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# EVALUATION OF LABORATORY-IMMUNOLOGIC METHODS OF CARDIOVASCULAR PATHOLOGY IN PSORIATIC ARTHRITIS PATIENTS

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**Abstract:**
According to modern researchers, one of the most common comorbid diseases in patients with psoriatic arthritis (PsA) is cardiovascular disease (CVD). The presence of CVD in PsA increases the risk of cardiovascular complications (CVC) that lead to early disability in patients with this disease. In general, it was found that early diagnosis of CVD in PsA, not only can prevent CVC, but also can significantly improve the tactics of patient management, prevention of complications and improve the quality of life of patients with PsA.

**Keywords:** psoriatic arthritis, cardiovascular diseases, laboratory-immunologic methods of research.

**INTRODUCTION.** One of the most serious cardiovascular complications associated with PsA is an increased risk of atherosclerosis and cardiovascular disease (CVD) [1,2,7].
Chronic inflammation, the hallmark of PsA, is known to contribute to the development and progression of atherosclerosis through various mechanisms such as endothelial dysfunction, impaired lipid metabolism, vascular remodeling, and prothrombotic state [3,4,8].
As a result, people with PsA are at increased risk of myocardial infarction, stroke, congestive heart failure, and cardiovascular mortality. Several factors contribute to the increased cardiovascular risk observed in patients with PsA [5,6].

**PURPOSE OF THE STUDY:** evaluation of the indicators of laboratory-immunologic methods of cardiovascular pathology in patients with psoriatic arthritis.

**MATERIAL AND METHODS OF THE STUDY.** The study involved 125 individuals who signed informed written consent. All patients depending on the presence of cardiovascular diseases (CVD) and PsA were randomized into three groups. Group I combined 62 patients with PsA complicated by CVD, group II included 32 patients with psoriatic arthritis (PsA) who did not have concomitant CVD. As an additional comparative (III) group, we included patients with clear signs of CVD without PsA.
All patients in all study groups underwent clinical, laboratory and instrumental methods of investigation. All patients underwent such laboratory examination methods as general blood count, urinalysis, acute-phase blood parameters, biochemical blood tests, lipid spectrum, coagulogram, cytokines and markers of endothelial damage.

**RESULTS OF THE STUDY.** In all study groups (I, II, III and in the control group) in outpatient conditions of SCAL and in inpatient multidisciplinary clinic of the Tashkent Medical Academy, as well as in the conditions of the central research laboratory of TMA a complex clinical, biochemical and laboratory research was carried out.
In the study of general blood analysis, we paid attention to hemoglobin, erythrocyte count, leukocyte count and erythrocyte sedimentation rate - ESR (Table 1).

**Table 1**
**Evaluation of the general blood analysis in I, II, III and control study groups (n=145)**

| GBA parameters | Control | I | II | III | p |
|---|---|---|---|---|---|
| **hemoglobin g/l** | 125,9 ±8.8 | 112,8± 17,7 | 110,3± 20,1 | 123,5± 15,1 | p>0 ,05 |
| **erythrocyte $10^{12}$/l** | 4,2±0, 5 | 3.8 ±0,6 | 3,6±0, 7 | 4,1±0, 5 | p>0 ,05 |
| **leukocyte $10^{9}$/l** | 6,5±1, 2 | 6,2±1, 3 | 6,9±2, 3 | 6,1±1, 3 | p>0 ,05 |

In group I the mean hemoglobin values were 112.8±17.7; in group II 110.3±20.1; in group III 123.5±15.1 and in control 125.9±8.8. In group I the

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mean red blood cell counts were 3.8±0.6; in group II 3.6±0.7; in group III 4.1±0.5 and in control 4.2±0.5. Considering the above mentioned, it can be verified that the majority of PsA patients in groups I and II, in contrast to groups III and control groups had autoimmune anemia due to decreased hemoglobin and erythrocyte count. Leukocytosis or leukopenia was not observed in all study groups.

In all study groups, acute phase proteins - CRP, rheumatoid factor - RF and erythrocyte sedimentation rate - ESR were interpreted to determine the inflammatory process (Table 2).

