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INDICATIONS OF COAGULOGRAM IN POST-COVID SYNDROME

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Coronavirus infection (COVID-19) is an acute infectious disease caused by the SARS-CoV-2 virus, which is manifested by the activation of the hemostasis system, which in severe cases can lead to the development of coagulopathy. Currently, it is not fully determined whether COVID-19 is the direct cause of these disorders or whether it exacerbates the infectious process. Asymptomatic development of COVID-19 and the development of clinically obvious thrombotic or thromboembolic complications remain unclear, which is mainly due to the difficulties in their diagnosis. Therefore, in the development of severe clinical forms of COVID-19, it is important to study the features of activation of the blood clotting system and, accordingly, to detect this disease early. Often, various clinical,COVID-19enough to solve a specific diagnostic and therapeutic problem during the treatment of patients with

In medicine, the concept of "post-covid syndrome", a new pathology with a unique clinical course, whose mechanism has not been fully revealed.concepts of post-covid syndrome first appeared in 2020.

Post-covid syndrome (from English-post-COVID-19 syndrome, Long COVID, post-acute sequelae of COVID-19 (PASC) chronic COVID syndrome (CCS). Post-covid syndrome is a condition in which the patient has experienced a coronavirus infection and the clinical symptoms last for more than 12 weeks. 2020 National Institute for Health and Research in England in December(NICE)- proposed the following classification of post-covid syndrome:

- acute COVID-19(if symptoms last up to a week);
- Persistently symptomatic COVID-19(if symptoms last 4-12 weeks;
- postcovid syndrome(symptoms persist for more than 12 weeks and disappear over time and many organs are damaged).

Also, in medicine, a new general term "long-lasting COVID" (iEnglish.long covid) began to be used. Symptoms likely to be observed in post-covid syndrome (constant or transient):

- ❖ Fatigue fatigue

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- Panting
- Inability to breathe fully
- A feeling of heaviness in the chest
- Myalgia
- Pain in the joints
- Loss of smell/nausea
- Hair loss
- Tooth loss
- Cystic changes in the upper and lower jaw
- Thyeri changes (vasculitis)
- Arrhythmia, tachycardia, dizziness
- Cognitive disturbances (fog in the head, disorientation, memory loss, panic)
- Gastrointestinal symptoms (diarrhea)
- Prolonged subfebrile body temperature increase, or hypothermia
- Also specific damage to organs and its symptoms.

Analysis of the current literature on the pathology of hemostasis in COVID-19. It is known that in the post-Covid period of the disease, changes occur in the blood coagulation system. The results of the research show that routine coagulogram parameters are not specific enough to prevent thrombosis in these patients, and as a result prescribe anticoagulant therapy. And so,coronavirusThe problem of monitoring complications in the period after requires further study.

COVID-19 can affect almost all organs, including the lungs, spleen, heart, blood vessels, gastrointestinal, urinary, nervous, hematopoietic, and immune systems [16, 3].

Pathogenetic aspects of COVID-19 are still being studied, the spread of infection, clinical manifestations, prevention and treatment of disease complications are changing [17].

The mortality rate was high in the early period of the disease, and later it was found that the main cause of mortality was the development of bleeding and thrombosis as a result of the activation of the blood coagulation system. In severe infectious diseases, the hemostasis system is activated and stops the spread of microorganisms in the body [13].

In COVID-19, severe hypercoagulation is observed in all joints of the blood coagulation system. Hypercoagulative changes in the blood coagulation system cause many thrombosis [5].

Among these, pulmonary embolism and deep vein thrombosis are the most

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common thrombotic complications, occurring in 25–30% of cases in critically ill patients. In patients with thrombotic complications, the death rate was found to be 5 times higher, and pulmonary embolism was observed mainly in the first 6 days of the disease [20].

Therefore, unlike other severe viral respiratory infections, COVID-19 is considered a severe thrombotic infectious disease [15].

89% of patients with severe and severe COVID-19 are aged 50-69, and these patients are at high risk of developing thromboembolic complications [6, 19].

Thromboembolic complications are more common when receiving antiaggregant and anticoagulant therapy. Such complications mainly occur in the blood vessels of the heart and brain. Pulmonary artery thromboembolism in COVID-19 causes pulmonary edema, increased pressure in the right ventricle of the heart, increased troponin levels, development of cardiogenic shock, and short-term death. In COVID-19, disability and death develop due to thrombosis and thromboembolism in vital organs. This is a consequence of endothelial cell alteration and "cytokine storm" induced by KI infection [5, 6].

