CORRELATIONS BETWEEN CLINICAL, LABORATORY AND INSTRUMENTAL CHARACTERISTICS OF PATIENTS WITH COVID-19 INFECTION

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ABSTRACT

Background. COVID-19 is a leading disease in terms of prevalence (more than 100 million cases) and mortality (more than 10.0%). Most often, COVID-19 is accompanied by hemostatic disorders (manifestations of COVID-19-associated coagulopathy) and blood coagulation.

Aim. To determine the levels of correlation between clinical and laboratory characteristics of hemostatic and coagulation disorders (according to the characteristics of ThromboElastoGraphy, TEG) in patients with COVID-19 infection.

Materials and Methods. The study was performed at the Department of Infectious and Pediatric Infectious Diseases, Parasitology, Phthisiology and Pulmonology of Kharkiv National Medical University in accordance with the existing recommendations of bioethical norms and rules. All patients signed an informed consent. 179 patients with COVID-19 were examined. The levels of correlation between laboratory and instrumental characteristics were determined using Spearman's rank correlation coefficient. The probability of differences was determined using the Mann-Whitney U-test.

Results & Conclusions. According to the results of the study, significant direct and inverse correlations were found. The identified correlations determined the possible interaction between TEG parameters and the characteristics of the systemic inflammatory response. The obtained results play a significant role for patients with COVID-19, because they can predict shifts in inflammatory systemic response parameters, which characterize the severity of the disease, based on the characteristics of TEG. The interrelationships of hemostatic and coagulation system disorders have been reliably determined by the presence of significant correlations between the characteristics of the inflammatory systemic response of patients with COVID-19 and TEG values. The obtained significant correlations were mostly weak, but there were also interdependencies of medium strength. In particular, Interleukin-6 and some other indicators on days 5–6 after hospitalization.

Keywords: *thromboelastography, TEG, coagulation system disorders.*

Introduction

Since the end of 2019, COVID-19 has become a major threat in a relatively short period of time, taking a leading position in terms of prevalence and mortality [1–10]. Scientists estimate the mortality rate from COVID-19 to be more than 10.0% [4]. Severe acute respiratory syndrome caused by coronavirus type 2 has infected more than 100 million people and caused the death of more than 2.5 million infected patients.

Among the significant variety of clinical symptoms, an extremely high level of hemocoagulation disorders and thromboembolic complications is particularly noteworthy. Based on the clinical and laboratory characteristics of hemostatic disorders (increased fibrinogen levels, mild or moderate thrombocytopenia, and significant increase in D-Dimer levels), they were identified as a separate type – COVID-19 associated coagulopathy [11].

Hypercoagulability in patients with COVID-19 has been widely confirmed, which is detected in at least 20% of infected patients [12], according to other reports, the rate reaches 55% [13]. Activation of the body's inflammatory reactions signi-

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ficantly increases the production of proinflammatory cytokines that stimulate coagulopathic changes [1; 13]. A significant proportion of patients with COVID-19 have coagulation disorders that can develop into disseminated intravascular coagulation or thrombotic microangiopathies [2]. Ahmed S. et al. [7] emphasize the high incidence of thrombosis, both arterial (myocardial infarction, stroke, etc.) and venous (pulmonary embolism, deep vein thrombosis, etc.). In moderate-to-severe COVID-19, coagulopathies are detected in almost all patients, which is a significant factor in in mortality [12]. At the same time, a sharp impact on the body's coagulation homeostasis is a trigger for the development of coagulopathy of exhaustion [1].

In addition, the development of sepsis-induced coagulopathy and disseminated intravascular coagulation has been identified in patients with COVID-19 [1]. These pathological conditions are the result of a complex interaction of cytokine storm and coagulopathy [7]. Changes in the coagulation system in patients with COVID-19 are described as COVID-19-associated coagulopathy [11], which has significant mortality rates [2], although the SARS-CoV-2 virus does not have a procoagulant effect on its own [1]. It is believed that changes in the coagulation system are due to an intense inflammatory response in these patients [1].

In recent years, ThromboElastoGraphy (TEG) has been used quite successfully to determine the existing disorders of the blood coagulation system, which determines the viscoelastic properties of the thrombus and identifies the degree and severity of such disorders [14].

Thus, given the high prevalence of coagulation disorders in patients with COVID-19 with the development of severe COVID-19-associated coagulopathies, determining the correlation levels of interdependencies between clinical and laboratory characteristics of hemostatic disorders and TEG parameters is of clinical relevance.

The aim of the study was to determine the levels of correlation between clinical and laboratory characteristics of hemostatic disorders and thromboelastographic parameters in patients with coronavirus infection.