**Table 2**
**Evaluation of the inflammatory process in I, II, III and control study groups (n=145)**

| Parameters of inflammatory markers | Control | I | II | III | p |
|---|---|---|---|---|---|
| CRP mg/l | 2,4±1,5 | 28,5±7,6 | 19,1±3,9 | 8,9±1,5 | p>0,05 |
| RF | negative | negative | negative | negative | - |
| ESR mm/hour | 5,2±2,1 | 32,2±8,1 | 14,3±2,6 | 4,2±1,1 | p>0,05 |

In study group I, the mean CRP values were 28.5±7.6; in group II, 19.1±3.9; in group III, 8.9±1.5; and in the control group, 2.4±1.5. In all study groups, patients were RF seronegative. In study group I, the mean values of ESR were 32.2±8.1; in group II, 14.3±2.6; in group III, 4.2±1.1 and in control group, 5.2±2.1. Based on the significant increase of CRP and ESR indices it was revealed active inflammatory process due to PsA in patients in I and II studied groups, in contrast to III and control groups.

In all studied groups, lipid spectrum was interpreted, which included HDL, LDL, TG, TCH and atherogenicity index - AI according to the formula (Table 3).

**Table 3**
**Evaluation of lipid spectrum and atherogenicity index in I, II, III and control study groups (n=145)**

| Parameters of the lipid spectrum | Control | I | II | III | p |
|---|---|---|---|---|---|
| HDL mmol/l | 1,5±0,1 | 0,8±0,1 | 1,1±0,1 | 0,7±0,1 | p>0,05 |
| LDL mmol/l | 2,2±0,1 | 4,5±0,3 | 2,9±0,3 | 4,6±0,4 | p>0,05 |
| TG mmol/l | 0,5±0,1 | 2,3±0,2 | 1,1±0,2 | 2,2±0,3 | p>0,05 |
| TCH mmol/l | 3.5±0,5 | 6.5±1,1 | 4.8±0,9 | 6,6±1,1 | p>0,05 |
| AI | 3,1±0,1 | 4,6±0,3 | 3,5±0,2 | 4,5±0,2 | p>0,05 |

In study group I, mean HDL values were 0.8±0.1; in group II, 1.1±0.1; in group III, 0.7±0.1 and in control, 1.5±0.1. In study group I, mean LDL values were 4.5±0.3; in group II, 2.9±0.3; in group III, 4.6±0.4 and in control, 2.2±0.1. In study group I, the mean TG values were 2.3±0.2; in group II, 1.1±0.2; in group III, 2.2±0.3 and in the control group, 0.5±0.1. In study group I, mean TCH values were 6.5±1.1; in group II, 4.8±0.9; in group III, 6.6±1.1 and in the control group, 3.5±0.5. In group I of the study, the mean AI figures were 4.6±0.3; in group II, 3.5±0.2; in group III, 4.5±0.2 and in the control group, 3.1±0.1. Based on these figures, it can be said that HDL was below normal in groups I and III, in contrast to groups II and control groups. LDL, TG, TCH and AI was higher than normal in groups I and III, in contrast to groups II and control groups. Analyzing the data we can say that the risk of atherosclerosis was significantly higher in groups I and III than in patients of groups II and control groups.

Coagulogram was analyzed in all groups - prothrombin time (PTT), prothrombin index (PTI), fibrinogen, international normalized ratio (INR), activated partial thromboplastin time (APTT) (Table 4).

**Table 4**
**Evaluation of coagulogram in I, II, III and control study groups (n=145)**

| Coagulogram parameters | Control | I | II | III | p |
|---|---|---|---|---|---|
| PTT second | 15,9±0,4 | 11,7±0,3 | 12,2±0,5 | 11,9±0,4 | p>0,05 |
| PTI % | 98,5±5,5 | 112,2±4,7 | 104,1±7,1 | 111,2±5,1 | p>0,05 |
| Fibrinogen g/l | 3,1±0,4 | 5,1±0,6 | 4,4±0,3 | 5,1±0,4 | p>0,05 |
| INR | 1,1±0,2 | 0,9±0,1 | 1,1±0,1 | 0,9±0,2 | p>0,05 |
| APTT second | 31,6±3,1 | 21,4±2,1 | 24,2±0,7 | 21,2±0,9 | p>0,05 |

In study group I, the mean PTT values were 11.7±0.3; in group II, 12.2±0.5; in group III, 11.9±0.4 and in control, 15.9±0.4. In study group I, the mean PTI

