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The role of laboratory biomarkers in diagnostics and management of COVID-19 patients

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Abstract

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the cause of a highly transmittable and heterogenic infection of the respiratory tract, characterized by a broad spectrum of clinical manifestations with a different degree of severity. Medical laboratories play an important role in early diagnosis and management of Coronavirus Disease 2019 (COVID-19) patients. Indeed, the results of several laboratory tests are essential for assessing the severity of the disease, selecting appropriate therapeutic procedures and monitoring treatment response. Routine laboratory testing in COVID-19 patients includes biomarkers of acute phase reaction, hematological and biochemical parameters that indicate tissue injury. The aim of this review paper is to describe the role of these biomarkers in the diagnostics and management of adult and pediatric COVID-19 patients.

Keywords: biomarker, COVID-19, laboratory

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Introduction

Coronavirus disease 2019 (COVID-19) is a new form of a respiratory and systemic disorder caused by the SARS-CoV-2 virus, which has affected millions of people worldwide. The total number of registered COVID-19 cases in Serbia until 20th March 2022 was 1,966,378 with 15.687 deaths (0.8%), according to the official Ministry of Health of the Republic of Serbia website (1). SARS-CoV-2 leads to a highly transmittable and heterogenic infection of the respiratory tract which is characterized by a broad spectrum of clinical manifestations with a different degree of severity. The leading complications of this infection are a severe pneumonia, which can lead to acute respiratory distress syndrome (ARDS) and multiorgan failure (MOF). Clinical laboratories have a very important role in the COVID-19 pandemic, which refers not only to screening and diagnosis of the disease, but also to monitoring changes in laboratory parameters in order to assess the severity of the disease and its progression (2,3). Furthermore, the results of routine biochemical and hematological laboratory tests (Table I) are essential in selecting appropriate therapeutic options and monitoring treatment response. In this review, we will discuss the role of main laboratory biomarkers in the management of COVID-19 patients. The specificity of laboratory diagnostics of COVID-19 in pediatric patients will also be addressed.

Table I Laboratory parameters for the diagnosis and management of COVID-19
Tabela I Laboratorijski parametri za dijagnostiku i praćenje COVID-19

Process	Inflammation	Tissue damage	Impaired coagulation
Laboratory parameters	WBC	LDH	D-dimer
	CRP	ALT	Platelets count
	Fibrinogen	AST	Prothrombin time
	PCT	Cardiac troponins	
	IL-6	NT-pro-BNP	
	Ferritin	Creatinine	
		Bilirubin	
		Albumin	

Biomarkers of inflammation

Excessive inflammation is considered to be the main cause of critical illness. A significant number of patients develop a severe form of the disease, often associated with systemic immune response driven by a “cytokine storm”. The “cytokine storm” is a consequence of ineffective control of inflammatory cytokine release due to abnormal immune-inflammatory response, and could be responsible for MOF and fatal progression of COVID-19 (4,5) This explains why inflammatory biomarkers, such as C-reactive

protein (CRP) and procalcitonin (PCT), play a principal role in early diagnostics, monitoring and management of patients with SARS-CoV-2 infection.

So far, numerous studies have focused on CRP levels in different stages of COVID-19 disease in order to determine the prognostic value and clinical usefulness of this widely used biomarker. These worldwide studies have shown that CRP, a biomarker that is usually not very increased in viral infections, might be useful in clinical practice, for early prediction of disease severity and for the assessment of treatment response of COVID-19 patients with respiratory failure (6). It has been noticed that circulating CRP levels in COVID-19 patients increase significantly in the early stage of disease. Moreover, the studies showed a positive correlation between baseline CRP levels and disease severity (7). Tan et al. (8) demonstrated that CRP has good diagnostic accuracy in predicting severe COVID-19 early (area under the curve [AUC]: 0.87; 95% confidence interval [CI]: 0.10–1.00). The sensitivity and specificity of CRP levels higher than 20.42 mg/L for severe COVID-19 were 83% and 91%, respectively. Furthermore, the levels of CRP correlate with computed tomography (CT) scan severity scores, making this biomarker even more precious in predicting outcomes in infected patients in terms of disease worsening (8).

