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Л.А. Анисько[1,2], И.А. Карпов[2]

[1]— УЗ «Городская клиническая инфекционная больница» г. Минска, Минск, Беларусь
[2]— УО «Белорусский государственный медицинский университет», Минск, Беларусь

НАРУШЕНИЯ СИСТЕМЫ ГЕМОСТАЗА КАК ПРЕДИКТОР НЕБЛАГОПРИЯТНОГО ИСХОДА COVID-19

L.A. Anisko[1,2], I.A. Karpov[2]

[1]— City Clinical Hospital of Infectious Diseases, Minsk, Belarus
[2]— Belarusian State Medical University, Minsk, Belarus

Disorders Of the Hemostatic System as A Predictor of Adverse Outcome In COVID-19

Резюме

Инфекция COVID-19 ассоциирована с высоким риском тромботических осложнений, определяющих тяжесть течения и исход заболевания. Выявление ключевых предикторов неблагоприятного исхода среди параметров системы гемостаза остается важной задачей. **Цель.** Оценить значимость показателей системы гемостаза в качестве предикторов неблагоприятного исхода у пациентов с COVID-19. **Материалы и методы.** Проведено одноцентровое ретроспективное когортное исследование, включившее 9256 пациентов с подтвержденным COVID-19. В зависимости от исхода (неблагоприятный/благоприятный) пациенты были разделены на группы. При поступлении оценивались показатели коагулограммы (активированное частичное тромбопластиновое врермя, протромбиновый индекс, тромбиновое время, фибриноген, D-димер, антитромбин III). Статистический анализ выполнялся с использованием U-критерия Манна-Уитни. Для описания количественных показателей использовались медиана, 25-й и 75-й процентили. **Результаты.** У пациентов с неблагоприятным исходом зафиксированы статистически значимые различия по сравнению с выжившими: значительное повышение уровня D-димера (медиана 464,5 нг/мл [Q25–Q75: 245,0–1120,0] нг/мл против 198,0 [110,0–350,0] нг/мл; p<0,0001) и фибриногена (5,62 [4,70–6,80] г/л против 5,03 [4,30–5,90] г/л; p<0,0001), снижение активности антитромбина III (81,0 % [73,0–89,0] против 99,0 % [90,0–108,0]; p=0,0001) и протромбинового индекса (83,0 % [77,0–90,0] против 93,0 % [88,0–98,0]; p<0,0001), а также удлинение тромбинового времени (15,4 с [14,5–16,5] против 14,9 с [14,2–15,8]; p=0,0001). Показатель АЧТВ значимо не отличался между группами (p=0,95). **Заключение.** У пациентов с неблагоприятным исходом COVID-19 выявлена выраженная гиперкоагуляция с признаками коагулопатии потребления, характеризующаяся резким повышением D-димера и фибриногена на фоне снижения антитромбина III и протромбинового индекса. Мониторинг этих показателей, особенно D-димера и антитромбина III, обладает высокой прогностической ценностью для стратификации риска и своевременной коррекции терапии.

Ключевые слова: *COVID-19, система гемостаза, коагулопатия, D-димер, антитромбин III, фибриноген, летальность, прогноз*

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Abstract

COVID-19 is associated with a high risk of thrombotic complications, which determine the severity and outcome of the disease. Identifying key predictors of adverse outcomes among hemostatic system parameters remains an important task. **Aime.** To assess the significance of hemostatic system parameters as predictors of adverse outcomes in patients with COVID-19. **Materials and methods.** A single-center retrospective cohort study

