

**Kolisnyk I.A.,**

*Higher State Educational Establishment of Ukraine  
"Ukrainian Medical Stomatological Academy", Associate Prof.,  
Ph.D. in Medical Sciences, Dental Faculty,*

**Korotich N.N.,**

*Higher State Educational Establishment of Ukraine  
"Ukrainian Medical Stomatological Academy", Associate Prof.,  
Ph.D. in Medical, Sciences, Dental Faculty,*

**Pankevych A.I.,**

*Higher State Educational Establishment of Ukraine  
"Ukrainian Medical Stomatological Academy", Associate Prof.,  
Ph.D. in Medical, Sciences, Dental Faculty*

## ***Correlation of some immunologic indices with the severity of pterygopalatine ganglionitis***

**Introduction.** Pterygopalatine ganglionitis (PPG) within the group of facial pain syndromes represents particular challenge both in dental practice and neurology.

Polymorphism of clinical manifestations significantly hinders the diagnosis of the disease and is associated with topographic and anatomical peculiarities of the pterygopalatine gland structure. The etiology and pathogenesis of this disease is not known to date, however, the role of immune system in the development of both acute and chronic inflammatory processes is proven and undeniable. Any chronic disease overloads the immune system and leads to formation of the secondary immune deficiency and impaired efficacy of corresponding host defense mechanisms [1, 2].

Publication data as well as findings of the previous studies show the occurrence of immune disorders in patients with PPG, particularly, the increased production of secretory IgA [3, 4]. Moreover, the dysfunction of immune system has been found in patients with head ganglionitis, which is manifested by the moderate activation of antibacterial mechanisms and suppression of interferogenesis concomitant with apparent disorder of secretory immunity [5]. Absence of clear view on the etiolo-

gy of such alterations encouraged us to carry out more profound analysis of immunologic indices of peripheral blood.

Thus, **our research was aimed at** study of the state of immune system in patients with pterygopalatine ganglionitis according to the degree of its severity.

### **Materials and Methods**

Immunoassay of 62 patients with PPG, aged 30 to 74 years, has been carried out. On the basis of clinical observation data the patients were assigned into three groups according to the degree of severity: minor (20 individuals), moderate (22 individuals) and severe (20 individuals) [6]. 10 individuals aged 40 to 60 years were assigned to control group.

The state of cell-mediated immunity has been evaluated by defining the number of CD3<sup>+</sup>, CD4<sup>+</sup>, CD8<sup>+</sup>, CD16<sup>+</sup>, CD20<sup>+</sup> cells and the CD4<sup>+</sup>/CD8<sup>+</sup> ratio (immunoregulatory index). Immunoglobulins of A, M, G class and the level of circulating immune complexes (CIC) in blood serum have been also determined.

### **Results**

Among the clinical indices, used to determine the degree of PPG severity degree special attention should be given to the description of pain attack, i.e., its daily intensity, duration and rate of occurrence. Dysaesthesia and palpatory tenderness of cranial nerves exit points has been also detected. The pain intensity has been evaluated by the Visual Analog Scale, VAS [7].

Clinically, the minor degree of severity is characterized by the local pain of minor intensity (up to 4-5 points), lasting up to 30 min, and the rate of attacks occurrence was 1 to 3 times daily. Normally, in minor form of PPG no dysaesthesia has been observed (59,09%); sometimes minor hyperesthesia has been detected. Palpation of projected cranial nerves exit points was painless (54,55%) or low painless (45,45%).

Patients with moderate degree of severity suffered from pain, extended to the whole side of the face, with intensity of 5-7 points, lasted from 30 min to 1-2 hours, and the rate of paroxysms occurrence was 4-5 times daily. Dysaesthesia was manifested by both hyperesthesia (74,42%) and hypesthesia (23,26%). Palpation of cranial nerves exit points was painful.

The severe course of the disease was characterized by the intense pain, extended to the whole side of the face, radiating in the neck, shoulder and scapula, of 8-10 points, lasted from 2 hours and more, and the rate of attacks occurrence was 4

to 6-10. Dysaesthesia was manifested more frequently by paresthesia (53,57%), or hypesthesia (28,57%), and rarely by hyperesthesia (17,86%). Palpation of cranial nerves exit points was painful or sharply painful.

The assessment of immunoassay findings has shown that the immunodeficiency disorder was tending to develop in different severity of the disease (Table 1).

**Table 1**

**Indices of cell-mediated and humoral immunity in patients with pterygopalatine ganglionitis and controls (M±m)**

Indices	Control group (n=10)	Patients with PPG		
		minor degree (n=20)	moderate degree (n=22)	severe degree (n=20)
CD3 <sup>+</sup> , %	71,5±1,23	68,23±1,03*	64,28±1,17*	60,34±1,02*
CD4 <sup>+</sup> , %	42,2±0,84	39,1±0,54*	37,87±0,64*	35,94±0,64*
CD8 <sup>+</sup> , %	20,2±0,36	20,9±0,42	21,35±0,48*	22,97±0,36*
CD4 <sup>+</sup> /CD8 <sup>+</sup>	2,09±0,03	1,87±0,04*	1,78±0,06*	1,58±0,04*
CD16 <sup>+</sup> , %	16,2±0,83	16,4±0,69	17,32±0,52	17,94±0,51*
CD20 <sup>+</sup> , %	13,5±0,75	13,2±0,59	13,12±0,29	12,97±0,29
IgA (g/l)	2,01±0,12	1,89±0,09	1,67±0,09*	1,12±0,06*
IgM (g/l)	1,21±0,09	1,32±0,07	1,41±0,07*	1,5±0,05*
IgG (g/l)	9,57±0,49	9,86±0,56	10,59±0,25*	11,26±0,25*
CIC (units of optical density)	0,04±0,005	0,05±0,004*	0,06±0,005*	0,08±0,005*

Note. \* – reliability of difference between the indices of patients and controls ( $p < 0,05$ ).

