DASS 42 questionnaire than men, and a higher frequency of anxiety (40 and 18%, respectively, p <0.001) and signs of stress (28 and 13%, respectively, p = 0.003). The analysis did not reveal a statistically significant dependence of scale scores and the frequency of psychoemotional disorders on age and place of residence. The identified high frequency of pathological emotional states requires further research. The questionnaire showed reliability and construct validity, the absence of cross-cultural differences in the perception of test questions among different groups of the population of the Republic of Sakha (Yakutia). The results of the study of the psychometric characteristics of the DASS-42 questionnaire indicate the possibility of its use as a tool for screening depression, anxiety and stress among the population of Yakutia.

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The authors declare no conflict of interest.

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FATTY LIVER DISEASE AS A RISK FACTOR FOR HEMOSTASIS DISORDERS IN PATIENTS WITH COVID-19

Purpose of the study: To study the association of GERD with hemostasis disorders in COVID-19.

Methods: A retrospective analysis of the results of 760 autopsies in 2021 was conducted. The studies were conducted at the pathology department of the State Budgetary Healthcare Institution Irkutsk Regional Clinical Hospital of the Order of the Badge of Honor. The analysis of the obtained data was carried out in the Statistica 13 program.

Object of the study: medical documentation - "Act of pathological anatomical autopsy". Results: A retrospective analysis of 760 autopsies of patients with COVID-19 performed in 2021 was carried out. There were 370 men (49%), age 66 [57.0; 74.0]; 390 women (51%), age 68.5 [60.0; 76.0], women were significantly older than men (p = 0.015). Hemostasis disorders were detected in 227 (30%) cases, age 68 [59.0; 76.0]. There were 122 men (54%); women 105 (46%). p=0.015

Conclusions: 1) Hemostasis disorders were detected in 30% of those who died from COVID-19. 2) FLD was more common (19%) in those who died from COVID-19 and had hemostasis disorders than in those who died from COVID-19 without hemostasis disorders (12%) p = 0.019 3) PE was detected in 15% of those who died from COVID-19.

4) The risk of developing hemostasis disorders in those who died from COVID-19 and had FLD is 1.4 times higher than in those who did not have FLD, and the risk of developing PE is 1.7 times higher. The obtained results may indicate an association of GBP with hemostatic disorders in COVID-19

Keywords: fatty liver disease, pulmonary embolism, COVID-19, hemostasis disorder, thrombosis.

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Introduction. Non-alcoholic fatty liver disease (NAFLD) is the most common liver disease in all countries, with a prevalence of 20-30% and continuing to increase [1]. Considering that it is far from always possible to clearly define the etiological factor in the development of fatty liver disease, and morphologically alcoholic and non-alcoholic fatty liver diseases are indistinguishable, many scientists use the term "fatty liver disease" or steatous liver disease [2].

In 2020, an international expert consensus statement was published proposing a new adaptive concept – MBD: Metabolic (dysfunction) associated fatty liver disease. The proposed interpretation of the disease makes it possible not only to emphasize the systemic and multifactorial pathogenesis of a unified lesion of the hepatic parenchyma (for example, the combination of dysmetabolic and alimentary-toxic), but also to personalize the scope and directions of therapeutic and diagnostic care for various clinical variants of CKD-associated comorbidity [13]. However, the 2024 clinical guidelines use the concept of non-alcoholic fatty liver disease.

The prevalence of CKD increases the interest of scientists in the study of pathology, which is accompanied by an annual increase in the number of publications on this topic. So, only on the pages of the PubMed electronic medical library for the period from 2000 to 2023, there are 38,854 publications on the request of "nonalcoolic fatty liver disease", of which 4,678 were published in 2023.

It has been observed and proven that NAFLD is associated with a lower life expectancy, the main cause of which is mortality from cardiovascular diseases, which has aroused the interest of cardiologists in this pathology [1, 3].

There is a link between CKD and the development of cardiovascular pathology, and one of the most important pathophysiological links is endothelial dysfunction (ED) [4]. It was found that the incidence of ED among patients with steatosis and steatohepatitis reaches 77% and 82%, respectively [5].