was conducted, including 9256 patients with confirmed COVID-19. Depending on the outcome (adverse/favorable), patients were divided into groups. Upon admission, coagulogram parameters (APTT, prothrombin index, thrombin time, fibrinogen, D-dimer, antithrombin III) were assessed using an ACL TOP 750 analyzer. Statistical analysis was performed using the Mann-Whitney U test. For the description of quantitative indicators, the median, the 25th and 75th percentiles were used. **Results.** Statistically significant differences were recorded in patients with adverse outcome compared to survivors: a significant increase in D-dimer level (median 464.5 [Q25–Q75: 245.0–1120.0] ng/mL vs. 198.0 [110.0–350.0] ng/mL; p<0.0001) and fibrinogen (5.62 [4.70–6.80] g/L vs. 5.03 [4.30–5.90] g/L; p<0.0001), a decrease in antithrombin III activity (81.0 % [73.0–89.0] vs. 99.0 % [90.0–108.0]; p=0.0001) and prothrombin index (83.0 % [77.0–90.0] vs. 93.0 % [88.0–98.0]; p<0.0001), as well as a prolongation of thrombin time (15.4 s [14.5–16.5] vs. 14.9 s [14.2–15.8]; p=0.0001). The APTT parameter did not differ significantly between the groups (p=0.95). **Conclusion.** Patients with adverse COVID-19 outcomes exhibited marked hypercoagulation with signs of consumption coagulopathy, characterized by a sharp increase in D-dimer and fibrinogen against a background of decreased antithrombin III and prothrombin index. Monitoring these parameters, especially D-dimer and antithrombin III, has high prognostic value for risk stratification and timely therapy adjustment.

Key words: *COVID-19, hemostatic system, coagulopathy, D-dimer, antithrombin III, fibrinogen, mortality, prognosis*

Conflict of interests

Co-author of the article Karpov I.A. is a member of the editorial board of the journal «The Russian Archives of Internal Medicine». The article passed the journal's peer review procedure. Karpov I.A. was not involved in the decision to publish this article. The authors did not declare any other conflicts of interest

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Conformity with the principles of ethics

The study was approved by the Local Ethics Committee of the "City Clinical Infectious Diseases Hospital" healthcare institution (Protocol No. 1, February 12, 2021). All patients signed an informed consent form.

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ACE2 — angiotensin-converting enzyme 2, aPPT — activated partial thromboplastin time, DIC — disseminated intravascular coagulation, PTI — prothrombin index

Introduction

Since the beginning of the COVID-19 pandemic, compelling evidence has been gathered indicating that SARS-CoV-2 causes not merely a respiratory infection, but a systemic disease characterised primarily by vascular injury (vasculopathy) and thromboinflammation [1]. A key mechanism initiating these processes is the interaction of the viral S-protein with the angiotensin-converting enzyme 2 (ACE2) receptor, which is expressed on the surface of endothelial cells, type II pneumocytes, enterocytes, and cells of other organs [2]. This interraction triggers a cascade of reactions leading to direct endothelial dysfunction, massive release of proinflammatory cytokines (the so-called "cytokine storm"), and subsequent activation of both the plasma and cellular components of the hemostatic system, resulting in a state of hypercoagulability [3].

The resulting phenomenon, which is often described as COVID-19-associated blood-clotting disorder, differs from the classic sepsis-associated DIC-syndrome [4, 5]. Marked hypercoagulability is characteristic of COVID-19 and, in the early phase, is manifested by a significant increase in D-dimer and fibrinogen levels — an acute-phase protein and substrate for blood clot formation, accompanied by only mild prolongation of standard coagulation tests, such as prothrombin time (PT) and activated partial thromboplastin time (aPTT), and by a relatively preserved or even elevated platelet count during the early stages of the disease [6]. This pattern reflects intensive blood clotting, primarily in the microcirculatory bloodstream, associated with generalised endothelial activation and inflammation.

Numerous prospective and retrospective studies conducted in different countries confirmed that the D-dimer level upon admission is a powerful independent prognostic marker of disease severity and mortality. Tang et al. (2020) demonstrated that in non-survivors, D-dimer levels were significantly higher than those observed in surviving subjects, and values > 1.0 μg/mL were associated with a high risk of mortality [7]. These data were later confirmed by large-scale meta-analyses where higher D-dimer levels were associated with 3–4-fold increase in the risk of death [8, 9].