The link between CRP and disease severity was further supported by the results of another study, which revealed higher levels of CRP on admission among non-survivors when compared to COVID-19 survivors. In particular, severe COVID-19 patients were divided into discharged group and deceased group, and the authors found a significant association between higher CRP levels and adverse disease outcome (7). Regarding the fact that only a few laboratory parameters can be used for monitoring treatment responses, significantly lower levels of CRP in responders compared to the non-responders group point out that the CRP level could be used to identify patients who would benefit from treatment with IL-6 blockers (3,9). However, clinical usefulness of CRP for predicting organ dysfunction in patients with severe complications and its use for treatment decision-making is yet to be proved and needs to be established in future studies.

PCT is a glycoprotein, synthesized by the C-cells of the thyroid gland under normal circumstances, and a precursor of calcitonin. Serum PCT levels are usually low or undetectable in healthy individuals. The hypothesis that PCT is a mediator of inflammation is supported by the evidence of structural homology between PCT and other cytokines, such as interleukin (IL)-1β, tumor necrosis factor (TNF)-α, and IL-6. These cytokines actively promote the synthesis of PCT in extrathyroidal sources (liver, kidney, lung, pancreas, intestine and leukocytes) in the presence of bacterial infection. In contrast, the synthesis of PCT is inhibited by increased concentrations of interferon (IFN)-γ in viral infection. Accordingly, serum PCT levels are increased during severe bacterial, parasitic or fungal infections, but relatively low in viral infections (10) Therefore, PCT levels can be used to distinguish between bacterial and viral infections (3).

Numerous studies investigating immunological characteristics of severe and fatal forms of COVID-19 have revealed that elevated levels of several inflammatory markers, including PCT, were positively associated with the severity of COVID-19 (11-14). In

addition, a concise meta-analysis by Lippi and Plebani (15) demonstrated that increased PCT values are associated with a nearly fivefold higher risk of severe SARS-CoV-2 infection, and that serial PCT measurement may be useful for predicting disease evolution towards a more severe form. In accordance with this, Malik et al. (16), in their meta-analysis of 21 studies, have confirmed that in non-complicated COVID-19 infection PCT may remain normal, but a continuous increase in PCT levels may indicate a bacterial co-infection and the development of a more complicated clinical picture (COVID-19 pneumonia and ARDS). Additionally, the authors confirmed that PCT, together with CRP, correlates with disease severity in ARDS patients, but PCT levels were significantly higher in ARDS patients with sepsis. Based on these findings, PCT seems to be a useful biomarker for early identification of septic complication (17).

A study conducted by Sayah et al. (4) demonstrated that PCT had a high prognostic relevance to assess severe forms of COVID-19 (AUC: 0.86; 95%CI: 5.44–28.39). The sensitivity and specificity of PCT levels higher than 0.138 ng/mL for severe COVID-19 were 76.3% and 79.5%, respectively. Furthermore, the cut-off values of PCT (0.16 ng/mL) had high diagnostic accuracy to predict mortality with high sensitivity (96.3%) and specificity (70.5%). It should be noted that PCT threshold in different studies varies from 0.07 ng/mL to 0.73 ng/mL (18,19). The explanation for this discrepancy could be related to a usually small sample size and different characteristics of study participants, including age, preexisting comorbidities (hypertension, chronic kidney disease, diabetes mellitus, and congestive heart failure) and therapy protocols.