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values were 112.2±4.7; in group II, 104.1±7.1; in group III, 111.2±5.1 and in the control group, 98.5±5.5. In study group I, the mean fibrinogen values were 5.1±0.6; in group II, 4.4±0.3; in group III, 5.1±0.4 and in the control group, 3.1±0.4. In study group I, the mean INR values were 0.9±0.1; in group II, 1.1±0.1; in group III, 0.9±0.2 and in the control group, 1.1±0.2. In group I of the study, the mean values of APTT were 21.4±2.1; in group II, 24.2±0.7; in group III, 21.2±0.9 and in the control group, 31.6±3.1. Based on the data of the study, it was found that PTT, INR and APTT were lower in groups I and III, in contrast to groups II and control groups, and PTI and fibrinogen were significantly higher in groups I and III, in contrast to groups II and control groups. Analyzing the above-mentioned indices revealed the presence of hypercoagulability and high risk of thrombosis in I and III studied groups, in contrast to II and control groups.

In all groups, blood biochemical tests were performed according to the standards - blood glucose and uric acid concentration (Table 5).

**Table 5**
**Evaluation of glucose and uric acid in I, II, III and control study groups (n=145)**

| **Parameters of biochemistry** | **Control** | **I** | **II** | **III** | **p** |
|---|---|---|---|---|---|
| **Glucose mmol/l** | 4,1±0,7 | 4,4±0,6 | 4,3±0,7 | 4,7±0,4 | p>0,05 |
| **Uric acid mg/dl** | 4,1±0,6 | 7,2±0,7 | 5,1±0,9 | 7,1±0,6 | p>0,05 |

In study group I, the mean glucose values were 4.4±0.6; in group II, 4.3±0.7; in group III, 4.7±0.4 and in control, 4.1±0.7. In study group I, the mean uric acid values were 7.2±0.7; in group II, 5.1±0.9; in group III, 7.1±0.6 and in the control group, 4.1±0.6. In all studied groups, blood glucose was within the normal range and uric acid was at the upper limit of normal in patients of group I and III, in contrast to group II and control groups.

In all groups, specific blood tests were performed to determine proinflammatory cytokine IL17 and endothelial damage marker ET1 by ELISA method (Table 6).

**Table 6**
**Evaluation of interleukin 17 and endothelin 1 in I, II, III and control study groups (n=145)**

| **Parameters of specific studies** | **Control** | **I** | **II** | **III** | **p** |
|---|---|---|---|---|---|
| **IL17 pg/ml** | 3,5±0,8 | 12,1±1,8 | 9,1±1,4 | 5,9±1,4 | p>0,05 |
| **ET1 pg/ml** | 11,6±3,4 | 51,7±12,4 | 30,1±6,6 | 45,5±7,8 | p>0,05 |

The mean values of proinflammatory cytokine IL17 in group I of the study were 12.1±1.8; in group II 9.1±1.4; in group III 5.9±1.4 and in control 3.5±0.8. The mean values of endothelial damage marker ET1 in group I were 51.7±12.4; in group II 30.1±6.6; in group III 45.5±7.8 and in control 11.6±3.4. The study showed that blood IL17 concentrations were significantly higher in group I (1.32 times higher than group II, 2.11 times higher than group III and 3.46 times higher than control group) and group II of the study (1.54 times higher than group III and 2.6 times higher than control group) in contrast to group III and control groups, and ET1 concentration was in higher amounts in I (1.14 times more than in group III, 1.72 times more than in group II, 4.46 times more than in control group) and III study groups (1.51 times more than in group II, 3.92 times more than in control group) in contrast to II and control groups ($p<0.05$). Analysis of the above data suggests a pronounced inflammatory process in I and II study groups and pronounced endothelial dysfunction in I and III study groups, compared to control groups.

**DISCUSSION OF THE STUDY RESULTS.** When laboratory data were processed, autoimmune anemia was observed due to decreased hemoglobin and erythrocyte count in the majority of PsA patients in groups I and II, in contrast to groups III and controls. Leukocytosis or leukopenia was not observed in all studied groups. There was also an increase in CRP and ESR due to active inflammatory process of PsA in patients of groups I and II, in contrast to groups III and control groups.

