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BLOOD MEASURES IN PATIENTS WITH COVID-19

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ABSTRACT
Coronavirus infection mainly affects the respiratory tract, but according to the latest information, COVID-19 is a systemic disease, affecting the respiratory, cardiovascular, gastrointestinal, urinary, neurological, hematopoietic and immune systems. Purpose of research is evaluation of blood and urine analysis. Research material on COVID-19 with 200 sick patients. Research methods: general blood analysis. The degree of leukocytosis, neutrophilia and lymphocytopenia depends on the extent to damage the lungs in coronavirus infection.

Keywords: COVID-19, blood, leukocyte, neutrophil, monocyte, lymphocyte, platelets.

Introduction

The importance of study. The infection of COVID-19 has led to a pandemic that has affected millions of people around the world. The Chinese Center for Disease Control and Prevention confirmed that this condition was caused by a new type of beta-coronaviruses after examining throat swabs from patients [25]. The coronavirus has been found to cause various diseases, including respiratory, intestinal, nervous and liver diseases.

While the coronavirus infection mainly affects the respiratory tract, according to the latest data, COVID-19 is a systemic disease that affects the respiratory, cardiovascular, gastrointestinal, urinary, neurological, hematopoietic and immune systems [16]. The pathogenesis of the new coronavirus infection has not been studied to the end, the information on the epidemiology, clinical features, prevention and treatment of the disease is constantly being updated [17 , 26]. According to the study of Assiri et al., a decrease in the number of platelets, leukopenia was noted in patients infected with COVID-19 [15].

In patients with coronavirus infection, symptoms of severe hypercoagulation are observed at all stages of hemostasis [10]. At the same time, in patients with COVID-19, platelet adhesion and aggregation activity increases, retraction time decreases. Platelet activity changes are related to the severity of COVID-19 [11].

Platelet activity does not reliably change in patients with mild coronavirus infection, while in moderate and severe coronavirus infection, platelet aggregation properties increase by 23-36%, and adhesion activity increases by 60-98%. This indicates that platelet hemostasis is shifted towards hypercoagulation [13]. Severe coronavirus infection is mainly observed in elderly

patients, and these patients have a high risk of developing thromboembolic complications [18]. Thromboembolic complications are mainly observed in the blood vessels of the heart and brain [2, 3, 12]. Patients with COVID-19 experience disability and death due to thromboses in vital organs [10, 11]. This is the result of Sars-CoV-2 induced endotheliocyte alteration and cytokine storm [13].

The spread of coronavirus in the body causes the development of a hyperimmune reaction - "cytokine storm": a large number of inflammatory interleukins, including S-reactive protein, serum ferritin, lactate dehydrogenase, D-dimer, 1-beta, 6, 2 interleukins, tumor necrosis factor and chemokines are produced [19].

In order to more accurately diagnose the state of thrombophilia, the study of the MTHFR gene A1298C (rs 1801131), C677T (rs 1801133) polymorphisms in the Uzbek population, and the significance of the C (rs 1801131) and T (rs 1801133) minor alleles of this gene in the pathogenesis of COVID-19 and hyperhomocysteinemia It is important to assess the extent of the disease of COVID-19 in observed patients [4, 8, 9]. By identifying this, various severe complications that can be caused by COVID-19 can be prevented by carrying out special preventive and therapeutic practices [5, 21, 22, 23].

Application of modern molecular diagnosis of genetic risk factors of thrombophilia to various fields of clinical medicine is very important for the prevention of many complications caused by thrombosis [14, 24].One of the main causes of death in coronavirus infection is the development of hypercoagulation, the development of thrombophilia and the increased risk of thrombosis [6, 7]. The development of pulmonary artery thromboembolism (PATE) in COVID-19 causes a decrease in blood circulation, increased tension in the right ventricle, increased troponin levels, development of cardiogenic shock, and short-term death [20].

Blood tests are important for doctors for early diagnosis of the disease, they provide information such as the inflammatory process, organ damage (kidney failure, liver failure), help to assess the severity of the disease. An increase in leukocytes, neutrophils or lymphocytes, and an increase in inflammatory markers (S-reactive protein) are characteristic of the inflammatory process. In addition, platelets also play an important role in the management of various inflammatory processes.

Purpose: in coronavirus infectionstudy of hematological changes.