Clinical usefulness of determining PCT levels in COVID-19 patients also lies in PCT-guided antibiotic stewardship, especially in the intensive care units, to prevent the development of severe disease progressing to sepsis and death. Namely, it is known that the administration of antibiotics covers bacterial infection (co-infection) and correlates with increased PCT levels (>0.5 ng/mL) (13). Heesom et al. (20) showed that the use of PCT as a guide for de-escalation of antibiotics significantly reduced antibiotic usage by 2 days in 26% COVID-19 patients with positive blood culture and PCT > 0.5 ng/mL. The experience of Clinical Hospital Centre "Dr Dragiša Mišović – Dedinje" shows that in the period from October to November 2021 the use of antibiotics in intensive care units (ICUs) was reduced, as the number of positive blood cultures was below 30% in nearly 500 COVID-19 patients with PCT > 0.5 ng/mL, which is important because it reduces the possibility of developing multidrug-resistant bacteria. However, larger studies are needed to establish PCT-guided antibiotic stewardship in COVID-19 patients.

Hematological parameters

Depending on the severity of infection, host status and disease phase, leukocyte count (WBC) could be increased or decreased. The most prominent finding is lymphopenia, which is noticed in 30-80% of hospitalized patients. Lymphopenia severity is a negative prognostic sign in severely ill patients for ARDS development, ICU admission, artificial ventilation and exitus. Lymphopenia is caused presumably by the fall in CD4+ and CD8+ T cell subpopulations, which is frequently followed by natural killer

and B cell decline as well. This could be associated with the cytokine storm development and progression (2,21,22). Our experience (Medical Biochemistry Laboratory, Health Center Sokobanja) regarding the results of ambulatory COVID-19 patients showed that women have an average 27% decrease in total WBC count (lymphocyte count decrease of 32%), while men experienced a slightly lower decrease of about 20% (lymphocyte count decrease of 26%), as compared to non-COVID subjects. Lymphopenia is frequently accompanied by neutrophilia, especially in more severe forms of the disease, which is a consequence of bacterial super-infection, but also indicates immunological disturbance. On the contrary, ambulatory patients with mild/moderate disease severity showed neutropenia (24% and 16% decrease in women and men, respectively, compared to non-COVID subjects). Blood monocyte count is usually decreased and it correlates with disease severity, which is explained by monocyte lung tissue infiltration (consumption from blood). Certain monocyte subsets produce IL-6, a pro-inflammatory cytokine, which further extends a cytokine storm in severely ill patients (22). Eosinophil and basophil count drop is a common characteristic of severely ill COVID-19 patients, because these WBC subpopulations have a role in pulmonary tissue hyper-reactivity evolution (21). Because of these prominent changes in WBC subtypes, several indices were recently evaluated as biomarkers of systemic inflammation, among which the most useful in COVID-19 patients are the neutrophil–lymphocyte ratio (NLR) and the monocyte–lymphocyte ratio (MLR) In particular, increased values of both NLR and MLR were indicators of a worse disease outcome (23,24).

Mild or moderately decreased erythrocyte (RBC) count in COVID-19 could rarely be seen. Uncommon severe anemia could be developed in patients with a worse disease prognosis. Anemia occurrence is explained through the autoimmune complication of COVID-19, with hemolysis as a consequence. Additionally, COVID-19 causes iron metabolism disturbance, connected with hepcidin, a protein with significant homology to viral spike-protein. This homology and virus host cell invasion causes intracellular iron accumulation and a subsequent fall in blood iron and haemoglobin level (2,23). Besides, SARS-COV-2 virus influences RBC rheological characteristics, thus increasing the possibility for thromboembolic consequences, which are not uncommon in severe COVID-19 patients (25,26).

Thrombocytopenia seen in COVID-19 patients is caused by autoimmune disturbances caused by the disease itself, or as a consequence of disseminated intravascular coagulation (DIC) (2,27). Moreover, thrombocytopenia could be in correlation with disease severity and a good prognostic parameter for the patients' survival. Ambulatory patients, according to our evidence, had a slight fall in the platelet count (about 20% regarding reference values in both genders) at the time of disease diagnosis.