When assessing the lipid spectrum, it was revealed that HDL was lower than normal in groups I and III, in contrast to groups II and control groups. LDL, TG, TCH and AI was above normal in groups I and III, in contrast to groups II and control groups. Analyzing the data we can say that in groups I and III the risk of atherosclerosis was significantly higher than in patients of groups II and control groups.

Evaluation of coagulogram of patients of all studied groups showed that PTT, INR and ATTP were lower in I and III groups, in contrast to II and control groups, and PTI and fibrinogen were significantly higher in I and III groups, in contrast to II and control groups. Analyzing the above indices revealed the presence of

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hypercoagulability and high risk of thrombosis in I and III studied groups, in contrast to II and control groups.

When examining biochemical laboratory data, it was revealed that blood glucose was within normal limits, and uric acid was at the upper limits of normal in patients of groups I and III, in contrast to groups II and control groups.

Evaluation of proinflammatory cytokines and markers of endothelial damage showed that blood concentrations of IL17 were significantly higher in I (1.32 times more than in group II, 2.11 times more than in group III and 3.46 times more than in the control group) and II study groups (1.54 times more than in group III and 2, 6 times more than in the control group) in contrast to III and control groups, and the concentration of ET1 was in greater amounts in I (1.14 times more than in group III, 1.72 times more than in group II, 4.46 times more than in the control group) and III study groups (1.51 times more than in group II, 3.92 times more than in the control group) in contrast to II and control groups ($p<0.05$). Analysis of the above data suggests a pronounced inflammatory process in I and II study groups and pronounced endothelial dysfunction in I and III study groups, compared to control groups.

IL17 is a cytokine that plays a crucial role in the pathogenesis of PsA. ET1 is a peptide that regulates various physiologic processes, including regulation of blood vessel constriction and inflammation. It is hypothesized that there is a potential link between IL17 and ET1 in the context of PsA. Presumably IL17 can induce the production and release of ET1 by endothelial cells and other cell types. This increased expression of ET1 may contribute to the inflammatory processes and blood vessel dysfunction observed in PsA. IL17 and ET1 exert pro-inflammatory effects on the joints of patients with psoriatic arthritis. IL17 promotes the recruitment of immune cells and stimulates the production of other cytokines, leading to chronic inflammation. ET1, on the other hand, promotes the migration of inflammatory cells and enhances the production of pro-inflammatory molecules.

IL17 induces angiogenesis, the formation of new blood vessels, which is a hallmark of psoriatic arthritis. ET1 is involved in various vascular processes, including vasoconstriction and angiogenesis, and its increased expression may contribute to the abnormal vascular changes seen in PsA. Likewise, IL17 and ET1 are associated with the joint remodeling and tissue damage seen in PsA. IL-17 stimulates the production of matrix metalloproteinases (MMPs) that damage joint tissues, whereas ET1 promotes the synthesis of collagen-degrading enzymes that lead to joint destruction. Targeting IL17 and ET1 pathways may have therapeutic potential in PsA. Several biologic drugs targeting IL17 or its receptor may show efficacy in reducing joint inflammation and skin lesions in patients with PsA. In addition, drugs that target the ET1 pathway, such as endothelin receptor antagonists, have been investigated as potential treatments for SSRIs in PsA. It is important to note that although studies suggest a link between IL17 and ET1 in PsA, the exact mechanisms and interactions between these molecules are still under investigation. Further studies are needed to fully understand the effect of IL-17 on ET-1 and its impact on pathogenesis and early diagnosis and prognosis of SWD in PsA [9,10].

**CONCLUSION.** Thus, the laboratory data showed autoimmune anemia due to decreased hemoglobin and erythrocyte count in the majority of PsA patients in groups I and II, in contrast to groups III and control groups. There was also an increase in SRP and ESR indices due to active inflammatory process of PsA in patients of groups I and II, in contrast to groups III and control groups. Analyzing the lipid spectrum we can say that in groups I and III the risk of atherosclerosis development was significantly higher than in patients of groups II and control groups. Coagulogram indicators revealed the presence of hypercoagulability and high risk of thrombosis in I and III studied groups, in contrast to II and control groups. Analysis of IL17 and ET1 indicates a pronounced inflammatory process in I and II study groups and pronounced endothelial dysfunction in I and III study groups, in comparison with control groups.

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