In response to the impact of the SARS-Cov-2 virus on the human body, an excessive amount of inflammatory interleukins, S-reactive protein, serum ferritin, lactate dehydrogenase, D-dimer, 1-beta, 6-, 2- interleukins, tumor necrosis factor and chemokines are produced. As a result, a hyperimmune reaction - "cytokine storm" develops, damages the vascular endothelium, activates the blood coagulation system and develops blood burns and thrombosis [21].

Blood tests not only provide information about the inflammatory process, damage to internal organs (kidney, liver failure), but are also very important for determining the severity of the disease and determining treatment tactics. In COVID-19, the level of leukocytosis, neutrophilia and leukoformula shift to the left, lymphocytopenia depends on the degree of lung damage caused by COVID-19 [20].

In mild COVID-19, coagulation hemostasis indicators do not change reliably, but in moderate and severe COVID-19, active partial thromboplastin time by 25.5 - 39.5%, prothrombin time by 24 - 37% it was found that thrombin time was reduced by 22.3 - 45.2%, prothrombin index was increased by 35-62%, and fibrinogen was increased by 57.6 - 80.2%. A strong hypercoagulable change in plasma hemostasis was observed in moderate and severe KI [3].

At the same time, in accordance with the severity of COVID-19, the adhesive and aggregation function of platelets increases, the retraction time decreases [6]. In mild COVID-19, platelet activity is normal, and in moderate and severe KI, an increase in platelet aggregation by 23-36% and adhesion by 60-98% was found. D-

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dimer, a product of thrombus breakdown, increases dramatically in COVID-19. This indicates a hypercoagulable change in platelet hemostasis [1].

Anticoagulants are used to prevent thrombotic complications of COVID-19, but the tactics of anticoagulant therapy have not been fully refined according to the severity of the coronavirus infection.

The purpose of the study: In the post-covid syndrome evaluation of coagulogram indicators in dynamics.

Research materials and methods. Clinical studies were conducted in 2022-2023 in the treatment departments of the Multidisciplinary Medical Center of the Khorezm region. In the study, 45 patients with comorbidities who recovered from coronavirus infection were examined.

All examined patients were divided into 2 groups: 1st group consisted of 20 mild patients, 2nd group consisted of 15 moderately severe patients. The control group consisted of 10 healthy individuals matched for sex and age.

Postcovid syndromediagnostic indicators in the "Provisional recommendations for the treatment of patients with coronavirus infection" were taken for the diagnosis of All patients in the study were diagnosed with a positive result in SARS-Cov-2 virus markers immunoassay and polymerase chain reaction.

Demographic description by gender is given in Table 1.1. Out of 45 examined patients, 19 (42.2%) were women and 26 (57.7%) were men. The age of the patients in the study ranged from 25 to 70 years, with a mean age of 45.4 ± 10.3 years. Patients with additional diseases such as diabetes, oncological diseases, liver and kidney diseases were not included in the research group.

Research results.

In a clinical study, 45 patients with coronavirus infection were examined. To study parameters of plasma hemostasis, active partial thromboplastin time (FQTV), prothrombin time (PTV), prothrombin index (PTI), international normalized ratio (XNM), fibrinogen, thrombin time (TV), D-dimer and Moravis method blood clotting time (CVT) was determined. At the same time, ferritin and S-reactive protein, which are markers of inflammation, were also examined.

When FQTV was examined to study the 1st stage of plasma hemostasis in patients with KI, the following results were obtained: FQTV in group 1 was 25.2 ± 2.1 s, in group 2 20.2 ± 1.8 s*, in group 3 16.1 ± 1.4 s***. In the control group, this indicator was 27.4 ± 2.2 seconds. In conclusion, it was found that FQTV was reduced in groups 1 and 2, which indicates the activation of the blood coagulation system.

PTV, PTI, XNM were examined to study the 2nd stage of blood coagulation. In

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these groups, similar changes were detected in the examination of the PTV: in the 1st group, the PTV was 11.1 ± 0.9 s, in the 2nd group, 9.3 ± 0.7 s*, in the 3rd

in the group it was 8.2 ± 0.7 s**, and in the control group it was 12.0 ± 1.2 s.

PTI was determined by PTV based on the formula, and PTI in group 1 was 108 ± 8.8%, in group 2 129 ± 8.2%*, in group 3 150 ± 12.3%*, and in the control group 98.0 ± 6, made up 6%.