Elevated D-dimer indicates increased risk of abnormal blood clotting and therefore its concentrations in plasma are commonly used in clinical practice to exclude a diagnosis of venous thromboembolism (VTE) or pulmonary embolism (PE). D-dimer is known to be a mixture of fragments of different weight, and test results can be reported in terms of

weight for units of volume or as fibrinogen equivalent units (FEU) (28). The levels of D-dimer are markedly increased in patients with severe COVID-19, using a cut off-value 0.5 μg/mL (29). According to Conte et al. (30), elevated D-dimer concentrations in COVID-19 patients are due to excessive inflammation, platelet activation, endothelial dysfunction, and stasis, more than hyperfibrinolysis. Recent studies have shown that elevated concentrations of D-dimer might be associated with COVID-19 progression. The concentrations of D-dimer in patients with COVID-19 admitted to the ICU were significantly increased (13). The results of one study in France showed that concentrations of D-Dimer <1.0 μg/mL had a negative predictive value of 90% for VTE and 98% for PE. The positive predictive value for VTE was 44% and 67% for D-dimer concentration ≥1.0 μg/mL and ≥ 3μg/mL, respectively (31). In addition to VTE and PE, D-dimer might be a manifestation of severe virus infection. Namely, Snijders et al. (32) showed that patients with severe pneumonia had significantly higher D-dimer concentrations, while D-dimer within reference range indicated low risk for complications. Increased D-dimer concentrations in patients with severe pneumonia caused by SARS-CoV-2 virus may be an indirect manifestation of an inflammatory reaction, since pro-inflammatory cytokines could cause an imbalance of coagulation and fibrinolysis in the alveoli, which may activate the fibrinolysis system, thus increasing the level of D-dimer (33).

Enzymes

The alteration of liver injury biomarkers, especially of the enzymes aspartate aminotransferase (AST) and alanine aminotransferase (ALT), is a very common laboratory finding in COVID-19 patients (34-36). Studies have shown that, even in milder forms of the disease, increased serum AST and ALT activities can indicate liver dysfunction (37). Some authors suggest that liver dysfunction in COVID-19 patients may be the result of secondary liver damage due to direct liver injury, systemic inflammatory response, congestive hepatopathy, hepatic ischemia, use of hepatotoxic drugs, or ARDS-induced hypoxia. Transaminases could also be increased as a consequence of COVID-caused muscle breakdown. Nevertheless, an increased level of liver enzymes was associated with worse prognosis (34,38,39).

Although liver damage in mild cases of COVID-19 is often transient and transaminases may return to normal values without any specific medical treatment (36,40), studies have shown that patients with liver comorbidities had abnormal ALT and AST levels during the progression of the disease (40,41). According to our experience (Biochemical Laboratory, Health Center Pančevo), an increase in transaminase activities has been observed as a result of liver comorbidities, but also as a consequence of medications used in COVID-19 treatment. It should be noted that monitoring of liver enzymes is equally important in the post-COVID period, especially in patients with liver comorbidities.

Serum lactate dehydrogenase (LDH) activity is generally elevated in COVID-19 patients and particularly increased in cases with severe form of the disease (42,43).

According to these findings, LDH can be used as an early marker of infection, but also as a predictor of disease severity (44). Furthermore, LDH levels were independent predictors of clinical outcome (36). In particular, increased LDH was the most frequent abnormal laboratory finding in hospitalized COVID-19 patients (35,44,45). Next, LDH levels correlated with the degree of tissue damage (44,45) and were significantly higher in all patients with significant pulmonary radiological findings (44), indicating that elevation of LDH is important marker of lung tissue damage. Finally, the levels of LDH were higher in patients requiring mechanical ventilation and antiviral treatment. Our experiences are similar; an increase in LDH activity was pronounced in severe disease cases, and in a majority of patients with extremely high LDH values, radiologically confirmed interstitial pneumonia was present.