The analysis of subpopulations of lymphocytes in patients with minor degree of PPG severity has shown insignificant changes in composition and ratio of such cells. Generally, the CD4<sup>+</sup> concentration was significantly decreased and level of CD8<sup>+</sup> cells was increased in the observed patients as compared with healthy individuals.

The CD4<sup>+</sup>/CD8<sup>+</sup> ratio (immunoregulatory index (IRI) is crucial in the immune system evaluation, since it reflects the adequacy of the immune response. The IRI

was significantly decreased in patients with PPG as compared with controls, indicating about the chronicity of the process.

No significant changes of humoral immunity indices have been observed. The CIC level was increased in contrast to controls.

Changes in both cellular and humoral components of the immune system have been detected in patients with moderate degree of PPG severity. The description of cell-mediated immunity of patients showed the significant decrease of CD3<sup>+</sup>, CD4<sup>+</sup> cells, the increase in number of CD8<sup>+</sup> cells, and imbalance of its populations, accompanied by the IRI lowering. The findings give grounds to assume the development of the secondary immune deficiency on the T-cell component.

Indices of humoral immunity in the observed patients showed that the IgA level was decreased. The decreased IgA in blood serum is typical for autoimmune diseases and can be the consequence of body sensitization to nerve tissue antigens.

The IgM level was a little increased in patients with PPG as compared with controls. IgM can activate the complement, which ensures the realization of complement-mediated cytotoxicity.

The IgG index was significantly increased in patients with PPG. The increase of IgG level occurs in recurring contact with antigen, indicating about the occurrence of chronic nidus of infection and its mediated role in etiology of ganglionitis. Since the IgG ensure secondary immune response, then sometimes its rise is observed in acquired defects of immune system.

The significant increase of the CIC level has been also detected in patients with PPG.

Changes in cell-mediated and humoral components of the immune system were apparent in patients with the severe PPG.

The significant decrease of CD3<sup>+</sup> cells was found in peripheral blood. The decrease in absolute number of blood T-lymphocytes indicates about the cell-mediated immunity deficiency and is observed in the inflammatory processes with chronic clinical course. Decreased CD4<sup>+</sup> concentration and increased CD8<sup>+</sup> level was observed in patients with PPG as compared with healthy individuals. The increased number of CD8<sup>+</sup> in blood indicates about the immunodeficiency and is observed in acquired immunodeficiency disorders.

The description of cell-mediated immunity in the observed patients showed the significant decrease of CD3<sup>+</sup>, CD4<sup>+</sup> cells and increased level of CD8<sup>+</sup>, CD16<sup>+</sup>, and

imbalance of its populations, accompanied by the IRI lowering. The findings show the occurrence of secondary immune deficiency on the T-cell component in the observed patients.

The decreased IPI confirms the disorder of cell-mediated component of immune system. Insignificant decrease in number of B-lymphocytes (CD20<sup>+</sup>) in the observed patients we associate with the duration of underlying disease and the rate of its occurrence, affecting the state of the immune system.

Indices of the humoral immunity showed the decreased level of serum IgA and increased IgM, IgG and CIC in the observed patients as compared with controls.

### **Conclusions**

In summary, disorder of cell-mediated component of immune system has been observed in patients with minor degree of severity; the secondary immune deficiency on the T-cell component, imbalance of humoral component of immune system and high CIC indices has been detected in patients with moderate degree of severity that may indicate about the occurrence of latent nidus of inflammation in the observed patients and indirectly confirm the autoimmune mechanisms of progression of the disease [1].

Apparent changes both in cell-mediated and humoral components of the immune system, i.e., the secondary immune deficiency on the T-cell component, imbalance of humoral component of immune system and high CIC indices have been detected in the severe PPG.

### **References:**

1. Дранник Г. Н. Клиническая иммунология и аллергология: пособие / Г. Н. Дранник. – 4-е изд., доп. – К.: Полиграф плюс, 2010. – 552 с.
2. Казмірчук В. Є. Клінічна імунологія та алергологія / В. Є. Казмірчук, Л. В. Ковальчук. – Вінниця: Нова книга, 2006. – 526 с.
3. Пузин М.Н Иммунологический статус у больных с болевыми синдромами в области лица и головы / М.Н. Пузин, Е.В. Гречко // Журн. невропатологии и психиатрии им. Корсакова. – 1989. – № 12. – С. 44-48.
4. Гречко В.Е. Иммунологические аспекты вегетативных прозопагий / В.Е. Гречко, М.Н. Пузин, К.Е. Балашов // Журн. невропатологии и психиатрии. – 1990. – № 1. – С. 53-57.

5. Николаева А.А. Клинико-иммунологическая характеристика и оптимизация терапии постгерпетического ганглионита у пациентов с лицевыми болями: автореф. дисс. на соискание научн. степени канд. мед. наук: спец. 14.01.14 «Стоматология» / Анастасия Александровна Николаева. – Екатеринбург, 2015. – 24 с.
6. Колісник І.А. Лікування крилопіднебінного гангліоніту імунобіологічними препаратами: дис... канд. мед. наук: 14.01.22 / Інна Анатоліївна Колісник. – Полтава, 2006. – 187 с.
7. Вейн А.М. Заболевания вегетативной нервной системы: Рук-во для врачей / А.М. Вейн. – М.: Медицина, 1991 – 622 с.