XNM is also an index of PTV, which shows how many times the blood has been diluted and is mainly needed for monitoring anticoagulant therapy. XNM was 1.0 ± 0.08 s in the control group, 0.92 ± 0.06 s in group 1, 0.77 ± 0.06 s in group 2, and 0.68 ± 0.05 in group 3. s*.

Determination of TV in KI also confirmed the hypercoagulable shift: TV in group 1 was 14.9 ± 1.1 sec, in group 2 11.2 ± 1.0 sec, in group 3 9.4 ± 0.7 sec, and in the control group the indicator was 22.6 ± 1.6 sec.

Fibrinogen is the 1st factor of plasma hemostasis, and its value was sharply increased in KI. Fibrinogen in group 1 was 3.86 ± 0.33 g/l, in group 2 4.62 ± 0.51 g/l*, in group 3 6.14 ± 0.68 g/l***, and in the control group It was found to be 2.54 ± 0.28 g/l (Table 1).Table 1.

Indicators of plasma hemostasis in KI

Groups	Control group (n=10)	group 1, (n=20)	group 2, (n=15)
FQTV, p	27.3 ± 2.4	25.2 ± 2.2	16.1 ± 1.4
PTV, s	12.0 ± 1.2	11.1 ± 0.9	8.2 ± 0.7 s**
PTI, %	98.0 ± 6.6	108 ± 8.7	150 ± 12.3*
XNM	1.0 ± 0.07	0.92 ± 0.06*	0.68 ± 0.05*
TV	22.6 ± 1.5	14.9 ± 1.1	9.4 ± 0.7
Fibrinogen	2.56 ± 0.28	3.86 ± 0.33	6.14±0.68***

Note: * - the difference compared to the control group is reliable (*-R<0.05; **-R<0.01; *** - R<0.001)

D-dimer as a thrombus degradation product has been found to be increased in KI, depending on the degree of thrombus formation and disintegration. D-dimer content in group 1 was 251 ± 20 ng/ml, in group 2 483 ± 32 ng/ml***, and in group 3665 ± 48 ng/ml***, while in the control group it was 196 ± 16 ng/ml.

Ferritin and C-reactive protein (SRO) are markers of inflammation, and their

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increase in KI reflects the degree of inflammation. In group 1, ferritin was 295 ± 31 µg/ml*, SRO was 8 ± 0.7 mg/l, while in group 2

ferritin 512 ± 42 µg/ml***, SRO 22 ± 1.8 mg/l, and in group 3 ferritin 784

± 65 µg/ml***, SRO was 62 ± 4.9 mg/l. In the control group, ferritin was 180 ± 16 µg/ml, SRO was 6.2 ± 0.4 mg/l.

Procalcitonin is a prohormone and is used as an early diagnosis of bacterial infection. Procalcitonin was 0.4 ± 0.03 ng/ml in group 1, 0.72 ± 0.05 ng/ml in group 2, and 1.4 ± 0.11 ng/ml in group 3, while control

group was 0.3 ± 0.02 ng/ml.

In conclusion, it can be said that group 1 patients with mild KI did not have significant changes in blood coagulation system and inflammatory markers, but group 2 patients with severe KI had a strong hypercoagulable tumor at all stages of plasma coagulation. changes and inflammatory processes were observed.Patients with concomitant diseasesfor the purpose of treatment, coronavirus base therapy was fully applied in accordance with the degree of the disease. Antiviral, antiaggregant, symptomatic treatment was carried out to the patients in accordance with the standards. In order to evaluate the effectiveness of treatment of hypercoagulation in the hemostasis coagulation system, patients of the 1st and 2nd groups were treated with anticoagulant therapy.in the background, by the 5th day XNM 1.06 ± 0.1*, by the 10th day 1.26 ± sec***organized.

As can be seen from the above data, treatment with Enoxiparin was more effective in normalizing the parameters of coagulation hemostasis compared to treatment with Heparin. At the same time, D-dimer, ferritin, and C-reactive protein levels were more effectively changed during treatment with Enoxiparin.

Conclusions.

1. The indications are that pre-hospital, in-hospital and post-hospital thromboprophylaxis therapy is indicated. When choosing the most appropriate pharmacological therapy, it is necessary to know the pharmacokinetic and pharmacodynamic properties of a certain drug, as well as its interaction with antiviral drugs. However, after the acute infection stage, it should be remembered that the patient's treatment is not complete. Post-COVID syndrome presents a challenge in daily practice for cardiologists and traumatologists, and its management must be tailored to the clinical characteristics of each patient.

2.Application of complex treatment with small molecular heparin in coronavirus infection led to effective elimination of hypercoagulation and reduction of days of inpatient treatment.

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