One of the basic mechanisms of endothelial dysfunction is a change in the synthesis and release of endothelial nitric oxide (NO), one of the most important regulators of the endothelial-vasal system. The leading cause of NO deficiency is considered to be the destruction or capture of NO by free radicals. The excessive formation of free radicals, which disrupt the endothelium-dependent relaxation of blood vessels and enhance the contractile reactions of smooth muscle, is triggered by the activation of chemical reactions, including lipid peroxidation (POL). At the same time, the formed POL products: malondialdehyde and 4-hydroxynonenal, trigger the formation of collagen, but already in the walls of blood vessels [6].

In the period from 2020 to the present, with the advent of the new COVID-19 coronavirus infection, which was characterized by a pandemic and was accompanied by high mortality, it was noted that a significant proportion of adverse outcomes were due to a catastrophe in the hemostatic system, including cerebral, coronary and mesenteric vascular thrombosis, one of the links in the pathogenesis of which is endothelial dysfunction. Scientific publications around the world report an increase in the incidence of pulmonary embolism (PE) among intensive care patients. In particular, it was shown that in 2020, during the COVID-19 pandemic, the incidence of PE among intensive care patients was 20.6%, which was more than three times higher than the number of similar cases in 2019 - 6.1% [7].

The suspected cause of vascular thrombosis in patients with COVID-19 is direct damage to the endothelium by the virus. The involvement of the intercellular substance in the inflammatory process and the subsequent effect on the subendothelial matrix containing tissue factor (TF) and collagen causes activation of the coagulation cascade along the external pathway. The effect of TF and collagen leads to the formation of thrombin and the conversion of fibrinogen into fibrin, which, together with platelet aggregates, forms blood clots [8].

The above proves that the mechanisms of hemorrhagic and thrombotic complications, including PE, in patients suffering from a new coronavirus infection have not been sufficiently studied and proven, the severity of the problem cannot be underestimated and research in this direction is actively continuing.

However, we were unable to find information on how susceptible hemorrhagic and thrombotic complications are in patients with CKD and COVID-19.

The aim of the study was to study the effect of CBP on hemostasis disorders in COVID-19.

Materials and methods. In order to study the association of fatty liver disease with hemostasis disorders in patients with COVID-19, a retrospective analysis of the results of 760 autopsies performed in 2021 at the Irkutsk Regional Bureau of Forensic Medical Examination was conducted. The concept of NAFLD was used in connection with the impossibility of differential diagnosis of NAFLD and ABP according to liver histology and insufficient anamnestic data.

The object of the study was medical documentation – the "Act of pathoanatomic autopsy", on the basis of which the causes of death, histological data of liver and cardiovascular system damage (vessels and myocardial condition) in people who died from COVID-19 were studied.

Inclusion criteria: the presence in the protocols of data on the morphological examination of the liver and heart muscle, blood vessels, a positive test for COVID-19.

Exclusion criteria: incomplete information about age, cause of death, and age under 17.

Statistical analysis of the obtained data was performed in the Statistica 13 for Windows program. The data in the groups were processed using classical methods for biomedical work using parametric and nonparametric statistical criteria (Student's t-test, Mann-Whitney U-test, Fisher's φ-test, χ2). To calculate the relative risk, a four-field conjugacy table was also constructed based on the number of subjects with certain values of factorial and performance characteristics.