Materials and Methods

The study was conducted at the Department of Infectious and Pediatric Infectious Diseases, Parasitology, Phthisiology and Pulmonology of Kharkiv National Medical University and at the Municipal Non-Profit Enterprise "Kharkiv Regional Infectious Diseases Hospital" of Kharkiv Regional Council in 2020–2024.

179 patients (96 men (53.63%) and 83 women (46.37%)) with COVID-19 aged 20–88 years (average age [58.75±13.82] years) were examined. The study was conducted in accordance with current international and national recommendations on bioethical norms and rules for research involving human subjects. All patients signed an informed consent.

COVID-19 was diagnosed using Enzyme-Linked ImmunoSorbent Assay (ELISA) and Polymerase Chain Reaction (PCR) in accordance with existing international and national recommendations. In addition to ELISA and PCR, all patients were examined using:

- biochemical blood tests (determination of interleukin-6 (IL-6), D-dimers, ProCalciTonin (PCT) and C-Reactive Protein (CRP) by conventional methods);

- TEG results (determination of the levels of Maximum Rate of Thrombus Formation (MRTG), Time to reach the Maximum Rate of Thrombus Generation (TMRTG), total Thrombus Generation (TG), Maximum Rate of Lysis (MRL), Time to Maximal Lysis Rate (TMRL), total Lysis (L36), Reaction time (R), clot formation time (K), a-Angle, Maximum Amplitude (MA), maximum amplitude time (PMA), maximum clot elasticity (G), clot density immediately (A) and After 30 (A30) min and After 60 (A60) min, Coagulation Index (CI), degree of amplitude reduction after 30 min (LY30) and 60 min (LY60), estimated value of LY30 (EPL), degree of area reduction after 30 (CL30) min and 60 (CL60) min time of A decrease by 2 mm (CLT), clot formation rate (TPI), maximum A (TMA), maximum Elasticity (E), time of formation of the first fibrin filaments (SP) and calculated CLT after 30 seconds (LTE)).

Statistical calculations of the results were performed using IBM SPSS 25.0 for Windows (USA). The levels of correlation were determined using Spearman's rank correlation coefficient (ρ). If it was in the range from 0 to -1.0, the correlation was considered inverse; if it was from 0 to 1.0, it was considered direct. Value ρ from 0 to 0.3 (from 0 to -0.3) was considered as a weak connection between the studied features; from 0.4 to 0.7 (from -0.4 to -0.7) – as moderate; and from 0.7 to 1.0 (from -0.7 to -1.0) – as high strength. The result was presented in the form of a ρ coefficient and the corresponding level of confidence p.

The probability of differences in the obtained features was determined using the Mann-Whitney

U-test. The threshold value of the statistical significance of the calculated traits was taken as 0.05 (p=0.05).

Results and Discussion

According to the correlation analysis of TEG indicators and characteristics of the inflammatory systemic response of the examined patients with COVID-19 infection (IL-6, quantitative values of D-Dimer, PCT, CRP), we identified significant correlations (both direct and inverse), which indicated the possibility of interdependencies between them (*Table*).

Table. Correlation matrix of IL-6, D-Dimer, CRP, PCT and TEG parameters of patients with acute respiratory failure obtained at hospitalization and on day 5–7 (5–6)

