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DIAGNOSTIC SIGNIFICANCE OF LACTATE DEHYDROGENASE IN PATIENTS WITH COVID-19 BASED ON CLINICAL AND LABORATORY DATA

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Lactate dehydrogenase is an intracellular enzyme that reflects the degree of tissue damage and the intensity of the inflammatory response. In COVID-19 infection, increased lactate dehydrogenase activity is associated with cytolytic damage to lung, liver, and heart cells, as well as the development of systemic hypoxia and metabolic dysfunction. A retrospective analysis of 102 patients treated between June and December 2020 was conducted. The diagnosis of COVID-19 was confirmed using a real-time polymerase chain reaction. Lactate dehydrogenase activity was measured at hospital admission using an automated biochemical analyzer, Cobas c311 (Roche Diagnostics GmbH), in accordance with international laboratory quality standards. The patients were divided into two groups: those with mild-to-moderate disease (n=82) and those with severe disease (n=20). The markers of severity according to which the distribution took place were: saturation (SpO2), computed tomography readings (CT1-4), and the level of C-reactive protein. The average LDH level in the group with a severe course was 913.3±84.7 u/l, significantly higher than in the group with mild/moderate severity (458.0±62.4 u/l; p<0.001). Elevated lactate dehydrogenase levels correlated with elevated ferritin, C-reactive protein, and D-dimer concentrations, indicating systemic inflammation, severe tissue hypoxia, and cellular stress. The observed relationship between lactate dehydrogenase activity and the severity of COVID-19 confirms the pathogenetic role of this enzyme in the development of cytokine storm and multiple organ dysfunction.

Key words: COVID-19, lactate dehydrogenase, biomarkers, inflammation, cytolysis, hypoxia, prognosis, severity of the disease, laboratory diagnostics.

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ДІАГНОСТИЧНЕ ЗНАЧЕННЯ ЛАКТАТДЕГІДРОГЕНАЗИ У ПАЦІЄНТІВ ІЗ COVID-19 НА ОСНОВІ КЛІНІКО-ЛАБОРАТОРНИХ ДАНИХ

Лактатдегідрогеназа є внутрішньоклітинним ферментом, який відображає ступінь ураження тканин та інтенсивність запальної реакції. При інфекції COVID-19, підвищена активність лактатдегідрогенази асоціюється з цитолітичним ураженням клітин легенів, печінки та серця, а також з розвитком системної гіпоксії та метаболічної дисфункції. Було проведено ретроспективний аналіз 102 пацієнтів, які проходили лікування у період з червня по грудень 2020 року. Діагноз COVID-19 було підтверджено методом полімеразної ланцюгової реакції в реальному часі. Активність лактатдегідрогенази визначали при надходженні до стаціонару з використанням автоматичного біохімічного аналізатора Cobas c311 (Roche Diagnostics GmbH) відповідно до міжнародних стандартів якості лабораторних досліджень. Пацієнтів було розділено на дві групи: з легким/середньотяжким перебігом (n=82) та тяжким перебігом захворювання (n=20). Маркерами тяжкості, за якими проводився розподіл, були сатурація кисню (SpO_2), дані комп'ютерної томографії (КТ1–4) та рівень С-реактивного білка. Середній рівень лактатдегідрогенази у групі важкого перебігу становив 913,3±84,7 Од/л, що статистично значуще вище порівняно з групою легкого/середньотяжкого перебігу (458,0±62,4 Од/л, p<0,001). Підвищені значення лактатдегідрогенази корелювали з підвищеними концентраціями феритину, С-реактивного білка та D-димеру, що вказувало на системне запалення, виражену тканинну гіпоксію та клітинний стрес. Спостережуваний зв'язок між активністю лактатдегідрогенази та тяжкістю COVID-19 підтверджує патогенетичну роль цього ферменту у розвитку цитокінового шторму та поліорганної дисфункції.

Ключові слова: COVID-19, лактатдегідрогеназа, біомаркери, запалення, цитоліз, гіпоксія, прогноз, тяжкість захворювання, лабораторна діагностика.