However, optimal threshold values of this marker for risk ranking; they can vary considerably depending on the study population, test method, and disease characteristics. Moreover, the prognostic value of an isolated measurement of D-dimer at hospital admission may be insufficient. Researchers emphasise a set of changes in hemostasis. Progressive thrombocytopenia during the course of the disease is an independent adverse prognostic factor, likely reflecting platelet consumption during blood clotting formation and the severity of endothelial injury [10].

Evaluation of the activity of the natural anticoagulant systems is of particular interest. Reduced activity of antithrombin III, the key physiological inhibitor of thrombin and other serine proteases, may indicate its consumption under conditions of extensive blood clot formation and/or impaired hepatic synthetic function, thereby exacerbating the procoagulant shift [5]. At the same time, higher levels of von Willebrand factor and soluble thrombomodulin have been studied as markers of endothelial trauma and activation [11]. Another feature of COVID-19-associated coagulopathy is the frequent detection of lupus anticoagulant, which may contribute to the pathophysiology, although its direct role in thrombogenesis and its impact on clinical outcomes require further clarification [12].

Thus, a comprehensive, multiparametric evaluation of hemostatic status extending beyond the scope of the routine coagulation profile appears to be of critical importance. Such an approach may not only enable highly accurate prediction of adverse outcomes but also facilitate the identification of patients who may benefit from intensified anticoagulant therapy, which is currently the subject of ongoing clinical investigations [13].

Study objective: To perform a comprehensive analysis of clinical and laboratory parameters of the hemostatic system in patients with COVID-19 of various severity in order to identify the independent predictors of adverse outcomes.

Materials and Methods

A single-centre retrospective cohort study was conducted. The study included 9,256 patients (3,980 men and 5,276 women; mean age: 61 ± 16.5 years) with confirmed COVID-19 (based on a positive nasopharyngeal swab PCR test) who were admitted at the Minsk City Clinical Infectious Diseases Hospital between March 2020 and December 2023. Inclusion criteria: age 18 years old and over; hospital admission during the study; laboratory-confirmed SARS-CoV-2 infection by polymerase chain reaction; and the availability of written informed consent provided by the patient or their legal representative for participation in the study and the use of anonymised medical data for scientific purposes, including publication. Exclusion criteria: age below 18 years old; congenital coagulation disorders, the use of therapeutic-dose anticoagulants prior to hospitalisation; and the absence of data on key hemostatic parameters at the time of admission.

Subjects were divided into two groups, depending on the outcome: the study group (patients with adverse outcome, n = 375) and the comparison group (patients with favourable outcome, n = 8,881). For certain laboratory parameters, the size of the analysed subgroups varied depending on the availability of examination data, as reflected in the results table (Table 1).

Venous blood samples were collected upon hospital admission before the initiation of anticoagulant therapy. Standard hemostatic parameters were determined using an ACL TOP 750 automated analyser (Werfen, Spain) with HemosIL reagent kits (Werfen, Spain) in accordance with the manufacturer's instructions. The following parameters were assessed: activated partial thromboplastin time (aPTT), s; antithrombin III, %; D-dimer, ng/mL; prothrombin index (PTI), %; thrombin time, s; and fibrinogen, g/L.

Statistical analysis was performed using R software version 4.2.1. Quantitative variables with non-normal distributions (assessed using the Shapiro-Wilk test) were described using the median (Me) and the 25th and 75th percentiles (Q25–Q75). Comparisons between the two independent groups (favourable and adverse outcomes) were performed using the nonparametric Mann-Whitney U test. Differences were considered statistically significant at $p < 0.05$. Due to the retrospective nature of the study and the lack of data regarding the exact timing of events, Kaplan-Meier survival analysis was not performed.

Results and Discussion

The comparative analysis of hemostatic parameters between patients with adverse and favourable outcomes revealed statistically significant differences in the majority of the variables studied (see table).