Indices		IL-6 (hospitalization), pg/ml	IL-6 (5–7 days) pg/ml	D-Dimer (hospitalization), FEU, ng/ml	D-Dimer (5–6 days), FEU, ng/ml	CRP (hospitalization), mg/l	CRP (5–6 days), mg/l	PCT (hospitalization), ng/ml	PCT (5–7 days), ng/ml
1		2	3	4	5	6	7	8	9
MRTG, mm/min	ρ	0.042	0.120	0.062	0.025	0.118	0.119	-0.054	-0.033
	p	0.573	0.110	0.413	0.744	0.117	0.112	0.469	0.665
TMRTG, min	ρ	-0.162*	-0.175**	-0.181**	-0.232**	-0.343***	-0.312**	-0.012	-0.039
	p	0.030	0.019	0.016	0.002	0.000	0.000	0.872	0.605
TG, mm/min	ρ	0.100	0.173*	-0.116	-0.185**	-0.130	-0.015	-0.092	-0.036
	p	0.184	0.021	0.123	0.013	0.084	0.843	0.220	0.634
MRL, mm/min	ρ	-0.085	-0.129	-0.012	0.011	0.096	0.030	0.058	0.126
	p	0.259	0.084	0.868	0.880	0.201	0.692	0.439	0.093
TMRL, min	ρ	-0.044	-0.034	-0.242***	-0.291***	-0.307***	-0.231**	-0.115	-0.180**
	p	0.562	0.651	0.001	0.000	0.000	0.002	0.125	0.016
L36, mm/min	ρ	-0.020	-0.132	0.045	0.100	0.134	0.031	0.055	0.068
	p	0.788	0.079	0.547	0.183	0.074	0.677	0.465	0.363
R, min	ρ	-0.030	-0.074	-0.190**	-0.312***	-0.258***	-0.177**	-0.029	-0.102
	p	0.689	0.327	0.011	0.000	0.000	0.017	0.696	0.175
K, min	ρ	-0.049	-0.106	-0.023	0.062	-0.157*	-0.190**	-0.024	-0.059
	p	0.517	0.160	0.765	0.409	0.036	0.011	0.751	0.433
A-Angle, °	ρ	0.117	0.156*	-0.001	-0.067	0.155*	0.191**	-0.044	0.071
	p	0.120	0.038	0.985	0.369	0.038	0.011	0.556	0.347
MA, min	ρ	-0.001	0.125	-0.191**	-0.184**	-0.065	-0.053	-0.083	-0.088
	p	0.985	0.095	0.010	0.014	0.385	0.483	0.267	0.241
PMA	ρ	-0.192**	-0.159*	0.161*	0.152*	-0.043	0.015	0.054	0.109
	p	0.010	0.033	0.031	0.042	0.565	0.844	0.473	0.146
G, d/sc	ρ	0.057	0.139	-0.226**	-0.239***	-0.083	0.030	-0.052	-0.080
	p	0.447	0.063	0.002	0.001	0.268	0.687	0.493	0.286
EPL, %	ρ	-0.190**	-0.269***	0.207**	0.268***	0.177**	0.116	0.067	0.130
	p	0.011	0.000	0.005	0.000	0.018	0.122	0.374	0.083
A, mm	ρ	0.157*	0.308***	-0.147*	-0.164*	-0.164*	0.007	-0.105	-0.122
	p	0.035	0.000	0.049	0.028	0.028	0.931	0.164	0.103
CI	ρ	0.013	0.128	-0.108	-0.099	-0.056	0.042	-0.019	0.017
	p	0.867	0.089	0.152	0.187	0.456	0.581	0.799	0.823
LY30, %	ρ	-0.134	-0.239***	0.106	0.150*	0.145	-0.011	0.053	0.038
	p	0.075	0.001	0.159	0.045	0.053	0.880	0.485	0.614
A30, mm	ρ	0.031	0.176**	-0.218**	-0.197**	-0.200**	-0.121	-0.112	-0.101
	p	0.678	0.018	0.003	0.008	0.007	0.107	0.136	0.179

Continuation of Table

1		2	3	4	5	6	7	8	9
CL30, %	ρ	0.118	0.189**	-0.193**	-0.191**	-0.167*	-0.054	-0.133	-0.069
	p	0.115	0.011	0.009	0.010	0.025	0.476	0.077	0.356
A60, mm	ρ	0.050	0.099	-0.111	-0.110	0.008	-0.089	0.018	0.026
	p	0.504	0.187	0.140	0.143	0.914	0.235	0.812	0.727
CL60, %	ρ	0.058	0.105	-0.219**	-0.211**	-0.153*	-0.155*	-0.070	-0.067
	p	0.444	0.160	0.003	0.005	0.041	0.038	0.355	0.376
LY60, %	ρ	-0.128	-0.152*	-0.040	-0.029	0.057	-0.008	-0.037	-0.149*
	p	0.087	0.042	0.600	0.703	0.449	0.914	0.626	0.046
CLT, min	ρ	0.014	-0.064	-0.284***	-0.197**	-0.148*	-0.118	-0.165*	-0.171*
	p	0.850	0.395	0.000	0.008	0.048	0.116	0.027	0.022
TPI, sec	ρ	0.093	0.205**	-0.007	-0.047	-0.002	0.187**	-0.023	0.079
	p	0.215	0.006	0.928	0.530	0.977	0.012	0.757	0.291
TMA, min	ρ	0.071	0.094	-0.198**	-0.125	-0.182**	-0.114	-0.069	-0.120
	p	0.345	0.211	0.008	0.097	0.015	0.128	0.361	0.108
E, d/sc	ρ	0.026	0.167*	0.062	0.030	0.098	0.136	0.017	0.030
	p	0.727	0.025	0.413	0.687	0.191	0.069	0.820	0.687
SP, min	ρ	-0.067	-0.048	-0.126	-0.215**	-0.230**	-0.126	-0.099	-0.104
	p	0.374	0.522	0.092	0.004	0.002	0.094	0.189	0.165
LTE, min	ρ	0.125	0.087	-0.065	0.000	-0.101	-0.021	-0.102	-0.232**
	p	0.095	0.248	0.387	0.996	0.177	0.779	0.175	0.002