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The COVID-19 pandemic, caused by the SARS-CoV-2 virus, has posed an unprecedented challenge to modern healthcare systems and science. The disease is characterized by a wide spectrum of clinical manifestations, ranging from asymptomatic and mild forms to a severe course with the development of bilateral pneumonia, acute respiratory distress syndrome (ARDS), sepsis, and multiple organ failure. This clinical polymorphism and the unpredictability of disease progression have necessitated the search for reliable laboratory indicators that can promptly reflect disease severity, predict outcomes, and assist in the selection of therapeutic strategies [1, 2, 4, 10].

Among numerous biochemical markers, lactate dehydrogenase (LDH) occupies a special place as a universal intracellular enzyme that catalyzes the conversion of pyruvate to lactate during anaerobic glycolysis. LDH is present in all tissues of the body, particularly in cells of the lungs, heart, liver, and skeletal muscles. Damage to cell membranes and the subsequent release of the enzyme into the bloodstream reflect the degree of tissue cytolysis and metabolic dysfunction. Elevated serum LDH levels in patients with COVID-19 are considered an integral indicator of systemic inflammation, hypoxia, and tissue destruction [6, 14].

The pathogenesis of COVID-19 involves

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complex interactions between virus-induced injury to the alveolar epithelium, endothelial dysfunction, and hypoxia. Alongside this, the so-called “cytokine storm” develops – hyperproduction of IL-6, TNF-α, and other inflammatory mediators – which contributes to tissue injury and a generalized inflammatory response [7, 12, 13]. Thus, LDH reflects not only the degree of cytolysis but also the depth of systemic metabolic disturbances, serving as a universal marker of the severity of the patient’s condition.

Post-COVID sequelae remain an important scientific and clinical problem and are accompanied by residual hypoxia, pulmonary fibrosis, and impaired cellular energy metabolism. Elevated LDH values often persist even after clinical recovery, indicating ongoing subclinical inflammation and incomplete restoration of tissue homeostasis [10, 11, 14].

Numerous studies demonstrate that LDH can be used as part of integrated diagnostic panels alongside C-reactive protein, ferritin, procalcitonin, and D-dimer, which substantially increases the accuracy of predicting a severe course of disease [9, 12, 15].

It should be emphasized that LDH is an accessible and inexpensive laboratory parameter measured in virtually all clinical laboratories. This makes it particularly valuable for practical healthcare, including in resource-limited settings. The combination of high sensitivity, reproducibility, and clinical relevance makes LDH an essential component of laboratory monitoring in COVID-19 and other infectious diseases accompanied by tissue injury [1, 5].

Therefore, investigating the diagnostic and prognostic significance of LDH in COVID-19 is of high scientific and practical relevance. LDH serves not only as an indicator of cellular destruction but also as a universal biomarker of systemic metabolic disturbances and inflammation. Its use facilitates early risk stratification, optimization of therapeutic tactics, and improvement of the effectiveness of laboratory monitoring both in the acute phase of infection and during rehabilitation.

The purpose of the study was to investigate the diagnostic significance of lactate dehydrogenase in patients with COVID-19, using clinical and laboratory data.

Materials and methods. The study was conducted in a retrospective format at the Mediclub clinic (Baku, Azerbaijan). Data from 102 patients with a laboratory – confirmed diagnosis of COVID-19 who were hospitalized from June 1 to December 31, 2020 were analyzed, corresponding to the first wave of the pandemic in the region. All patients had a positive SARS-CoV-2 RNA test result confirmed by real-time polymerase chain reaction (PCR) using the CFX-96 amplifier (Bio-Rad, USA). To exclude false-positive results, negative and positive controls were used, as well as an internal quality standard. To confirm the primary PCR result, the PCR analysis was repeated after 24–48 hours.

