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THE STATE OF HEMATOLOGICAL PARAMETERS IN APLASTIC ANEMIA

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Summary

Purpose of the study. To analyze laboratory changes in peripheral blood and myelogram in adult patients with aplastic anemia.

Methods. The material for clinical and laboratory studies in the work were patients with AA (n=86) who sought diagnostic help and subsequent inpatient examination at the republican specialized Scientific and Practical Medical Center of Hematology (RSNPMCG, Tashkent) from 2019 to 2023. Patients with AA ranged in age from 18 to 79 years, while the median age was 40.8±1.8 years. The diagnosis was made taking into account clinical and laboratory data.

The research methods included laboratory examination (general blood test (UAC) and myelogram) and statistical methods of processing the results using the PC application package "OpenEpi 2009, Version 2.3".

Conclusions. Despite the severity of form A, UAC is characterized by a decrease in hemoglobin concentration, the number of erythrocytes, platelets, and leukocytes due to neutropenia, lymphocytosis, and acceleration of ESR. The myelogram shows a decrease in the cellularity of the red bone marrow, three-stage cytopenia and replacement of the red brain with a fatty brain.

Key words: UAC, myelogram, three-stage cytopenia, fatty brain.

Introduction. AA is a rare type of bone marrow insufficiency syndrome (BCM), which is characterized by severe pancytopenia and bone marrow hypoplasia of varying severity [1,2,4]. Acquired AA, also called idiopathic AA, accounts for the majority (\sim 70%) of all newly diagnosed cases [5,7,10].

The diagnosis of aplastic anemia should be suspected in any patient presenting with pancytopenia. Aplastic anemia is a diagnosis of exclusion [3].







The severity of AA was assessed in accordance with the parameters of the blood test and the results of the bone marrow examination. Severe AA (SAA) was defined as a BM cell count of < 25% or 25-50% with < 30% residual hematopoietic cells and at least two of the following: (I) absolute neutrophil count < 0.5×109 /l, (II) platelets < 20×109 /L and (III) the number of reticulocytes < 20×10 9 /l . Patients with AA who did not meet the SAA criteria were classified as non-severe AA (NSAA). The result of the treatment was based on previous literature [6,8,9].

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Results. In patients with AA, a decrease in hemoglobin concentration of cytopenia (decrease in the number of erythrocytes, platelets and leukocytes), absolute neutropenia, relative lymphocytosis and acceleration of ESR were found in clinical blood analysis compared with healthy ones.

Analyzing the average values of the indicators of the general blood test in the main group of patients with AA in comparison with the control, the changes characteristic of the disease manifested by cytopenia were established. In patients with AA, there was a significant decrease in hemoglobin by 2.5 times $(52.8\pm1.7 \text{ g/l versus } 134.2\pm2.4 \text{ g/l; P<0.01})$ and erythrocytes by 2.0 times $(1.7\pm0.07 \times 1012/l \text{ versus } 3.4\pm0.5 \times 1012/l; P<0.05)$.

At the same time, the median platelet count was statistically significantly reduced by 21.1 times (13.6 \pm 3.0 x 1012/l versus 287.2 \pm 2.1 x 1012/l; P<0.001), and leukocytes by 6.2 times (1.2 \pm 0.05 x 1012/l versus 7.4 \pm 1.2 x 1012/l; P<0.001).

The above data show some of the main changes in the UAC characteristic of AA. Meanwhile, the key factor in the UAC for confirming the diagnosis of AA was a decrease in the absolute number of neutrophils among patients by 11.2 times



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compared with the control ($0.423\pm0.03 \times 1012/l$ versus $4,743\pm0.25 \times 1012/l$; P<0.001).

A 1.7-fold increase in the number of lymphocytic cells was also specific for this pathology ($61.0\pm1.1\%$ vs. $35.5\pm1.4\%$; P<0.05), as well as a 7.2-fold increase in the level of ESR (52.0 ± 2.1 mm/h vs. 7.2 ± 1.4 mm/h; P<0.001)

Thus, the laboratory signs of AA in the UAC were normocytic anemia, severe cytopenia (erythro-, thrombo- and leukopenia), neutropenia, relative lymphocytosis and accelerated ESR. Moreover, these signs differed in severity depending on the severity of AA, respectively, having deeper disorders in severe and superheavy forms, which were associated with the severity of clinical manifestations in patients with AA.

The results of morphological and quantitative analysis of the bone marrow showed a picture of small cells in the normoblastic type of hematopoiesis with a content of $0.18\pm0.009\%$ blast cells in the main AA group, which showed no increase in their content.

Microscopy revealed fatty voids, as well as single areas containing reticular, erythroid and lymphoid cells. The sum of erythroid elements was reduced to $6.4\pm0.2\%$ of their minimum normal number by 2.3 times (14.5%; P<0.01), and from the maximum by 4.1 times (26.5%; P<0.001). The number of lymphocytes, which amounted to 24.3 $\pm0.4\%$ of the maximum normal number, was increased by 1.8 times (13.7%; P<0.05), and from the minimum allowable by 5.6 (4.3%; P<0.001).

Meanwhile, the content of plasma cells, eosinophils, basophils and monocytes were within the reference norms (see Table 3.13). While the number of neutrophils and megakaryocytes (MCC) significantly decreased below the permissible normal values by 2.0 (26.1-1.0% vs. 52.7%; P<0.05) and $2.5 \text{ times} (2.0\pm0.1 \text{ against } 5; \text{P}<0.01)$, respectively.

Thus, the myelogram picture was characterized by bone marrow hypocellularity, manifested by three-stage cytopenia, narrowing of granulocytic and erythroid sprouts with normoblastic type of hematopoiesis, the presence of voids and the replacement of the red brain with yellow. All these signs are key characteristics of AA.

Conclusion. In the UAC, all degrees of severity of AA are characterized by a decrease in hemoglobin concentration, the number of erythrocytes, platelets, and leukocytes due to neutropenia, lymphocytosis, and acceleration of ESR. The myelogram shows a decrease in the cellularity of the red bone marrow, three-stage cytopenia and replacement of the red brain with a fatty brain.



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