The use of the comparison drug diclofenac sodium had a more pronounced inhibitory effect on the cyclooxygenase pathway of conversion of arachidonic acid and, thus, reduced the intensity of synthesis of prostaglandins and other mediators in the inflammatory focus by 76%. Quercetin was significantly inferior to diclofenac sodium and alcoholic extracts of the Aristolochia clematitis on this model of inflammation. In our opinion, this phenomenon is explained by the absence in the mechanism of anti-inflammatory action of Quercetin of influence on COX-dependent link of inflammatory reaction as this means influences, first of all, a lipoxygenase way of transformation of arachidonic acid and leukotriene synthesis.

Conclusions. 1. The anti-inflammatory effect of four extracts from different vegetative parts of the Aristolochia clematitis was studied for the first time.

2. It was found that at the studied dose (35 mg/kg) extracts of the Aristolochia clematitis had an inhibitory effect on the cyclooxygenase pathway of arachidonic acid conversion and, thus, their use helped to reduce the synthesis of prostaglandins and other mediators in the inflammatory focus and led to anti-inflammatory effects.

3. In a number of the presented extracts at a dose of 35 mg / kg with the severity of the anti-inflammatory effect, the extracts can be arranged in the following order: alcohol extract from the aboveground part of the flowering Aristolochia clematitis, extraction 70% ethanol > alcohol extract from the aboveground part of the flowering Aristolochia clematitis, extraction 96% ethano l> aqueous extract from the roots of flowering Aristolochia clematitis > aqueous extract from the aboveground part of the flowering Aristolochia clematitis.

PATHOGENESIS OF CORONAVIRUS-INDUCED COAGULOPATHY IN COVID-19

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Introduction. Hypercoagulation can be viewed from the point of view of the Virchow triad. All three factors contributing to the development of thrombosis are also found in patients with severe COVID-19.

Endothelial cell damage. There is evidence of the direct introduction of the SARS-CoV-2 virus into endothelial cells, which can potentially lead to their damage. It has been hypothesized that endothelial injury, microvascular inflammation, endothelial exocytosis and/or endotheliitis play a major role in the pathogenesis of acute respiratory distress syndrome and organ failure in patients with severe COVID-19. Other observations suggested that neutrophil extracellular traps (NETs), a form of decondensed chromatin from dead or dying neutrophils, are involved in the prothrombotic state in COVID-19. Another source of endothelial damage is mediators of acute inflammatory reactions such as cytokines (for example, IL-6) and other factors of the acute phase of inflammation. Complement-dependent endothelial damage also contributes to it.

Violation of blood flow. Prolonged immobility can lead to hemostasis in any patient, regardless of whether or not they have COVID-19.

Hypercoagulation. Changes in several circulating prothrombotic factors are known in severe COVID-19 patients: increased factor VIII levels; increased levels of fibrinogen; circulating prothrombotic microparticles; neutrophil extracellular traps (NETs); increased blood viscosity.

Aim. Study of indicators of the hemostasis system in patients with severe COVID-19.

Materials and methods. The indicators of the blood coagulation system were studied in a series of cases in 29 patients in an extremely serious condition who were being treated in the intensive care unit of the Kharkiv Regional Clinical Infectious Disease Hospital. Studies were performed on arterial blood, as arterial catheters were placed in the patients. Since most patients were receiving low molecular weight heparin, heparinase was used.

Results and discussion. In all cases, a significantly increased level of D-dimer was observed (median values Me (LQ, UQ) - 4487 ng/ml (1197.00; 16954.00), correlating with the severity of the disease. D-dimer - represents fragments of the fibrin molecule formed during its decay (proteolytic degradation) under the action of plasmin The presence of a D-dimer in blood plasma indicates the formation and degradation of a fibrin clot within the vascular bed and reflects the activation of both hemostasis and fibrinolysis.

Prothrombin time and activated partial thromboplastin time were normal or slightly lengthened.

For viral infections, the formation of antiphospholipid antibodies is characteristic, under the influence of which an extension of the activated partial thromboplastin time can occur and does not mean antiphospholipid syndrome.

The platelet count is normal or slightly increased (an average of 348 thousand units/μl).

The fibrinogen content is increased: on average 680 mg/dl (range 234-1344 mg/dl).

The von Willebrand factor antigen content is significantly increased (mean 529, range 210–863), which corresponds to endothelial damage.

We studied the thromboelastography indices and obtained the following results:

- the reaction time is shortened due to the early formation of a significant amount of thrombin - "thrombin burst", which is observed in 50% of patients;
- the time of clot formation is shortened in 83% of patients (increased fibrin formation);
- the maximum amplitude is increased in 83% of patients (increased clot strength);
- the thrombus lysis index after 30 minutes is reduced by 100%, which indicates a decrease in the activity of fibrinolysis.

The predominant variants of coagulation disorders in patients with COVID-19 suggest the development of hypercoagulability, which is consistent with data from uncontrolled clinical studies indicating an increased risk of venous thromboembolism.

Some experts refer to the increased risk of venous thromboembolism in COVID-19 as "thrombotic inflammation" or COVID-19-associated coagulopathy (CAC). This condition is significantly different from disseminated intravascular coagulation syndrome (DIC), although DIC has been reported in some patients with severe COVID-19.

Conclusion. The severe course of COVID-19 is associated with increased blood clotting, which can lead to thromboembolic complications. One of the most important elements of therapy should be timely adequate anticoagulant prophylaxis and effective treatment of acute thromboembolic complications. Some markers of changes in the coagulation process, for example, D-dimer, correlate with the severity of the disease, a high concentration of which is a predictor of death.