Patients were allocated into two clinical groups: Group 1 (mild and moderate course, n=82) – patients with moderate dyspnea, SpO_2 > 94 %, minimal CT changes, and a CRP level<60 mg/L.; Group 2 (severe course, n=20) – patients with pronounced respiratory failure (SpO_2<90 %), signs of ARDS, lung involvement of more than 50 % according to CT data, a high level of C-reactive protein (CRP > 100 mg/L), and the need for oxygen therapy.

The inclusion criteria were individuals with a confirmed COVID-19 result, age older than 18 years, and availability of biochemical blood test results at admission. Patients with active oncological processes, pronounced renal or hepatic failure, pregnant women, and individuals receiving immunosuppressive therapy were excluded.

Determination of LDH activity was performed at hospital admission, prior to initiation of specific therapy. Blood sampling was performed from the cubital vein in the fasting state into tubes without anticoagulant; centrifugation was carried out at 3000 rpm for 10 minutes. Reference values for LDH were 135–214 U/L for women and 135–225 U/L for men. An increase of more than twofold above the upper limit of normal was regarded as a sign of pronounced cytolysis.

As noted above, inclusion in diagnostic panels, alongside LDH, of markers such as CRP, D-dimer, ferritin, and procalcitonin, as well as hematological parameters (leukocytes, lymphocytes, platelets, and hemoglobin), increases the accuracy of predicting a severe course of disease. Measurement of CRP, D-dimer, ferritin, and LDH was performed on the Cobas c311 automated biochemical analyzer (Roche Diagnostics GmbH, Germany). For each analytical series, original Roche reagents and certified calibrators were used. Procalcitonin was measured on an iChroma analyzer (Boditech Med Inc., South Korea), and hematological parameters were determined using a Sysmex XT2000i hematology analyzer (Japan). All investigations were performed under biosafety level BSL-2 conditions in compliance with ISO 15189:2022 and ISO 9001:2015 requirements. Internal laboratory quality control (IQC) was performed daily, and participation in external assessment programs (EQA/PT) ensured independent validation of results.

Statistical processing of the data was performed using Microsoft Excel 2021 and Python 3.9. For descriptive statistics, the mean (M), standard deviation (SD), median, quartile intervals (Q25–Q75), and minimum and maximum values were calculated. Intergroup differences were assessed using Student’s t-test and the Mann–Whitney U test. Correlations between LDH and clinical and laboratory parameters were analyzed using Spearman’s correlation coefficient (r_s). Statistical significance was accepted at $p<0.05$.

Ethical principles were observed in accordance with the Declaration of Helsinki (2013). All patient personal data were de-identified; the study was approved by the Local Ethics Committee of the Mediclub clinic (protocol No. 03/2020). Since the study was retrospective, written informed consent from each patient was not available.

Results of the study and their discussion. A total of 102 patients with laboratory-confirmed SARS-CoV-2 infection were included in the study. According to the clinical course, cases were classified as severe (n=20), mild (n=47), and moderate (n=35). For statistical comparison, mild and moderate courses were combined into one group (mild/moderate, n=82). Table 1 presents the demographic and laboratory characteristics of the analyzed groups.