According to studies (35,45), reducing LDH levels predicts a favorable disease outcome. On the other hand, increased LDH on admission is associated with ARDS and in-hospital mortality (46). Our experience also shows that the degree of LDH increase could indicate a worse prognosis and lethal outcome. This was particularly pronounced in patients who had a very fast increase and extremely high values of LDH. These patients commonly have associated co-morbidities, such as hypertension and diabetes mellitus. Overall, the presented data clearly indicate that the enzymes AST, ALT and LDH are among the important biomarkers for patient management, such as diagnosis, assessment of disease severity and prognosis.

Serum glucose and lipid status parameters

Numerous studies suggested that individuals with comorbidities, such as diabetes, are prone to COVID-19, with a higher mortality risk (47). Additionally, the latest studies showed that COVID-19 causes alterations in glucose metabolism in approximately 50% of hospitalized COVID patients (48), including worsening of hyperglycemia in patients with diabetes, new hyperglycemia in non-diabetic patients, or hypoglycemia during hospitalization in both, with or without diabetes (49). Results confirmed that impaired glucose regulation correlated with increased severity and mortality in COVID-19 (49). Hyperglycemia stimulates the expression of ACE2 receptors, which are the major cell receptors for SARS-CoV-2, resulting in hyper-immune and inflammatory response. ACE2 receptors have been found in pancreas islets (50), as well as an increased viral load in pancreatic cells during the infection, potentially leading to pancreatic injury and disturbed insulin production (51). This may be one of the reasons for hyperglycemia in COVID-19 patients without a prior history of diabetes (52), which suggests that SARS-CoV-2 infection may contribute to the exacerbation or development of diabetes. Notably, diabetic patients with better glycemic control during COVID-19 infection were more likely to have an improvement in clinical outcome than those with poorly controlled glycemia (53).

Dyslipidemia can occur due to chronic inflammation caused by viral infection, and lipid metabolism plays a key role in the life cycle of the virus (54). Recent studies suggest that patients with dyslipidemia are at a higher risk of severe COVID-19, but also that

COVID-19 infection affects lipid profile, leading to dyslipidemia. A meta-analysis study conducted by Mahat R. et al. (55) showed that significant reductions in the levels of total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C) and low-density lipoprotein cholesterol (LDL-C) were associated with COVID-19 severity and mortality. However, the mechanism responsible for reduced TC, HDL-C and LDL-C levels in severe patients with COVID-19 is not clearly understood. It is well-known that the liver plays an important role in lipid metabolism, and SARS-CoV-2 can cause liver damage and thus disrupt lipoprotein uptake and biosynthesis. In a meta-analysis by Atmosudigdo et al. (56), which included 3663 patients with COVID-19, dyslipidemia occurred in 18% of patients. Furthermore, dyslipidemia was found to be associated with an increased risk of severe COVID-19 course and 39% higher risk of mortality (56). During COVID-19, a temporary disorder of lipid metabolism has been observed, mainly caused by impaired HDL structure and functionality (57). The researchers concluded that, during the severe course of COVID-19, HDL-C concentrations were dramatically reduced in non-survivors, while in recovered patients a gradual increase in the concentrations of HDL-C and LDL-C could be a consequence of an improvement in the patients' condition (58).