The most notable differences were observed in D-dimer levels. The median value of this marker in patients with adverse outcome (464.5 ng/mL) was more than two times higher than in patients with favourable outcome (198.0 ng/mL) ($p < 0.0001$) (Fig.1). A significant elevation in D-dimer levels strongly indicates marked activation of fibrinolysis and intense blood clot formation in patients with an unfavourable prognosis, which is consistent with numerous studies confirming the role of D-dimer as a key predictor of adverse outcomes in COVID-19 patients [7–9]. However, it is worth noting that D-dimer threshold values proposed in literature vary; and in this study, the median value in patients with adverse outcomes was 464.5 ng/mL, which is below the threshold value of 1000 ng/mL, but is significantly above the levels in the comparison group, emphasising the importance of the relative elevation and follow-up [14]. The data are consistent with the meta-analysis results generated by Lippi G. and Favaloro E.J. (2020), who noted that the absolute D-dimer value upon hospital admission is a reliable marker of disease severity; however, the optimal threshold can vary depending on the population and methods used [8]. Moreover, the findings of our study, i.e., a more than twofold increase in

the median D-dimer level in the adverse outcome group, are quantitatively consistent with the results reported by Tang N. et al. (2020), who observed a similar difference between survivors and non-survivors [7].

Significantly lower antithrombin III activity (median value 81.0 % vs 99.0 % in survivors, p = 0.0001) in patients with adverse outcome of COVID-19 infection points at depletion of the most important natural anticoagulant mechanism. This reduction may result from consumption during blood clot formation and/or impaired hepatic synthetic function, thereby contributing to the prothrombotic state (Figure 2), and is consistent with findings from other studies describing decreased antithrombin III levels as a marker of severe consumptive coagulopathy in COVID-19 [15]. The results confirm the findings by White D. et al. (2021), who reported significantly lower antithrombin III activity in patients with severe COVID-19, associated with higher consumption during generalised coagulation activation [15]. This represents an important distinction from classical sepsis-associated disseminated intravascular coagulation (DIC), in which antithrombin III levels also decline, but typically at later stages, whereas in COVID-19 this feature may be observed earlier in the disease course [4, 5].

Table 1. *Alterations of hemostatic parameters in COVID-19 patients based on clinical outcome.*

Parameter	Group	N	Median (Q25-Q75)	p
APTT, sec	Adverse outcome	375	32.8 (29.5–36.2)	0.95
	Favorable outcome	8881	32.2 (29.0–36.0)	
Antithrombin III, %	Adverse outcome	38	81.0 (73.0–89.0)	0.0001
	Favorable outcome	90	99.0 (90.0–108.0)	
D-dimer, ng/mL	Adverse outcome	372	464.5 (245.0–1120.0)	0.0000
	Favorable outcome	9367	198.0 (110.0–350.0)	
Prothrombin Index, %	Adverse outcome	255	83.0 (77.0–90.0)	0.0000
	Favorable outcome	5189	93.0 (88.0–98.0)	
Thrombin Time, sec	Adverse outcome	189	15.4 (14.5–16.5)	0.0001
	Favorable outcome	3214	14.9 (14.2–15.8)	
Fibrinogen, g/L	Adverse outcome	364	5.62 (4.70–6.80)	0.0000
	Favorable outcome	9019	5.03 (4.30–5.90)	

Note: APTT — activated partial thromboplastin time; N — number of observations; Q25–Q75 — 25th and 75th percentiles (interquartile range); p — level of statistical significance of differences between groups (Mann-Whitney test)