Table 1

Demographic and laboratory characteristics of patients with COVID-19

Parameter	Mild/Moderate (n=82)	Severe (n=20)	p
Number of patients, n	82	20	–
Age, years	54.34±14.25 (n=82)	58.40±11.59 (n=20)	0.188
Men, n (%)	57 (69.5 %)	13 (65.0 %)	0.789*
Women, n (%)	25 (30.5 %)	7 (35.0 %)	0.789*
IL-6, pg/mL	39.04±47.28 (n=82)	316.94±85.73 (n=18)	<0.001
CRP, mg/L	66.86±94.93 (n=49)	94.59±113.96 (n=14)	0.162
LDH, U/L	458.02±437.55 (n=43)	913.33±705.54 (n=10)	0.005
Ferritin, ng/mL	978.54±1455.05 (n=53)	1307.18±732.71 (n=16)	0.003
D-dimer, µg/mL	1.29±2.15 (n=49)	3.08±2.89 (n=14)	0.002
Procalcitonin, ng/mL	1.62±4.78 (n=44)	1.22±1.68 (n=18)	0.009
WBC, $\times 10^9$/L	8.68±4.69 (n=40)	10.44±4.23 (n=11)	0.166
NEUT, $\times 10^9$/L	6.44±4.39 (n=39)	8.56±4.22 (n=11)	0.111
LYMPH, $\times 10^9$/L	1.64±0.95 (n=39)	1.38±0.56 (n=11)	0.893
MONO, $\times 10^9$/L	0.61±0.23 (n=38)	0.43±0.28 (n=11)	0.040
MONO %, %	9.06±4.29 (n=39)	4.35±2.35 (n=11)	0.001
EO, $\times 10^9$/L	0.04±0.09 (n=40)	0.09±0.10 (n=11)	0.048
BASO, $\times 10^9$/L	0.01±0.01 (n=40)	0.03±0.03 (n=11)	0.012
Hb, g/L	130.23±20.60 (n=40)	120.45±13.36 (n=11)	0.082
PLT, $\times 10^9$/L	206.13±70.07 (n=40)	242.91±122.71 (n=11)	0.416
ESR, mm/h	43.43±35.33 (n=35)	66.56±38.50 (n=9)	0.073

Note: Data are presented as mean±SD; n in parentheses indicates the number of patients with the available parameter. p values were calculated using the Mann – Whitney U test (two-sided). *For sex, Fisher's exact test was used.

The mean age was 54.34±14.25 years in the mild/moderate group (n=82) and 58.40±11.59 years in the severe group (n=20); the differences did not reach statistical significance (p=0.188). In the mild/moderate group there were 57 men and 25 women, and in the severe group 13 men and 7 women; there were no sex-related differences (Fisher p=0.789). Therefore, the identified differences in laboratory parameters predominantly reflect disease severity.

IL-6 was determined in 100 patients (mild/moderate n=82; severe n=18) and increased sharply in severe disease: 39.04±47.28 pg/mL versus 316.94±85.73 pg/mL, respectively (p<0.001). Despite the high variability of cytokine markers, the distribution of IL-6 demonstrated a pronounced shift toward higher values in severe patients, which corresponds to the hyperinflammatory profile of COVID-19. A key marker of tissue injury was lactate dehydrogenase (LDH). LDH values were available for 53 patients (mild/moderate n=43; severe n=10). In patients with severe disease, the mean LDH level was 913.33±705.54 U/L, whereas in the mild/moderate group it was 458.02±437.55 U/L; the differences were statistically significant (p=0.005). Thus, severe disease was accompanied by an almost twofold increase in LDH, indicating more pronounced cytolysis and/or hypoxic–metabolic stress in severe COVID-19.

The diagnostic informativeness of LDH for distinguishing severe from non-severe disease was assessed by ROC analysis in the subset of patients with available LDH (total number in both groups n=53, including 10 severe cases). The area under the ROC curve was AUC=0.788. The optimal cut-off according to the Youden index was 401 U/L; sensitivity was 80.0 % and specificity was 72.1 %. The positive predictive value (PPV) reached 40.0 %, and the negative predictive value (NPV) was 93.9 %, which underscores the potential usefulness of LDH as a test for ruling out severe disease when values are below the threshold.

Markers of coagulopathy and hyperinflammation also differed between groups. D-dimer was determined in 63 patients (mild/moderate n=49; severe n=14) and was significantly higher in severe disease: 1.29±2.15 µg/mL versus 3.08±2.89 µg/mL (p=0.002). Ferritin was determined in 69 patients (mild/moderate n=53; severe n=16) and was higher in the severe group: 978.54±1455.05 ng/mL versus 1307.18±732.71 ng/mL (p=0.003). For ferritin, significant distributional asymmetry and extremely high values were noted, which increased the SD; nevertheless, the intergroup shift toward higher values in severe disease persisted.