Laboratory diagnostics of COVID-19 in pediatric population

In comparison to adults, healthy children with COVID-19 are more likely to be asymptomatic or to have mild-to-moderate illness (59,60). Most of them did not require hospitalization, or the hospitalization passed without major complications. Studies conducted in the United States and Canada (61,62) showed that 83% of hospitalized children in pediatric ICUs had comorbidities, and only 0.5% of those children required treatment with invasive mechanical ventilation. The difference in the clinical condition between adult and pediatric patients can be explained by the anatomy and morphology of the respiratory tract, as well as by the activity of the immune system. The SARS-CoV-2 virus achieves its effects by binding to cellular ACE2 receptors. The distribution of these receptors depends on the maturity and differentiation of alveolar epithelial cells, which is reduced in the pediatric population, so binding and endocytosis of the virus is also reduced (63). According to the hypothesis set by Carsetti et al. (64), children's immune system is better prepared for new pathogens due to the presence of innate IgM antibodies and the ability to produce antibodies with broader reactivity. During the aging process, continuous antigenic stimulation and modification in the function and structure of the thymus leads to changes in the differentiation of native T cells into effector T cells and memory T cells, which causes a difference in the immune response (65). In pediatric age, the presence of other pathogens in the respiratory tract is inevitably more frequent and there is a competition of SARS-CoV-2 virus with these pathogens, so the growth and proliferation of SARS-CoV-2 virus is limited. As in the case of adult patients with asymptomatic and mild disease form, there are no specific biomarkers that can indicate the presence of the disease in SARS-CoV-2 positive children. A recent meta-analysis of clinical features and laboratory findings in 32 studies of COVID-19 pediatric patients showed that the majority of infected children had decreased hemoglobin oxygen

saturation and elevated D-dimer, while the prevalence of lymphopenia, leukopenia and elevated CRP concentrations were less common (66).

However, in April 2020, severe systemic hyperinflammatory disease was reported in Europe and United States, occurring approximately 4 weeks after acute SARS-CoV-2 infection (61). This novel entity was named multisystem inflammatory syndrome in children, by the Center for Disease Control and Prevention (CDC), or multisystem inflammatory disease in children or adolescent temporarily related to COVID-19 (MIS-C) by the World Health Organization (67). According to CDC, the case definition for MIS-C is: an individual aged less than 21 years presenting with prolonged fever, laboratory evidence of inflammation and evidence of clinically severe illness requiring hospitalization, with two or more organ system affected (i.e. cardiac, renal, gastrointestinal, respiratory, hematologic, neurological, dermatologic), and no alternative plausible diagnosis (68). Although information on the incidence is still limited, MIS-C appears to be a rare phenomenon with different racial and ethnical distribution (69). Neutralizing antibodies to SARS-CoV-2 have been found in the majority of patients with MIS-C, with greater titers of IgG than IgM antibodies. If reverse transcription polymerase chain reaction (RT-PCR) for SARS-CoV-2 from the respiratory tract were positive at the time of clinical presentation, the cycle threshold (Ct) values were high, indicating a reduced viral load (67,70).

Longitudinal laboratory investigations in MIS-C patients are essential for treatment and outcome prediction. The most important parameters are routine hematological and biochemistry parameters, including inflammatory, cardiac and coagulation markers. The majority of patients with MIS-C are characterized by lymphopenia, neutrophilia, anemia, and increased erythrocyte sedimentation rate. Nearly half of the patients show thrombocytopenia, and about a third of the patients have thrombocytosis. In addition, hypoalbuminemia, hyponatremia, elevated activity of aminotransferases and concentration of creatinine are commonly seen in these patients (71,72). All patients have increased levels of acute-phase reactants. The highest concentrations of CRP, fibrinogen, ferritin, and PCT have been reported in critically ill patients requiring admission to an ICU (73). A high concentration of D-dimer, associated with dysregulation of other coagulation parameters, is a common feature in severely ill patients, and indicates a risk of thrombosis, although reported clinical cases have been rare (74). Increased concentrations of IL-6 are found in most children with MIS-C, while concentrations of multiple cytokines, including IL-8, IL-17, IFN-γ, TNF-α and IL-10 are inconsistently determined in plasma (75,76). Considering that cardiac involvement is very common in MIS-C, elevation of cardiac markers is observed. Cardiac troponins (cTnI or cTnT) are elevated in many patients with MIS-C, while B-type natriuretic peptide and N-terminal proB-type natriuretic peptide (NT-pro-BNP) levels are markedly elevated in most of the cases, indicating cardiomyocyte damage and the extent of vasculopathy (71,74).