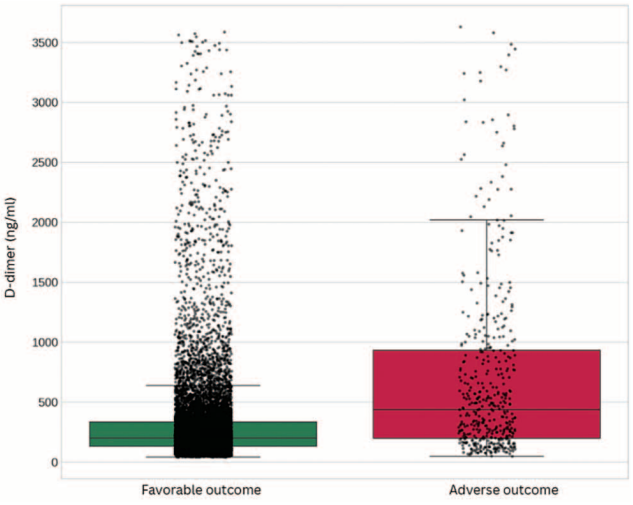

Figure 1. *Distribution of COVID-19 infection disease outcomes depending on the level of D-dimers at hospital admission*

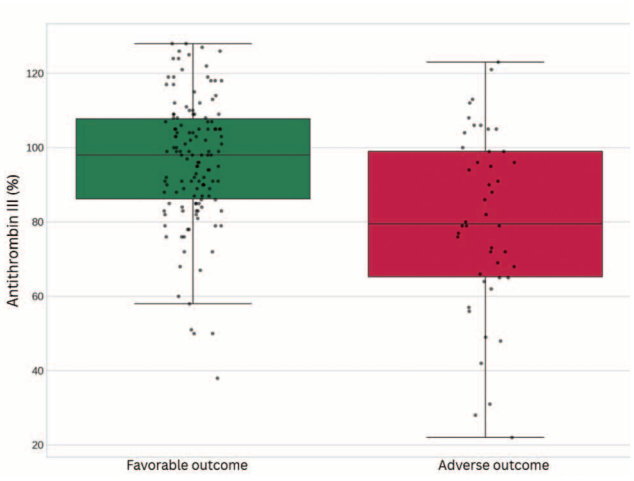

Figure 2. *Distribution of COVID-19 infection outcomes depending on antithrombin III level upon hospital admission*

Statistically significant reduction in prothrombin index (median value 83.0 % vs 93.0 %, p < 0.0001) in patients with adverse outcome reflects impaired extrinsic blood coagulation, which is likely to be associated with more marked hepatic damage or vitamin K deficiency in patients with severe disease. Similar changes were described in articles about blood-clotting disorder in COVID-19 patients, where prolonged prothrombin time (and lower PTI) was associated with poorer outcomes [7]. It can be caused both by direct viral damage to hepatic cells, expressing ACE2 receptor, and hypoxia or use of medications in critically ill patients [2].

Fibrinogen concentration was significantly higher in patients with adverse outcome of COVID-19 infection (median value 5.62 g/L vs 5.03 g/L, p < 0.0001). Elevated fibrinogen levels, as an acute-phase protein, may reflect the intensity of the systemic inflammatory response (hypercytokinemia), which is directly associated with activation of the coagulation system [3, 11]. Although the trend toward increased fibrinogen levels in patients with severe disease or fatal outcomes was statistically significant, it was less pronounced in our study than that observed for D-dimer. This finding is consistent with the observations of Iba T. et al. (2020), who reported that COVID-19 is characterised by elevated or normal fibrinogen levels even in the presence of overt thrombosis, distinguishing it from other forms of consumptive blood-clotting disorder and underscoring the role of hyperinflammation in the pathogenesis of thrombosis [4]. Therefore, significantly higher, but not extreme fibrinogen levels, in this study contributes to hypercoagulation and inflammation.

A slight but statistically significant prolongation of thrombin time (median value 15.4 s vs 14.9 s, p = 0.0001) in patients with adverse outcomes may indicate qualitative alterations in fibrinogen or the presence of coagulation inhibitors in the plasma, which may also reflect severe metabolic disturbances. These slight changes in standard global tests in marked hypercoagulability are a characteristic feature of COVID-19-induced coagulopathy [6].

