On March 11, 2020, the World Health Organization declared COVID-19 as a global pandemic. Today, the medications used to treat this disease are not well understood and many of them cause serious complications in patients.

A group of doctors GBUZ LO " Kirishskaya KMB", headed by candidate of medical Sciences (PhD), Chief Doctor of it by Dr. Stanislav Serafimov on the basis of analysis of scientific works of scientists from Russia, China, UK, Singapore and the United States has developed protocols and implemented treatment methods COVID-19 medium and severe degrees of severity. Scientific article about the matter is published at [1].

The priority of treatment was chosen in two directions: the fight to prevent the development of "cytokine storm" and the fight against coagulopathy.

The main drug for the prevention of "cytokine storm" is low-molecular weight heparin enoxaparin sodium (Enixum®) in therapeutic doses. We use this drug for all patients who have SpO2 ≤ 93% (with COPD SpO2 ≤ 84%) without oxygen subsidies.

Remarkably, we noted a significant decrease in IL-6 levels (except in the group of patients with an increased indicator of quantitative procalcitonin).

Our observations are confirmed by our Chinese colleagues who analyzed electronic hospital records of patients with COVID-19 [2].

Given the significant reduction in IL-6 levels, we were able to almost completely stop using the drug Tocilizumab (Actemra®), the use of which can lead to a serious complication — sepsis.

An additional positive effect may still occur due to the connection of heparin with the Spike protein (S1 receptor) — thus "Enixum®" blocks the entry of the SARS-CoV-2 virus into the human cell.

The second problem that leads to the transfer of patients to a ALV is coagulopathy, which manifests itself in massive vascular microthrombosis and pronounced hypoxia in the tissues.

Hypoxia is confirmed by the following parameters: severe weakness, severe headache and muscle pain, increased D-dimer, AST, LDH. After reading the article [3]. We are interested in using Famotidine for the treatment of our patients.
Studying the instructions for the use of Famotidine, we noted that it affects the formation of prothrombin.

When the virus damages the endothelium, a large amount of prothrombin is released, which contributes to massive intravascular blood clotting.
In some patients, despite the use of low-molecular-weight heparin in therapeutic doses, we observed a sharp increase during the day of D-dimer > 3000 ng / ml and a decrease in SpO2 saturation <94% - on oxygen subsidies, which indicates a poor prognostic indicator for the patient and a possible transfer of the patient to invasive artificial ventilation in the near future.

We used these patients intravenously Famotidine in a dosage of 40 mg 2 times a day and intravenous Verapamil in a therapeutic dose within an hour after the second administration of Famotidine (in order to relieve vascular spasm). As a result, after a day, we received a decrease

in the D-dimer index from > 3000 ng / ml to 300-400 ng / ml and an increase in SpO2 >95% on oxygen subsidies.

We assume the following mechanism of action of drugs (see figure). Using enoxaparin sodium, we reduce the release of Willebrand factor from the vascular endothelium and block Ha-activity. Famotidine blocks the production of prothrombin, a deficiency of which blocks the transition of fibrinogen to fibrin. Thus, the fibrin in the blood decreases sharply, which is confirmed by a decrease in the D-dimer. In parallel, plasmin increases naturally in the blood, which dissolves intravascular blood clots. This is how endogenous thrombolysis occurs.

No less surprising was the second fact that we noticed after two days of using Famotidine in our clinic. Against the background of treatment with enoxaparin sodium after the use of Famotidine at a dosage of 40 mg 2 times a day per os in patients requiring oxygen subsidies and patients from risk groups, the number of patients with C-reactive protein>70mg/l decreased by 2 times. We consider it appropriate to prescribe all patients when receiving Famotidine 40 mg 2 times a day orally against the background of the use of enoxaparin sodium. In this way, we will reduce the number of patients in Intensive care units (ICU) and the use of AVL.

We suspect that severe COVID-19 pneumonia causes respiratory failure through pulmonary vessel microthrombs and endothelial dysfunction. Treatment of COVID-19 pneumonia may require thrombolysis (Actilyse®) in extremely severe cases of the disease in order to prevent a shock lung. This hypothesis is shared by our USA colleagues[4].

Our recommendation: all patients entering the hospital at risk or requiring oxygen subsidies should be prescribed enoxaparin sodium 120 mg (160 mg) per day and Famotidine 80 mg per day.

Today, according to the scheme enoxaporin sodium 120 mg per day (160 mg)+ Famotidine 80 mg per day were treated more than 300 people they all have objective evidence of the effectiveness of treatment based on a significant decrease in the blood D-dimer!

Using the treatment regimen enoxaparin sodium + Famotidine, it is possible to treat patients with COVID-19 in an outpatient setting and prevent hospitalization in hospitals!!!