Notes: probability of differences * – p≤0.05; ** – p≤0.01; *** – p≤0.001; L36 – total Lysis; R – Reaction time; K – clot formation time; PMA – maximum amplitude time; G – maximum clot elasticity; A – clot density immediately; A30 – clot density After 30 min; A60 – clot density After 60 min; LY30 – degree of amplitude reduction after 30 min; LY60 – degree of amplitude reduction after 60 min; EPL – estimated value of LY30; CL30 – degree of area reduction after 30 min; CL60 – degree of area reduction after 60 min; CLT – time of A decrease by 2 mm; TPI – clot formation rate; TMA – maximum A; E – maximum Elasticity; SP – time of formation of the first fibrin filaments; LTE – calculated CLT after 30 seconds.

In the analysis of correlation relationships, direct and inverse significant correlations were identified, which determined the possible interaction between TEG parameters and characteristics of the inflammatory systemic response. The identified interdependencies play a significant role for patients with COVID-19, as they can predict shifts in inflammatory systemic response parameters (IL-6, D-dimers, CRP), which characterize the severity of the disease, based on the characteristics of TEG. So, there were significant direct correlations between IL-6 levels (obtained during hospitalization) and TEG values, which indicated a possible increase in IL-6 with an increase in A values (ρ=0.157; p=0.035) and the opposite (a decrease in IL-6 levels was noted) with an increase in TMRTG (ρ=-0.162; p=0.030), PMA (ρ=-0.192; p=0.010) and EPL (ρ=-0.190; p=0.011). It should be noted that the correlations obtained in all cases were of a weak nature.

When determining the correlation relationships between the IL-6 values of patients with COVID-19 infection obtained on days 5–7 after hospitalization, a greater number of existing significant relationships were noted. Thus, there were direct interdependencies of weak strength, indicating the possibility of increasing the quantitative levels of IL-6 with an increase in TG (ρ=0,173, p=0.021), A-Angle (ρ=0.156, p=0.038), A (ρ==0.308, p=0.000), A30 (ρ=0.176, p=0.018), CL30 (ρ=0.189, p=0.011), TPI (ρ=0.205, p=0.006) and E (ρ=0.167, p=0.025). In turn, inverse weak correlations were also identified, which stated a possible increase in IL-6 in patients with COVID-19 infection with an increase in the following TEG values: TMRTG (ρ=-0.175, p=0.019), PMA (ρ==-0.159, p=0.033), EPL (ρ=-0.269, p=0.000) and LY30 (ρ=-0.239, p=0.001) and LY60 (ρ=-0.152, p=0.042).

Correlations were determined for D-Dimer of patients with COVID-19 obtained during hospitalization and on day 5–6. Thus, we found probable weak correlation effects that determined possible increases in the levels of D-Dimers obtained during hospitalization: direct, with an increase in the values of PMA (ρ=0.161, p=0.031) and EPL (ρ=

=0.207, p=0.005), and inverse - with a decrease in the values of A (ρ=-0.147, p=0.049), A30 (ρ=-0.218, p=0.003), CL30 (ρ=-0,193, p=0.009), CL60 (ρ=-0.219, p=0.003), CLT (ρ=-0.284, p==0.000), TMA (ρ=-0.198, p=0.008), TMRTG (ρ==-0.181, p=0.016), TMRL (ρ=-0.242, p=0.001), R (ρ=-0.190, p=0.011) and MA (ρ=-0.191, p==0.010) and G (ρ=-0.226, p=0.002).

In addition, we found significant correlations for the quantitative levels of D-dimers in patients with COVID-19 obtained on days 5–6 after hospitalization. We identified the available direct probable weak correlations, which indicated a reliable possibility of increasing the levels of D-Dimers with an increase in PMA (ρ=0.152, p=0.042), EPL (ρ=0.268, p=0.000) and LY30 (ρ=0.150, p=0.045) and vice versa, that determined the increase of D-dimers with a decrease in the values of TMRTG (ρ=-0.232, p=0.002), TG (ρ=-0.185, p=0.013), TMRL (ρ=-0.291, p=0.000), R (medium strength, ρ=-0.312, p=0,000), MA (ρ=-0.184, p=0.014), G (ρ=-0.239, p=0.001), A (ρ=-0.164, p=0.028), A30 (ρ=-0.197, p=0.008), CL30 (ρ=-0.191, p=0.010), CL60 (ρ=-0.211, p=0.005) and CLT (ρ=-0.197, p=0.008) and SP (ρ=-0.215, p=0.004).