In contrast, activated partial thromboplastin time (aPTT) did not differ significantly between the groups (p = 0.95), suggesting that the intrinsic coagulation pathway is less involved in the pathological process or that its alterations are compensated by other factors. This pattern is characteristic of COVID-19-induced coagulopathy and distinguishes it from classical disseminated intravascular coagulation (DIC) [4, 6]. These findings are fully consistent with the international guidelines developed by ISTH (Thachil J. et al., 2020), where it is emphasised that normal or slightly altered aPTT values in the presence of elevated D-dimer and fibrinogen levels is a typical laboratory finding in COVID-19 and should alert clinicians to the risk of thrombosis [6].

Therefore, the combination of the observed alterations (marked elevation of D-dimer, moderate increase in fibrinogen levels, reduced antithrombin III activity and PTI, together with unchanged aPTT) constitutes a laboratory profile characteristic of COVID-19-induced coagulopathy, with features of consumptive coagulopathy in the setting of pronounced hypercoagulability and inflammation. This profile is qualitatively consistent with the findings reported in most contemporary studies [4, 5, 6, 15], while the quantitative values obtained in the present study cohort further expand the existing body of evidence by providing a more detailed characterisation of these parameter changes in this patient population.

Conclusion

Patients with adverse outcomes of COVID-19 infection demonstrate marked hypercoagulation, characterised by significantly higher D-dimer and fibrinogen levels, as well as depleted anticoagulant potential (reduced antithrombin III concentration) and impaired procoagulant synthesis by the liver (reduced PTI). These changes give rise to a pattern of COVID-19-induced coagulopathy characterised by features of both a procoagulant shift and consumption of coagulation factors. Monitoring of these parameters, especially of D-dimer and antithrombin III levels, has high predictive value for timely therapy adjustment and improvement of disease outcomes.

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Карпов И.А. — концепция и дизайн исследования, контроль проведения исследования, интерпретация данных, редактирование рукописи

Author Contribution:

All the authors contributed significantly to the study and the article, read and approved the final version of the article before publication

Anisko L.A. — conceptualization and study design, data collection and processing, statistical data analysis, writing and editing the article

Karpov I.A. — conceptualization and study design, study supervision, data interpretation, article editing

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Информация об авторах

Анисько Людмила Александровна — к.м.н., доцент кафедры инфекционных болезней с курсом ПКиП учреждения образования «Белорусский государственный медицинский университет», заведующий клинико-диагностической лабораторией учреждения здравоохранения «Городская клиническая инфекционная больница» г. Минск, Республика Беларусь, ORCID ID: https://orcid.org/0000-0002-5466-2590; e-mail: lanisko@internet.ru

Карпов Игорь Александрович — д.м.н., профессор, чл.-корр. НААН Республики Беларусь, заведующий кафедрой учреждения образования «Белорусский государственный медицинский университет», Минск, ORCID ID: https://orcid.org/0009-0004-5432-2133; e-mail: vip.kia1957@gmail.com

Author information

Luidmila A. Anisko — MD, Cand. Sci. (Medicine), Associate Professor of the Department of Infectious Diseases "Belarusian State Medical University"; Head of the Clinical Diagnostic Laboratory of the "City Clinical Infectious Diseases Hospital", Minsk, Republic of Belarus; ORCID ID: https://orcid.org/0000-0002-5466-2590; eLibrary SPIN: 8389-1870; e-mail: lanisko@internet.ru

Igor A. Karpov — MD, Dr. Sci. (Medicine), Professor; Corresponding Member of the National Academy of Sciences of Belarus, Head of the Department of Infectious Diseases "Belarusian State Medical University"; Minsk, ORCID ID: https://orcid.org/0009-0004-5432-2133; eLibrary SPIN: 6594-8929; e-mail: vip.kia1957@gmail.com

Автор, ответственный за переписку / Corresponding author