured after a course of specific therapy with anthelminthic drugs [8].

Even long parasitizing in the patient may not lead to severe acute clinical manifestations. The long course of many parasitic diseases is accompanied by various nonspecific clinical signs: fatigue, weakness, loss of appetite. General practitioners do
not usually associate these signs of body asthenia with parasites, which in turn leads to late and often to a false diagnosis.

Allergyization and immunosuppression are the most common pathological factors of virtually all pathogens of parasitic diseases. Allergyization in lambliasis and intestinal helminthiasis initiates or supports chronic allergic dermatosis (neurodermatitis, eczema). There is a link between helminthiasis and alopecia as well as with nodal depigmentation of the skin [8]. Other parasitic diseases: toxocarosis, hymenolepiasis, pulmonary distomiasis may cause asthma and chronic bronchitis. An examination of almost 200 patients with asthma who were treated at the clinic of the Institute of Immunology, Ministry of Health of Russia, carried out by the specialists of E.I. Martsinovskii Institute of Medical Parasitology and Tropical Medicine found parasitic pathology in 34.5% of patients.

In this regard, the most urgent task in the research of the problem of parasitic infestations is the study of relationships between the parasite and the definitive host. One of the approaches in this area can be studying processes of adaptation disorders under the influence of parasites on the host [4].

Adaptation to various damaging factors is carried out at all levels of the body, but mainly its mechanism is implemented through changes in the central nervous system and in the hypothalamic-pituitary-adrenal system [3, 6].

Overexertion of regulation systems can lead to depletion of the body's defenses, reducing its functionality. Prolonged exposure to any parasitic infestations as stress factors can lead to the activation of the nervous and hormonal regulation while maintaining the adaptive capacity of the body and may become a pathogenetic basis of various functional disorders and lesions along with lower adaptive capacity [5].

Thus, the study of the role of hormonal and metabolic regulatory systems in parasitosis has a scientific interest in terms of the formation of host-parasite relationships, deepening knowledge on the pathogenesis of parasitic infestations (for example demodicosis) organ pathology and protective-adaptive reactions, and for justifying corrective therapy and enhancement of the treatment efficiency.

The study of demodicosis in young people remains the focus of dermatologists, due to a great social and psychological significance of the disease. Demodicosis incidence in young people is about 90% of boys and 80% of girls under the age of 21 [16, 17]. The peak incidence of the disease occurs in girls aged 14-16 and in boys aged 16-17 [10]. Development of demodicosis leads to a severe psychological trauma that affects social adaptation of teenagers. According to the results of special psychological studies conducted by T. Kramer in 1998 [15], 38% of adolescents aged 13-16 develop psychological disorders associated with demodicosis.

In this regard, in recent years, interest in the demodicosis has increased sharply and there was a need for a deeper study of this disease, the development of new methods of diagnosis, treatment and prevention. This, in turn, determines the relevance of studies on the state of compensatory-adaptive reactions in demodicosis.

Objective. To evaluate changes in hormonal control of the definitive host developing in response to parasitic infestation in patients with demodicosis.

Material and methods. While performing the experiments, we applied commonly used laboratory methods and clinical examination of patients, parasitological and hormonal, instrumental and statistical methods as well.

To perform the tasks, we examined 109 people (63 men and 46 women) aged from 20 to 60 and 20 healthy people of similar age and gender who were treated at the Department of Medical Parasitology and Tropical Diseases in 2008-2011., and who underwent a comprehensive clinical, laboratory and instrumental examination. To assess the hormonal status, basal levels of pituitary hormones (follicle-stimulating (FSH), luteinizing hormone (LH), prolactin) were studied as well as peripheral endocrine glands hormones (testosterone, estradiol, progesterone).

The severity of lesions was assessed by the standard classification of acne - mild comedonal and superficial papulopustular ones with a small number of pustular acne-elements (0.25-1.5 points on the scale of Allen, Smith), a form of moderate severity-abundant papulopustular rash and comedones (2-5.5 points on the scale of Allen, Smith), a severe form along with numerous comedones there were a lot of deep papulopustules, nodular and nodular-cystic components (6-8 points on the scale of Allen, Smith).

Statistical handling of the research results was carried out on a computer IBM PC/AT using appropriate software for correlation analysis. In some cases, we used the method of statistical analysis by means of the fitting criterion $\chi^2$ (correspondence tables 2 x 2). Based on the fact that the average values of many of the studied parameters were
within the mark, we used more informative way for such cases - the analysis of the incidence of abnormal values. We considered them to be abnormal when the deviations of the mean values were of 2 (root-mean-square deviation).

**Results.** The duration of suffering from demodicosis on the day of examination, according to the patients ranged from several months to 2-5 years.

The examination of patients with demodicosis was carried out by a detailed survey and careful checking up. It made it possible to reveal at first glance, even minor symptoms that do not immediately attract attention, and patients had not found them important.

Clinical observation of patients with demodicosis shows that, in outward appearance, the toxicity manifests with eritematosus spots on the skin, focal or diffuse infiltration, peeling into tiny follicles or rough plates, formation of pink or red follicular papules of varying size, papulo-vesicles, papulo-pustules and independent macropustules. And still, there were some complaints of patients, accompanied by subjective feelings: itching, burning, tightening skin, reducing the elasticity and softness of the skin, the feeling of the parasite penetrating into and crawling underneath the skin.