Post-COVID laboratory panel

So far, the epidemiological and clinical characteristics, pathogenesis, and complications in patients with COVID-19 in the acute phase have been explicitly described. However, a worrying aspect of this disease are the post-acute consequences of COVID-19, also called long COVID or "post-COVID syndrome". This syndrome includes persistent symptoms that could be related to residual inflammation (convalescent phase), organ damage, non-specific effects from the hospitalization or prolonged ventilation (post-intensive care syndrome), social isolation or impact on pre-existing health conditions (77). To date, there is not much data about the long-term course of the symptoms, quality of life, laboratory parameters, and association of symptoms with SARS-CoV-2 antibody titers at 12 months after acute infection (78). Although studies have evaluated different laboratory parameters in patients with "post-COVID syndrome" (79), a unique protocol as to which parameters should be monitored has not yet been defined. This depends on the symptoms that patients developed during or after COVID-19 infection. Common symptoms in people with long COVID are sensory (loss of taste and anosmia), neurological (problems with concentration and "brain fog"), and cardiorespiratory (fatigue, dyspnea, reduced exercise capacity) problems (78). Depending on the symptoms, patients should be monitored for complete blood count (CBC), especially hemoglobin, WBC and lymphocytes. Serum creatinine and estimated glomerular filtration rate (eGFR) should be determined to assess renal function, creatine kinase (CK), LDH and cardiac troponins for possible myocardial damage, and liver enzymes and total bilirubin for liver injury. Special tests should be performed in patients whose symptoms last longer than 12 weeks and when particular conditions are suspected: FT4 and TSH (thyroid dysfunction), NT-pro-BNP (differentiation of impaired pulmonary function and heart disease), D-dimer and fibrinogen (coagulation disorders), different autoantibodies (rheumatoid arthritis and other autoimmune diseases). An important fact is that the absence of abnormalities in laboratory findings does not rule out the existence of "post COVID syndrome".

Conclusion

As the pandemic has developed and lasted for more than two years continually, due to frequent mutations of the virus, there has been a constant need to develop and adjust the strategy for COVID-19 diagnosis, treatment and monitoring. The results of routine laboratory tests are essential for assessing the severity of the disease, selecting appropriate therapeutic procedures and monitoring treatment response. The main laboratory parameters for the diagnosis and management of COVID-19 include biomarkers of inflammation, tissue damage and impaired coagulation. In addition, certain inflammatory, cardiac and coagulation parameters may indicate disease progression and adverse outcome, thus providing important prognostic information. Despite current knowledge, additional studies are needed to establish the optimal use of laboratory analyses, as well as to confirm the effectiveness of laboratory tests to predict the clinical outcome of the patients.

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Uloga laboratorijskih biomarkera u dijagnostici i praćenju pacijenata sa COVID-19

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Kratak sadržaj

Novi koronavirus SARS-CoV-2 je uzročnik lako prenosive i veoma heterogene infekcije respiratornog trakta, koju karakteriše širok spektar kliničkih manifestacija, različitog stepena težine. Medicinske laboratorije igraju važnu ulogu u ranoj dijagnostici i praćenju pacijenata sa COVID-19. Rezultati nekoliko laboratorijskih testova su od suštinskog značaja za procenu težine bolesti, odabir odgovarajućih terapijskih procedura i praćenje odgovora na terapiju. Rutinska laboratorijska ispitivanja kod pacijenata sa COVID-19 uključuju biomarkere akutne faze, hematološke i biohemijske parametre koji ukazuju na oštećenje tkiva. Cilj ovog rada je da se prikaže uloga tih biomarkera u dijagnostici i praćenju toka bolesti kod odraslih i pedijatrijskih pacijenata sa COVID-19.

Ključne reči: biomarker, COVID-19, laboratorijska dijagnostika