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Violation of Cellular and Humoral Immunity in Patients with First Emerged and Progressive Angina

Viktor Lyzogub¹, Tetyana Zavalskaya¹, Khaled Ahmad Khalil Abu Sara¹, Mariya Sayuk^{2*}

1. Department of Internal Medicine № 4, O.O. Bogomolets National Medical University Kyiv, Ukraine.
2. Department of Therapy and Family Medicine of Faculty of Postdiploma Education, Ivano-Frankivsk National Medical University, Ivano-Frankivsk, Ukraine
[E-mail: marial164@mail.ru]

The study involved 67 patients with unstable angina (UA) aged 55 to 69 years - 20 patients with first emerged angina (FEA) and 47 patients with progressive angina (PSA). The control group (CG) consisted of 22 healthy persons. Defining populations and subpopulations of lymphocytes were carried out using monoclonal antibodies. Definition Immunoglobulin's IgG, IgA, IgM and circulating immune complexes (CIC) in serum blood was performed by the method of Mancini. In patients with FEA and PSA compared with healthy individuals revealed significantly lower levels of lymphocyte populations. In patients with PRS, in contrast to patients with FEA, found significantly higher populations of B lymphocytes (CD22+) healthy individuals. Thus, in patients with severe forms of ischemic heart disease(IHD), coronary circulation destabilization accompanied by an imbalance of populations and subpopulations of lymphocytes manifested inhibition of cellular immunity due to a population of T-lymphocytes (CD3 +) and T-suppressor (CD8 +). In patients with PRS, except the above, found increased activity of cellular immunity by subpopulation of T-helper cells (CD4 +) and a population of B-lymphocytes (CD22 +). Noteworthy is the fact that patients with PRS compared with patients with FEA determined significantly higher values lymphocyte levels, populations of T-lymphocytes (CD3 +), subpopulation of T-helper cells (CD4 +), suggesting a more pronounced inflammatory processes in patients with PRS. Analyzing changes in humeral immunity in patients with FEA and PSA compared with CG, found under significant increase of IgG, circulating immune complexes (CIC). In patients with PRS compared with patients with FEA IgG level was significantly increased 3.2 times. Therefore in violation of the coronary circulation is increased activity of humeral immunity, whereby patients with PSA expressed more than patients FEA.

Keyword: First Emerged Angina, Progressive Angina, Cellular Immunity, Humoral Immunity, Lymphocytes.

1. Introduction

The attention of researchers is constantly attracting mechanisms of the pathogenesis of atherosclerosis as a dominant factor for coronary heart disease (CHD)^[2,6]. The main areas of research are the study dyslipidemia and immune inflammation^[1]. The hypothesis about the role of inflammation in atherosclerosis has advanced R. Virchow in 1856^[10]. During the following decades, this issue devoted their work such

prominent scholars as R. Ross^[8,9], V. Mazurov^[5], J. Willerson^[11]. At present it is proved that the activity of T-cell and humeral components of the immune inflammation associated with lipid per oxidation and protein^[4]. Of particular importance is the study of immunological reactivity in patients with severe forms of ischemic heart disease: unstable angina (UA) and myocardial infarction (MI).

The purpose of the study is Detecting of cell-mediated and humoral immunity in patients with first emerged angina (FEA) and progressive angina (PSA).

2. Material and methods

The study involved 67 patients with unstable angina (UA) aged 55 to 69 years (mean age was $61,6 \pm 7,5$ years) - 20 patients with FEA and 47 patients with PSA. The control group (CG) consisted of 22 healthy persons. Diagnosis UA established on the basis of generally accepted criteria proposed by World Health Organization (WHO) experts^[3]. According to the classification given in the guidelines for the diagnosis and treatment of the UA of the U.S.A agency policy on health and scientific research on the recommendation of E. Braun Wald (1996)^[7], in the study were taken ill with FEA (diagnosis established within 28 days from the appearance of the first angina attack) and patients with PSA (the transition of stable angina in higher functional class). In the survey did not include patients with heart failure II B and III stages,

atrial fibrillation, concomitant diseases at the stage of decomposition, cancer, diseases of the musculoskeletal system.

Defining populations and subpopulations of lymphocytes were carried out using monoclonal antibodies (definition phenotype of lymphocytes in tests rosette with particles coated with a monoclonal antibody CD3 +, CD4 +, CD8 +, CD22 +), where CD3 + - population of T lymphocytes, CD4 + - subpopulation of T-helper cells, CD8 + - subpopulation of T suppressor cells, CD22 + - population of B lymphocytes. Definition Immunoglobulin's IgG, IgA, IgM and circulating immune complexes (CIC) in serum blood was performed by the method of Mancini.

3. Results and discussion

Comparison of cellular and humoral immunity in patients with FEA and PSA together and CG respectively revealed significant changes on most indicat PSA, as presented in Table 1.