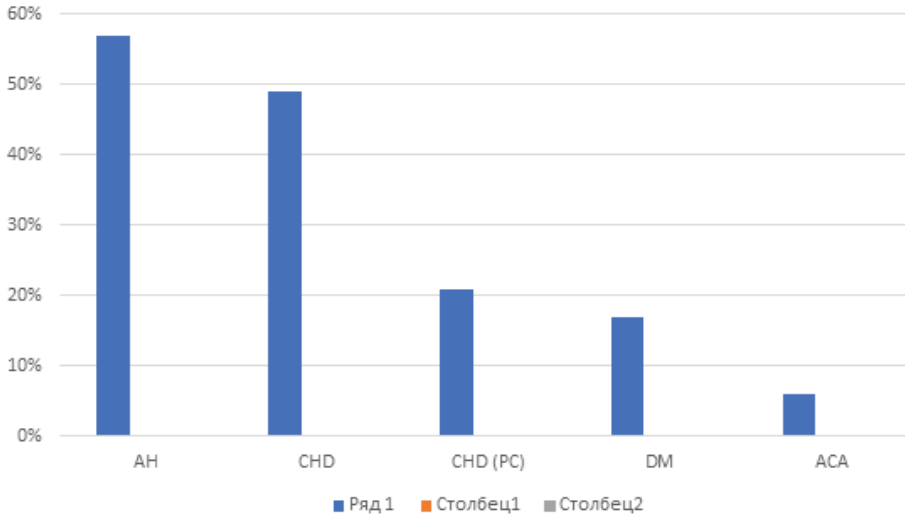
The boundaries of the confidence interval were calculated - 95% CI. The values of relative risk and the boundaries of the confidence interval were compared with unity.

Results. A rating analysis of 760 cases of COVID-19 infection in 2021 has been published. Male 370 (49%), age 66.0 [57.0; 74.0]; female 390 (51%), age of COVID-19 68.5 [60.0; 76.0], women were significantly older than men (p=0.015).

TBP was detected in 106 (14%) cases, men were 59 (57%), 47 (43%) women. Cardiovascular pathology and CKD were detected in 51 (48%) cases, of which hypertension accounted for 29 (57%), CHD – 24 (49%), PIC – 10 (21%), ONCC in 3 cases (6%), type 2 diabetes mellitus - 7 (17%) cases (Figure).

Hemostasis disorders among all those who died from COVID-19 were detected in 227 (30%) cases, age 68.0 [59.0; 76.0]. In men, hemostasis disorders were significantly more common than in women in 122 cases and 105, respectively (p=0.015).

Hemostasis disorders had the following manifestations: mesenteric thrombosis – 12 (2%); deep vein thrombosis of the lower extremities - 28(4%); ischemic stroke – 14 (2%); PE – 100 (13%); DIC syndrome -73 (10%).



Concomitant pathology of cardiovascular diseases and type 2 diabetes mellitus in patients with fatty liver disease and COVID-19

Overweight and obesity among all those who died from COVID-19 (n=760) were detected in 232 (31%) cases, and among those with hemostasis disorders (n=227) in 65 (29%) cases. There were no significant differences in the incidence of obesity among those who died from COVID-19 (depending on the presence or absence of hemorrhagic complications) (p=0.47).

Among those who died from COVID-19 with hemostasis disorders (n=227), CKD was more common in 42 (19%) than among non- of those who had (n=532) 64 (12%). The differences are significant (p=0.019).

To confirm the association of fatty liver changes with the development of hemostasis disorders, the relative risk was calculated (Table 1).

Calculations show that CKD is associated with impaired hemostasis in COVID-19. The risk of developing hemostasis disorders in the group of COVID-19 patients with PD is 1.4 times higher than in the group without PD.

When hemostasis was impaired in people with CKD and COVID-19 (n=106), PE was most common - in 24% (25) of cases, which is significantly more common than in people without CKD - in 14% (106) (p<0.05) (Table 2).

To confirm the association of CKD with PE in patients with COVID-19, the relative risk was calculated (Table 3).

Calculations have shown that RBP is associated with the development of PE in COVID-19. The presence of CKD in people with COVID-19 increases the risk of PE by 1.7 times.

Discussion: Currently, the incidence of CKD is pandemic and has a high prevalence. The liver plays a central role in metabolism and detoxification, as well as in protein synthesis, including coagulation factors. Endothelial dysfunction can affect the blood supply to the liver, exacerbating existing diseases. It is known that changes in liver function are observed in COVID-19, which may be associated with high levels of inflammatory cytokines, cytolytic effects, as well as microcirculation disorders. This can lead to increased levels of transaminases in the blood and more serious conditions such as acute liver damage. In addition, against the background of COVID-19, some patients have disorders in the hemostasis system [15].

There are many factors that can contribute to a hypercoagulable condition in patients with COVID-19. The main one is endothelial dysfunction, since damage to endothelial cells excessively activates platelets and the coagulation system [10].