It should be noted that the correlation relationships for CRP and TEG in patients with COVID-19 showed a similar trend. Thus, CRP values obtained during hospitalization showed significant direct weak correlations, which indicated a possible increase in its levels with an increase in A-Angle (ρ=0.155, p=0.038) and EPL (ρ=0.177, p=0.018). At the same time, inverse (mostly weak) interdependencies were also identified, which determined the possibility of increasing CRP levels with a decrease in TMRTG (medium strength, ρ==-0.343, p=0.000), TMRL (medium strength, ρ==-0.307, p=0.000), R (ρ=-0.258, p=0.000), K (ρ==-0.157, p=0.036), A (ρ=-0.164, p=0.028), A30 levels (ρ=-0.200, p=0.007), CL30 (ρ=-0.167, p=0.025), CL60 (ρ=-0.153, p=0.041), CLT (ρ==-0.148, p=0.048) and TMA (ρ=-0.182, p=0.015) and SP (ρ=-0.230, p=0.002).

For the CRP values of patients with COVID-19 infection obtained on day 5-6 after hospitalization, we determined mainly unreliable correlations of low strength. Thus, there were significant direct, mostly weak interdependencies that determined the possibility of increasing CRP levels with an increase in A-Angle (ρ=0.191, p=0.011) and TPI (ρ=0.187, p=0.012) and inverse, indicating a possible increase in CRP in patients with COVID-19 with a decrease in TMRTG values (medium strength, ρ=-0.312, p=0.000), TMRL (ρ=-0.231, p=0.002), R (ρ=-0.177, p=0.017) and K (ρ=-0.190, p=0.011) and CL60 (ρ=-0.155, p==0.038).

A similar trend was identified in relation to the correlations between PCT parameters of patients with COVID-19 obtained on days 5–7 after hospitalization and TEG characteristics. Thus, the presence of probable weak inverse correlations was noted only for TMRL (ρ=-0.180, p=0.016), LY60 (ρ=-0.149, p=0.046), CLT (ρ=-0.171, p=0.022) and LTE (ρ=-0.232, p=0.002), which determined possible increases in quantitative PCT levels with a decrease in these TEG values.

Our conclusions regarding the relationship between clinical and laboratory characteristics of hemostatic disorders (manifestations of COVID-19-associated coagulopathy) and coagulation disorders (determined by TEG) are fully consistent with other global studies. Thus, the meta-analysis conducted by Coomes E.A. et al [15] identified significant positive correlations between IL-6 levels and bilateral lung damage and maximum body temperature: ρ=0.45 (p=0.001) and ρ=0.52 (p==0.001), respectively. They determined that increased dysregulation of the immune response to an antigen is a leading element in the development of target organ damage and subsequent mortality [15]. An increase in the cytokine response in patients with COVID-19 and acute respiratory distress syndrome, accompanied by a significant predominance of IL-6, is an integral part of the pathogenesis and dysregulation of the immune response [15].

Other studies have shown that along with diffuse alveolar lesions, patients with COVID-19 are diagnosed with fibrin thrombi [3]. Studies have shown a 9-fold higher incidence of alveolar-capillary microthrombosis in patients with COVID-19 compared to patients with influenza [6]. At the same time, fibrin-rich microthrombosis of the small pulmonary vasculature is detected in almost 80–100% of cases [6]. In addition to elevated levels of D-dimers, they may have skin manifestations on the extremities as a clinical sign of microvascular thrombosis [3]. Thus, plasma levels of D-Dimers are considered as a direct prognostic marker of COVID-19: the levels of this fibrin degradation product are significantly increased in patients with severe COVID-19 [6; 13].

Conclusions

Based on the study, the interrelationships of hemostatic and coagulation disorders were reliably determined by the presence of significant correlations (mostly of low strength) between the

characteristics of the inflammatory systemic response of patients with COVID-19 and thromboelastographic values.

DECLARATIONS:

Disclosure Statement

The authors have no potential conflicts of interest to disclosure, including specific financial interests, relationships, and/or affiliations relevant to the subject matter or materials included.

Data Transparency

The data can be requested from the authors.

Statement of Ethics

The authors have no ethical conflicts to disclosure.

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Consent for publication

All authors give their consent to publication.

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