Table 1: Indicat PSA of immune reactivity in patients with FEA And PSA compared with CG, (M ± m)

| Index | CG(I) | FEA (II) | PSA (III) | P I-II | P I-III | PII-III |
|--------------------|------------|------------|------------|--------|---------|---------|
| Lymphocytes | 29,95±0,61 | 14,1±0,24 | 17,1± 0,12 | <0,01 | <0,01 | <0,05 |
| CD3+ (%) | 49,3±2,7 | 33,8±0,27 | 37,9± 0,56 | <0,01 | <0,01 | <0,05 |
| CD4+ (%) | 31,19±1,32 | 32,5±1,81 | 39,7± 0,45 | >0,05 | <0,01 | <0,01 |
| CD8+ (%) | 20,59±0,97 | 11,5±0,63 | 13,8± 0,76 | <0,01 | <0,01 | >0,05 |
| CD22+ (%) | 19,46±1,5 | 21,1±0,61 | 23,6± 0,67 | >0,05 | <0,01 | >0,05 |
| CD4/CD8 | 1,58±0,01 | 2,83±0,04 | 2,87±0,06 | <0,05 | <0,05 | >0,05 |
| IgA (G/l) | 2,34±0,07 | 1,70±0,04 | 3,2±0,05 | <0,05 | <0,05 | <0,05 |
| IgM (G/l) | 1,05±0,09 | 1,0±0,03 | 1,1 ±0,05 | >0,05 | >0,05 | >0,05 |
| IgG (G/l) | 10,96±0,84 | 12,12±0,65 | 16,4 ±0,05 | <0,05 | <0,01 | <0,01 |
| CIC | 1,42 ±0,06 | 4,5±0,47 | 7,8 ±0,04 | <0,01 | <0,01 | <0,01 |

In patients with FEA and PSA compared with healthy individuals revealed significantly lower levels of lymphocyte populations, respectively, by 53% (p <0.01) and 43% (p <0.01), the level populations of T-lymphocytes (CD3 +) - 31, 5% (p <0.01) and 23.2% (p <0.01)., level subpopulation of T-suppressor (CD8 +) - by 44.2% (p <0.01) and 33% (p <0.01). In patients with both clinical forms of UA compared with CG observed significantly higher values immunoregulatory index (IRI) (CD4/CD8)

respectively 1.7 (p <0.01) and 1.8 (p <0.01) times. The level of T-helper cells (CD4 +) in patients with FEA were not significantly different from CG (p > 0.05), and in patients with PRS, this figure was significantly higher by 21.5% (p <0.01). In patients with PRS, in contrast to patients with FEA, found significantly higher populations of B lymphocytes (CD22 +) by 17.4% (p <0.01) compared with healthy individuals.

Thus, in patients with severe forms of ischemic heart disease (IHD), coronary circulation destabilization accompanied by an imbalance of populations and subpopulations of lymphocytes manifested inhibition of cellular immunity due to a population of T-lymphocytes (CD3 +) and T-suppressor (CD8 +). In patients with PRS, except the above, found increased activity of cellular immunity by subpopulation of T-helper cells (CD4 +) and a population of B-lymphocytes (CD22 +).

Noteworthy is the fact that patients with PRS compared with patients with FEA determined significantly higher values lymphocyte levels by 17.5% ($p < 0.05$), populations of T-lymphocytes (CD3 +) - by 10.9% ($p < 0.05$), subpopulation of T-helper cells (CD4 +) - by 18.3% ($p < 0.01$), suggesting a more pronounced inflammatory processes in patients with PRS.

Analyzing changes in humoral immunity in patients with FEA and PSA compared with CG, found under significant increase of IgG in 1.1 times ($p < 0.05$) and 1.5 times ($p < 0.01$). In patients with PRS compared with patients with FEA IgG level was significantly increased 3.2 times ($p < 0.01$). On increasing the activity of humeral immunity in patients with NS compared with healthy individuals shows significantly higher levels of circulating immune complexes (CIC in both study groups, respectively 3.2 and 5.5 times (in both cases ($p < 0.01$)). B turn, the level of CIC in patients with PRS compared with patients with UVD 1.7 times higher ($p < 0.01$). Therefore in violation of the coronary circulation is increased activity of humeral immunity, whereby patients with PSA expressed more than patients FEA.

4. Conclusions

1. In patients with FEA and PSA compared with healthy individuals disturbed balance of populations and subpopulations of lymphocytes with increased activity of B-lymphocytes, and decreased activity of T-lymphocytes and T-suppress PSA.
2. In patients with PRS, in contrast to patients with FEA, increasing subpopulation of T-helper cells.

3. In patients with FEA and PSA compared with CG revealed increased activity of humoral immunity by increasing levels of IgG and increased CIC.
4. In patients with PRS compared with patients with FEA observed a significant increase of IgG and CIC.

5. References

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