In most patients, 71.55% (78 people) the disease debuted in their childhood and adolescence. In primary treatment, patients complained of rashes on the face 99.1% (108 people), on the face and back - 30.3% (33 people), on the face and chest - 4.5% (5 people) on the face, chest and back 11.6% (13 people), only on the face 53.5% (58 people).

In our study, patients with moderate (2-5.5 points on the scale of Allen and Smith), made up 50%.

As a result of our studies we have found differences in the contents of the studied hormones in the blood and their relationships, which indicates a change in the function of the pituitary-gonadal system and disturbance which are characteristic of healthy human hormonal relationships that have a different course depending on gender. In men, there was increasing concentrations of estradiol and progesterone and testosterone decline against a moderate increase of gonadotropin hormones. In women, the content of testosterone and progesterone increased while estradiol relatively decreased against the background of relatively slight increase of LH and FSH decline.

In healthy women the prolactin rate was higher than in men, which corresponds to the medical literature data.

In patients with demodicosis the content of prolactin in the blood ranged widely (from 100 to 1400 mg/l). In patients with normal liver function prolactin age differences remained. In patients aged older than 40 years, the hormone (311.43 ± 31.51 mg/ml) was significantly higher than under 40 years (218.13 ± 23.31 mg/l). In women the average prolactin rate was not significantly different from the mark (Table 1).

**Table 1 – The concentration of LH and FSH in blood of patients with demodicosis (X ± Sx)**

<table>
<thead>
<tr>
<th>Studied data</th>
<th>Gender</th>
<th>Patients with demodicosis</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>LH (pmol/l)</td>
<td>A</td>
<td>6.83 ± 0.87</td>
<td>5.97 ± 0.63</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>8.17 ± 0.93</td>
<td>9.12 ± 0.41</td>
</tr>
<tr>
<td>FSH (pmol/l)</td>
<td>A</td>
<td>5.78 ± 0.45</td>
<td>3.62 ± 0.42</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>4.84 ± 0.62</td>
<td>7.89 ± 0.17</td>
</tr>
<tr>
<td>FSH/LH</td>
<td>A</td>
<td>0.85</td>
<td>0.60</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>0.59</td>
<td>0.86</td>
</tr>
</tbody>
</table>

Note:
* - reliability of differences from control;
× - reliability of sex differences;
■ - reliability of differences between groups;
A – men;
B – women

In demodicosis the changes of ratios of gonadotropin and sex steroid hormones and a reduction of correlation between them indicate a disturbance of optimal relationships typical in healthy people, and changing regulatory processes, which in demodicosis develope earlier than morphological ones.
Basal hormone rate changed differently immediately after the treatment. Average prolactin rate in men and women increased after the treatment.

The data show that after the treatment of demodicosis there are some changes in the content and ratios in the studied hormones.

Especially noteworthy is the reduction of these hormones (estradiol and prolactin) with a negative therapeutic effect, while with positive clinical effect the level of these hormones usually increased (Table 2).

### Table 2 – Concentration of sex steroid hormones in blood of patients with demodicosis (X ± S,

<table>
<thead>
<tr>
<th>Studied data</th>
<th>Gender</th>
<th>Patients with demodicosis</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Estradiol (nmol/l)</td>
<td>A</td>
<td>0.17 ± 0.01</td>
<td>0.11 ± 0.02*</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>0.23 ± 0.05</td>
<td>0.84 ± 0.02*</td>
</tr>
<tr>
<td>Testosterone (nmol/l)</td>
<td>A</td>
<td>11.70 ± 2.68</td>
<td>21.88 ± 0.76*</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>7.68 ± 1.51</td>
<td>1.88 ± 0.05*</td>
</tr>
<tr>
<td>Progesterone (nmol/l)</td>
<td>A</td>
<td>5.24 ± 1.37</td>
<td>1.04 ± 0.04*</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>9.59 ± 2.39</td>
<td>3.50 ± 0.07*</td>
</tr>
<tr>
<td>Estradiol/Testosterone</td>
<td>A</td>
<td>0.73*</td>
<td>0.25*</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>0.94*</td>
<td>3.62*</td>
</tr>
</tbody>
</table>

*Note:*
- × – reliability of the sex differences (P ≤ 0.05);
- ■ – reliability of differences from control (P ≤ 0.05);
- A – men;
- B – women

At this stage of research it is difficult to decide whether changes in the content of hormones are the result of negative therapeutic effect or whether they cause it. But it is safe to note that the reduction of the above hormones in the blood after specific treatment of demodicosis is an unfavorable prognostic sign.

This underlines the important role of hormonal disorders in the development and course of demodicosis and organ pathology in it, the more pronounced the longer duration of the disease.

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### Висновки

Identified patterns of the changes and the nature of the dynamics of hormonal relationships are not specific, but they deepen the knowledge on the pathogenesis of demodicosis and development of various organ pathologies; they also create certain preconditions for directional correction of compensatory-adaptive possibilities of the host organism.

### References (список літератури)


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