C-reactive protein was determined in 63 patients (mild/moderate n=49; severe n=14) and tended to be higher in severe disease: 66.86±94.93 mg/L versus 94.59±113.96 mg/L; however, statistically significant differences were not obtained (p=0.162). Procalcitonin was determined in 62 patients (mild/moderate n=44; severe n=18). Its mean values were 1.62±4.78 ng/mL and 1.22±1.68 ng/mL, respectively; at the same time, the distributions differed statistically significantly (p=0.009). Given that the mean value in the non-severe group was higher, this result is likely driven by pronounced outliers and heterogeneity of clinical phenotypes, including a possible contribution of bacterial complications in some patients.

Correlation analysis confirmed that LDH is associated with several key pathophysiological axes of COVID-19. A moderate positive correlation was identified between LDH and IL-6 (ρ=0.443; p=0.0010; n=52). LDH also correlated with CRP (ρ=0.460; p=0.0007; n=51). The strongest association was observed between LDH and ferritin (ρ=0.718; p<0.001; n=50), as well as between LDH and D-dimer (ρ=0.635; p=0.000024; n=37). The correlation between LDH and procalcitonin was also positive (ρ=0.430; p=0.0089; n=36). These results indicate that increased LDH reflects not an isolated cytolysis process but a complex combination of inflammation, hypercoagulation, and metabolic stress.

Hemogram parameters were available for a smaller proportion of patients and were considered as additional characteristics of the inflammatory profile. Total white blood cell count (WBC) was determined in 51 patients (mild/moderate n=40; severe n=11) and did not differ statistically significantly: 8.68±4.69×10^9/L versus 10.44±4.23×10^9/L (p=0.166). Absolute neutrophil count (NEUT) showed a tendency to increase in severe disease: 6.44±4.39×10^9/L (n=39) versus 8.56±4.22×10^9/L (n=11), p=0.111. Absolute lymphocyte count (LYMPH) was comparable: 1.64±0.95×10^9/L (n=39) and 1.38±0.56×10^9/L (n=11), p=0.893, which does not confirm a pronounced intergroup difference in this subset of data

During data quality control, one clearly erroneous monocyte (MONO) value that did not correspond to the reference range was identified and excluded from monocyte calculations. Monocytes were 0.61±0.23×10^9/L in the mild/moderate group (n=38) and 0.43±0.28×10^9/L in the severe group (n=11), p=0.040. The monocyte proportion (MONO %) was substantially lower in severe disease: 9.06±4.29 % (n=39) versus 4.35±2.35 % (n=11), p=0.001. The study showed that the absolute eosinophil count (EO) was higher in the severe group: 0.04±0.09×10^9/L (n=40) and 0.09±0.10×10^9/L (n=11), p=0.048; basophils (BASO) were also higher: 0.01±0.01×10^9/L (n=40) versus 0.03±0.03×10^9/L (n=11), p=0.012.

Erythroid lineage and platelet indices did not differ significantly. Hemoglobin (Hb) was 130.23±20.60 g/L in the mild/moderate group (n=40) and 120.45±13.36 g/L in the severe group (n=11), showing a trend without statistical significance (p=0.082). Platelet count (PLT) was 206.13±70.07×10^9/L (n=40) and 242.91±122.71×10^9/L (n=11), respectively (p=0.416). ESR was determined in 44 patients and was higher in severe disease: 43.43±35.33 mm/h (n=35) and 66.56±38.50 mm/h (n=9); however, the differences did not reach statistical significance (p=0.073).

From a practical standpoint, the combination of elevated LDH with high IL-6, D-dimer, and ferritin values may be used for early risk stratification, selection of monitoring intensity, and timely transfer of the patient to a unit with a higher level of care. At the same time, the obtained data underscore the need for a standard set of tests at admission and in dynamics, which will allow more accurate threshold estimation, reduce sampling bias, and increase the reproducibility of results across different clinical units.

