

The Use of Biomarkers in Diagnosis of COVID-19

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Perspective

A systematic review of the literature was carried out to identify relevant articles using six different databases. Keywords to refine the search included COVID-19, SARS-CoV-2, Biomarkers, among others. Thirty-four relevant articles were identified which reviewed the following biomarkers: C-reactive protein, serum amyloid A, interleukin-6, lactate dehydrogenase, neutrophil-to-lymphocyte ratio, D-dimer, cardiac troponin, renal biomarkers, lymphocytes and platelet count. Of these, all but two, showed significantly higher levels in patients with severe complications of COVID-19 infection compared to their non-severe counterparts. Lymphocytes and platelet count showed significantly lower levels in severe patients compared to non-severe patient.

As of the 28th April 2020, the COVID-19 pandemic has infiltrated over 200 countries and affected over three million confirmed people. We review different biomarkers to evaluate if they are able to predict clinical outcomes and correlate with the severity of COVID-19 disease.

Common laboratory values may provide key insights into patients with COVID-19, the illness caused by the SARS-CoV-2 virus, as well as the viral infection itself. Studies reveal telling associations between severe disease and levels of procalcitonin (PCT) and of cardiovascular markers, as well as thrombocytopenia. Analytes such as D-dimer might signify a higher mortality risk factor in hospitalized patients, while others may be useful in explaining epidemiological findings in COVID-19.

Several studies have explored these associations. In one meta-analysis, Italian researchers reported that risk of severe SARS-CoV-2 infection was

nearly five times higher in COVID-19 patients with raised PCT levels. PCT synthesis is inhibited by interferon-gamma (INF)- γ , whose concentration increases during viral infections. "This explains why the frequency of raised PCT (>0.5mg/L) levels in COVID-19 patients at admittance in a cohort of 1,099 Chinese patients has been reported as 5.5%," study co-author Mario Plebani, MD, of the University Hospital of Padova, Italy, told CLN Stat.

Angiotensin-converting enzyme polymorphism (ACE) is a known factor in coronavirus infection. "The angiotensin-converting 1 (ACE1) enzyme is characterized by a genetic deletion/insertion (D/I) polymorphism in intron 16, which is associated with alterations in circulating and tissue concentrations of ACE," authors of another paper indicated. They theorized that the differences in prevalence of COVID-19 among continental European countries might in part be due to D/I genotype distribution variability.

Given the frequency and nonspecific nature of abnormal troponin results among patients with COVID-19 infection, clinicians are advised to only measure troponin if the diagnosis of acute myocardial infarction (MI) is being considered on clinical grounds, and an abnormal troponin should not be considered evidence for an acute MI without corroborating evidence," he indicated, offering similar advice for BNP or NT-proBNP. The meta-analysis only covered four studies. Study authors urged for more trials to validate these findings. In the meantime, clinical labs "should be aware that PCT measurement, namely increased levels, would reflect bacterial coinfection in COVID-19 patients developing severe form of disease," Plebani said. Platelet count is a simple, economic, rapid, and accessible laboratory parameter to discriminate between COVID-19 patients with and without severe disease, he added. Despite these findings, Januzzi cautioned against testing patients for hs-cTnI or natriuretic peptides in the absence of solid clinical information.

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