Material and Methods

As an object of investigation, medical histories of 200 patients treated with COVID-19 at the Tashkent Medical Academy were retrospectively examined. Patients were divided into the following groups: Group 1 - 50 (25%) patients with mild COVID-19, Group 2 - 96 (48%) patients with moderate COVID-19, and Group 3 - 54 (27%) patients with severe degree of COVID-19. The control group consisted of 30 age- and sex-matched healthy individuals. Methods: General blood analysis with leukoformula in Hematological analizator MINDRAY BC-5000 with reagents of HUMAN (Germany).

Result

During the incubation period and early stage of coronavirus infection, peripheral blood parameters showed normal values. Lung damage was not detected in 1 group of patients, leukocytosis was more than 8.4 ± 2,1 x 10^9/l** and neutrophilia with rod end segmented nucleus neutrophil (RNN and SNN) was 74±6,8 %, and lymphocytopenia was 15±1,2%*** , monocytosis was 11±1,2%**.

In group 2, patients infected with the corona virus in the middle severe stage showed respiratory failure and 20% of patients with lung damage, patients had leukocytosis 16±1.3x10^9/l***, neutrophilia 80±8,2%**, lymphocytopenia 14±2,5%*** and monocytes 6±0,5% (Table 1).

Table 1 HEMATOLOGICAL INDICATIONS IN COVID-19

Groups	Leukocyte, x10 ^{9} /l	Myelocyte, %	Metamie-locyte,%	RNN and SNN neutrophil , %	Lymphocyte, %	Monocyte, %
Control group (n=30)	6.8±0.6	-	-	62±4.6	30±2,4	8±0.6
Group 1 (n=50)	8.4±2,1**	-	-	74±6,8	15±1,2***	11±1,2**
Group 2 (n=96)	16±1.3***	-	-	80±8,2**	14±2,5***	6±0,5
Group 3 (n=42)	19.4±4.8***	-	-	82±9.2***	12±2.4***	6±0,4
Group 3 (n=12)	25.8±3.4***	2 ±0.3	5 ± 0.4	78±7.4***	8±1.7***	7±0,6

Note: * - the differences between the indicators of the examined control group and patients with COVID-19 are reliable ($p < 0.05$), ** - $p < 0.01$ and *** - $p < 0.001$.

In 42 patients with severe coronavirus infection of group 3, with more than 50% lung damage, leukocytosis was 19.4±4.8 x10^9/l***, neutrophilia was 82±9.2%***, lymphocytopenia was 12±2.4%*** and monocytes 6±0,4%. In 12 patients of group 3, leukocytosis was 25.8±3.4 x10^9/l ***, neutrophilia was 78±7.4%***, leukoformula unchanged it happened. myelocytes 2 ± 0.3% and metamyelocytes 5 ± 0.4% appeared, lymphocytopenia was 8±1.7%*** and monocytes 7±0,6% (fig. 1).

In the control group, the number of leukocytes was 6.8±0.6 x10^9/l, neutrophiles was 62±4.6%, lymphocytes was 30±2,4% and monocytes was 8±0,6%.