Comparison of our observations with published results from other authors demonstrates complete consistency. For example, in the retrospective cohort by Zhou et al., elevated LDH was included among the factors associated with an unfavorable course and was linked to an increased risk of death, underscoring the prognostic significance of this marker in hospitalized patients [15]. A meta-analysis by Henry et al. also showed that LDH is statistically significantly higher in patients with severe COVID-19 compared with non-severe forms and proposed considering it an accessible prognostic biomarker reflecting the severity of tissue injury and systemic inflammation [4]. In the study by Poggiali et al., the combination of elevated LDH and CRP levels was associated with the risk of respiratory failure, which is in good agreement with the association we identified between LDH and clinical severity and inflammatory parameters [7].

Pathophysiologically, LDH elevation in COVID-19 is likely due to several interrelated mechanisms. First, virus-induced injury to the alveolar epithelium and endothelium disrupts microcirculation and gas exchange, resulting in tissue hypoxia. Under hypoxic conditions, anaerobic glycolysis is enhanced and the intracellular pyruvate–lactate flux increases; with membrane damage, LDH is released into the systemic circulation as a marker of cytolysis. Second, the hyperinflammatory response and cytokine activation increase oxidative stress, disrupt energy metabolism, and trigger cascades of cell death, which also contributes to increased LDH. Third, COVID-19 is frequently accompanied by endothelial dysfunction and microthrombosis, which enhances ischemic–hypoxic tissue injury and may explain the observed association of LDH with D-dimer.

Importantly, in our study LDH demonstrated correlations with several key laboratory axes of COVID-19: IL-6/CRP (inflammation), ferritin (intensity of the inflammatory response and metabolic stress), and D-dimer (coagulopathy). This “integral” profile explains why LDH should be interpreted as part of a panel rather than as an isolated test. More recent publications also emphasize the value of a combined approach: Li et al. showed that LDH is associated with severity and complications and may be included in integrated diagnostic/prognostic models [6], and Zeng et al. confirmed the prognostic significance of LDH in clinical and biochemical assessments of disease course [14]. In this context, our ROC result and the strong associations of LDH with ferritin and D-dimer support the conclusion that LDH reflects not only “local” lung injury but also the systemic nature of the pathological process.

Interpretation of LDH thresholds requires caution. The selected cut-off provided an acceptable balance of sensitivity and specificity, and the high negative predictive value in the subset of patients with measured LDH indicates that low LDH values may be useful for ruling out severe disease at the time of initial assessment. However, the moderate positive predictive value emphasizes that elevated LDH should not serve as the sole criterion for escalation of care: decisions regarding transfer to the intensive care unit, intensification of oxygen support, or changes in therapy should be based on a comprehensive assessment of clinical status (SpO_2, work of breathing, CT findings), biomarker dynamics, and comorbidity background.

Limitations. The limitations of the study include its retrospective design and the characteristics of the sample, which may limit the generalizability of the results to other populations and clinical settings. Some concomitant factors (comorbidity, treatment characteristics, and the timing of biomaterial collection) could have influenced LDH levels and other laboratory parameters and could not always be fully accounted for in the analysis. In addition, LDH is a non-specific marker; therefore, interpretation of its elevation requires caution and correlation with clinical data and other biomarkers. Larger prospective studies are needed to confirm the identified cut-off values and prognostic models.

Conclusion

The conducted study confirms our assumption that lactate dehydrogenase level is a reliable integral biomarker of COVID-19 severity and reflects the degree of tissue injury, inflammatory activity, and metabolic dysfunction. A significant increase in LDH in patients with a severe disease course correlated with high levels of C-reactive protein, ferritin, and D-dimer, indicating the presence of pronounced cytolytic and hypoxic syndromes. Determination of LDH at hospital admission enables early risk stratification by the risk of developing complications, prediction of potential clinical deterioration, and timely decision-making regarding therapeutic tactics and monitoring. The obtained results confirm the need for further research to refine LDH cut-off values, to evaluate dynamic changes in the enzyme during treatment, and to develop clinical interpretation algorithms. The use of LDH as part of comprehensive biomarker models may improve the accuracy of predicting severe forms of COVID-19 and contribute to the improvement of personalized approaches to patient management.

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