As a result of our study, 30% (227) of those who died from COVID-19 had hemostasis disorders, which were more common in men (54% (122 cases) than in women (46% (105 cases)). Hemostasis disorders had the following manifestations: mesenteric thrombosis – 12 (2%); deep vein thrombosis of the lower extremities - 28 (4%); ischemic stroke – 14 (2%); PE – 100 (13%); DIC-73 (10%).

The predominance of these hemostasis disorders among men may be explained by lower levels of estrogens, which are known to have a protective effect on the endothelium (through genomic and non-genomic mechanisms) [15].

Accordingly, this contributes to a greater susceptibility to endothelial damage and activation of the calycrein-kinin system. In addition, activation in the hemostasis system predisposes a higher level of testosterone [15].

Previously, the association of endothelial damage with the risk of acute car-

Table 1

Relative risk of developing hemostasis disorders associated with fatty changes in the liver

	Violation of hemostasis	There are no hemostasis disorders
FLD	42(40%)	64(60%)
No FLD	185(28%)	469(72%)
The frequency difference is statistically significant p<0.05		
		ДИ 95%
Relative risk (RR) ± standard error of relative risk (S)	1.4±0.13	1.0-1.8

Table 2

Association of fatty liver disease with conditions associated with hemostasis disorders

	FLD (n=106)	No FLD (n=654)	p
Mesenteric thrombosis	3(3%)	9(1%)	0.26
Deep vein thrombosis	4(4%)	24(4%)	0.95
Ischemic stroke	0(0%)	14(2%)	-
PE	25(24%)	91(14%)	**0.011***
DIC syndrome	13(12%)	60(9%)	0.32

Note: p is the Chi-Square criterion, *- significant differences of p<0.05

Table 3

Relative risk of pulmonary embolism associated with fatty liver disease

	PE	No PE
FLD (n=106)	25 (24)	81 (76)
No FLD (n=653)	91 (14)	562 (86)
The frequency difference is statistically significant p=0.011		
		ДИ 95%
Relative risk (RR) ± standard error of relative risk (S)	1.7±0.2	1.1-2.5

diovascular disasters has been proven, so the relative risk of death from CVD in men with CVD is 1.4 times higher than in women [16], which places the male sex as a risk factor for vascular damage.

We found that acute cardiovascular disasters associated with hemostasis disorders in COVID-19 deaths were 1.4 times more common among people with CKD than without it, 19% (42) and 12% (64), respectively (p = 0.019). And the risk of PE in patients with fatty liver disease is 1.7 times higher.

Based on the results of our study, we concluded that CKD significantly increases the risk of dysfunction in the hemostatic system and, as a result, acute vascular catastrophes such as PE, venous thrombosis and mesenteric thrombosis in patients with COVID-19. This is confirmed by other studies that report both prothrombotic and hypofibrinolytic changes in the blood coagulation system in patients with CKD [15]. The researchers point to an increase in the level of blood coagulation factor (VIII) and an inhibitor of plasminogen activator-1, the most important regulator of the fibrinolytic system, as well as a decrease in the level of natural anticoagulants — proteins S and C. Studies of dynamic coagulation analysis, including thromboelastometry and thrombin generation, have confirmed the theory of prothrombotic imbalance in CKD, as well as the presence of more hemostatically active immature platelets, as evidenced by an increased average platelet volume [11]. These pathogenetic links cause hemostasis disorders in patients with CKD, and when infected with COVID-19, changes in the hemostasis system are aggravated, which causes an increased risk of thrombosis and thromboembolism.

Also, in such studies, the results were presented when COVID-19 infection worsens the course of CKD, manifested by increased activity of liver enzymes (AST, ALT, GGT), the severity of which varies from minor to moderate. It was found that liver dysfunction, determined by changes in the level of liver enzymes and albumin, was detected in 43% of patients with CKD and COVID-19 [12].

From the above, it can be concluded that liver damage can be considered as an independent predictor of higher mortality in patients with COVID-19.