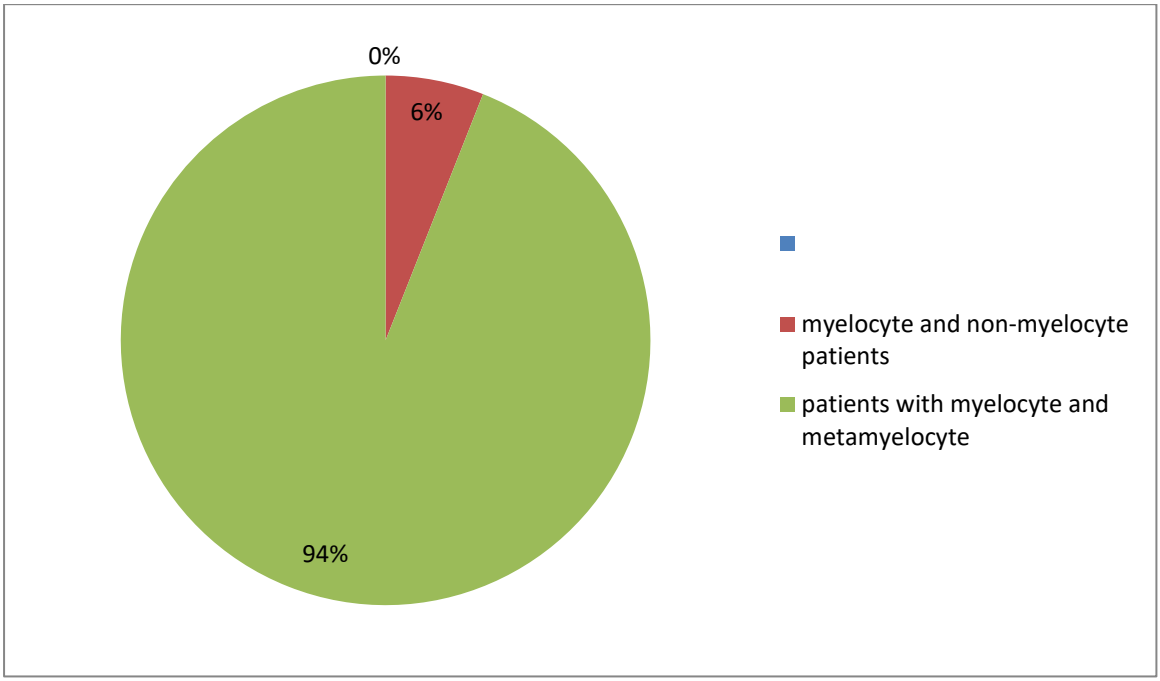

Figure 1. Leukoformula shift to the left in group 3 patients.

According to the study, the number of platelets in group 1 was 229±33 x10^9/l, in group 2 it was 282±38 x10^9/l, and in group 3 it was found to be 296±42 x10^9/l· The average number of platelets in 8 patients in 3 groups was 102 ± 11 x10^9/l***. The average number of platelets in the control group was 256±39 x10^9/l (Fig. 2).

Mechanisms of development of thrombocytopenia in COVID-19 are associated with inflammation and hypercoagulation. In this case, thrombocytopenia develops due to excessive destruction of platelets as a result of immune reactions in response to inflammation, consumption of platelets when many thrombi are formed, side effects of drugs. At the same time, congenital and acquired thrombocytopenias unrelated to coronavirus infection should also be taken into account.

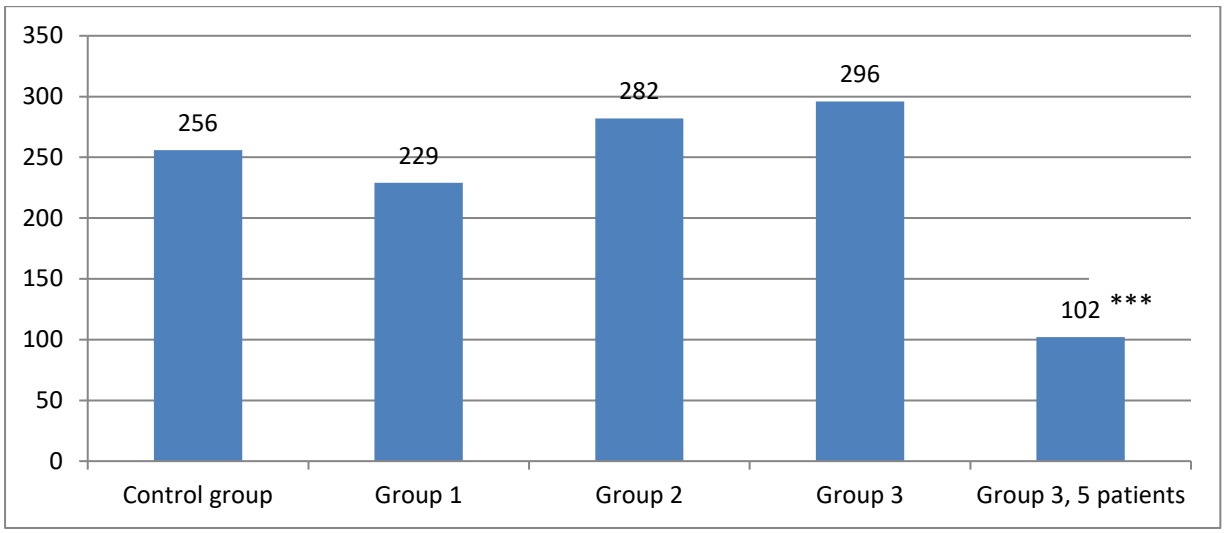

Figure 2. The number of platelets in COVID-19.

In conclusion, the number of platelets in most patients with coronavirus infection was normal, and it was found that the number of platelets decreased only in 5 patients with severe cases.

Summary:

1. The level of leukocytosis, neutrophilia, and lymphocytopenia in patients infected with COVID-19 depends on the extent to which the coronavirus infection has damaged the lungs.
2. The appearance of myelocytes and metamyelocytes in the leukoformula, and a decrease of lymphocytes below 10% were observed in severe cases of coronavirus infection.
3. In most patients with coronavirus infection, the number of platelets is normal, only 5 (10%) patients with a severe degree have been decrease in the number of platelets

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