Conclusions: Hemostasis disorders were found in 30% of those who died from COVID-19. CKD was more common (19%) in those who died from COVID-19 and had a hemostasis disorder than in those who died from COVID-19 without a hemostasis disorder (12%) (p = 0.019). Hemostasis disorders were more common in men (54%) than in women (46%) (p=0.015). In 24% of those who died from COVID-19 and had a concomitant disease with TB, the cause of death was PE. The risk of developing hemostasis disorders in those who died from COVID-19 and had CKD is 1.4 times higher than in those who did not have CKD, and the risk of developing PE in this group of patients is 1.7 times higher.

Conclusion: Fatty liver disease is a significant risk factor for hemostasis disorders in patients with COVID-19. Studies show that the presence of CKD is associated with dysregulation of inflammatory processes, which can significantly increase the likelihood of thrombosis.

Patients with CKD have elevated levels of pro-inflammatory cytokines, which can lead to platelet activation and decreased anticoagulant activity. These changes in hemostasis can be aggravated by concomitant diseases such as cardiovascular pathologies, which further increases the risks.

Therefore, it is important to carry out early diagnosis and regular monitoring of hemostasis in patients with CKD infected with COVID-19, as well as to apply individualized treatment approaches in order to minimize the risks of thrombosis and improve clinical outcomes. In the future, it is necessary to continue research in this area, which will allow for a deeper understanding of the mechanisms of interaction between TBD and COVID-19 in the context of hemostasis.

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CLINICAL CASE

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CLINICAL CASE OF RHEUMATIC CHOREA WITH CARDIAC INVOLVEMENT IN A 13-YEAR-OLD CHILD IN THE REPUBLIC OF SAKHA (YAKUTIA)

During the last two decades, the prevalence of acute rheumatic fever has significantly decreased to isolated cases nationwide. In this article, a clinical case of rheumatic chorea, with choreic hyperkinesis syndrome, in a 13-year-old child with cardiac involvement is described. Modern concepts of therapy of rheumatic chorea are presented.

Keywords: chorea, streptococcus, fever, myocarditis, children, Sakha, Yakutia.

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Introduction. Rheumatic chorea (syn. Sydenham's chorea, minor chorea) is a post-streptococcal immune-mediated inflammatory neuropsychiatric movement disorder. The disease has been known since the Middle Ages and was called "St. Vitus' dance" at the beginning of observation. In 1686 Thomas Sydenham described "a kind of convulsion which affects boys and girls from the tenth year of life to puberty". Sydenham's chorea, is a great diagnostic criterion for acute rheumatic fever (ARF). It debuts between 5 and 15 years of age, with a peak at 8-9 years of age [12], girls are more commonly affected [4,5,9].

Rheumatic chorea is an inflammatory postinfectious lesion of the CNS that occurs 4-8 weeks after a streptococcal infection, predominantly pharyngitis. Typically, bilateral involvement is noted, but approximately 20% of patients have hemichorea. The disease is a consequence of an autoimmune reaction following infection with " group A β-hemolytic streptococcus" (GABHS). The phenomenon of molecular mimicry is thought to underlie the pathogenesis of the disease. After frequent macroorganism contacts with GABHS, predisposed individuals develop autoreactive lymphocytes and antibodies directed against epitopes of group A β-hemolytic streptococcus that cross-react with human cells [8]. This cross-reactive immune reaction explains the heterogeneity of the symptoms of ARF, which is usually manifested by skin lesions (subcutaneous nodules, erythema annulare), joint pain, fever, and cardiac involvement (myocarditis, transient atrio-ventricular block, heart valve endocarditis).

The clinical picture is composed of choreic hyperkinesis, muscle hypotonia (up to imitation of paralysis), disorders of statics and coordination, vascular dystonia, emotional lability, and psychiatric disorders. The duration of the attack is 3-6 months, residual phenomena can be observed for a year.

The Jones criteria [3] are used to diagnose acute rheumatic fever (Table 1).

Differential diagnosis. The most difficult cases to diagnose are cases of isolated minor chorea. The circle of differential diagnosis includes PANDAS, obsessive-compulsive syndrome, transient tics, autoimmune encephalitis, systemic autoimmune diseases (systemic lupus erythematosus, antiphospholipid syndrome), primary angiitis of the central