Commentary

CLINICAL INVESTIGATION

# Difference between COVID-19 1-year mortality and lung injury biomarkers

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## Commentary

The first wave of the COVID-19 worldwide pandemic began in late 2019. We assessed the relationships between the biomarkers, COVID-19 pneumonia severity, and 1-year mortality in this cross-sectional and observational investigation. 2019 coronavirus sickness was brought on by SARS-CoV-2 (COVID-19). The first wave of the COVID-19 worldwide pandemic began in late 2019. COVID-19 frequently exhibits lung injury, such as pneumonitis with histological confirmation or interstitial damage and fibrosis. In COVID-19, pneumonia is the main cause of morbidity and mortality. Patients with COVID-19 may have asymptomatic or overt adult respiratory distress syndrome that necessitates intubation due to lung involvement. The efficient use of medical resources depends on knowing how severely the lung has been affected. Several noninvasive imaging techniques, including chest radiography, Computed Tomography (CT), and lung ultrasonography, can be used to evaluate the extent of lung injury in COVID-19. The CT of the chest serves as the foundation for many radiological results.

Depending on the stage and severity of COVID-19, there are differences in the lung involvement and radiological findings. The GGO is usually seen in the early stages, whereas consolidation predominates in the latter stages, particularly in severe and critical patients. The inflammatory exudate-infiltrated epithelium and alveoli are damaged, as shown by the CT findings. Particularly in the early stages of the disease, chest CT has a higher sensitivity and greater proficiency in identifying the distinctive features. However, because of the expense and radiation exposure, a CT scan cannot be utilized as a standard screening tool in the Emergency Department (ED).

To screen, diagnose, and treat patients with significant lung injury in the ER, confirmatory biomarkers are required. Similar to this, it is still unclear what factors contributed to the poor COVID-19 outcomes during the pandemic's initial wave. In the majority of cases, the pathophysiology of severe and quick lung damage is unknown. SARS-CoV-2 causes endotheliitis in the heart, lung, kidney, liver, and other tissues, according to histological and postmortem studies. In COVID-19, the pulmonary vascular system exhibits thrombosis, microvascular dysfunction, and inflammation together. To risk stratify the hospitalized COVID patients, several biomarkers are used. The numerous biological systems that these biomarkers reflect include tissue injury (Lactate Dehydrogenase (LDH) and cardiac troponins), inflammation (ferritin), White Blood Cell Count (WBC), Neutrophil-To-Lymphocyte Ratio (NLR), and High-Sensitive C-Reactive Protein (CRP).

The COVID-19 phenotype has altered dramatically over the past two years. The complexity of COVID-19 has increased due to the availability of efficient vaccines and resources, efficient treatment strategies, and the discovery of novel variants with various clinical characteristics. The COVID-19 first wave's severe outcome indicators are still of interest. For COVID-19 with extensive lung involvement, there is a need for conveniently accessible and inexpensive biomarkers. We provide the COVID-19 predictors of severe CT-SS and 1-year mortality in the current investigation.

Poor outcomes after COVID-19 are indicated by severe lung injury and 1-year death. According to the study, there is a difference between 1-year mortality

in COVID-19 and the biomarkers of lung injury. The COVID-19 biomarker LDH is sensitive and specific for identifying patients with significant lung injury. However, 1-year mortality is not correlated with LDH or CT-SS. The sole biomarker identified in the first wave of COVID-19 that was related to mortality is D-dimer. D-dimer is a tiny protein fragment and a byproduct of fibrin degradation. After fibrinolysis breaks down a blood clot, d-dimer is found in the bloodstream. An intriguing paradox is displayed by 8 COVID-19 patients. In COVID-19, the clinical phenotype is defined by a hypercoagulable state, reduced fibrinolytic ability, and paradoxical elevation of D-dimer.

D-dimer levels are linked to disease severity and mortality in COVID-19, according to several retrospective investigations. The intra-alveolar fibrin accumulation is a distinctive feature of the lung injury in COVID-19. The fibrin protein degradation product known as d-dimer is created by plasmin during the fibrinolytic process. An indirect biomarker of thrombosis is d-dimer. The circulation's levels of D-dimers rise as a result of fibrinolysis and the proteolytic degradation of fibrin in the capillary beds of the lung segments.

Global statistics show that there are regional variations in the risk factors, phenotype, and outcomes of COVID-19. Early in 2021, Turkey began immunizing against COVID-19 with the Corona Vac vaccine. Priority was given to elderly patients with chronic conditions and healthcare staff when administering vaccinations. Later in 2021, Pfizer-BioNTech vaccination dosages became widely accessible. The disease phenotype and course of COVID-19 drastically modified after immunization. Similar to this, during pandemics new variations, efficient treatments, and international responses developed. Since the initial wave of COVID-19, the phenotypic has undergone tremendous alteration. After 2021, the delta and omicron versions entered widespread circulation. At the time of our investigation, the first varieties dominated the world.

A common metabolic measurement included in the standard laboratory panel is LDH. In the absence of imaging results, estimating the degree of lung injury is difficult. Every patient who enters the emergency room with a clinical suspicion of COVID-19 cannot be given a CT scan. Two main worries are the price and radiation exposure. According to the study, LDH is a sensitive and accurate biomarker for predicting a severe CT-SS. In terms of predicting severe CT-SS, LDH performs better than hs-CRP, d-dimer, cTn, uric acid, and ferritin. Respiratory rate or room air oxygen saturation are employed as early screening criteria in the first wave of pandemics. LDH is a common and accessible biomarker everywhere. The initial evaluation methods may benefit from including LDH level.

We look at a representative sample of Istanbul, Turkey COVID-19 patients. The study's sample size is modest and cross-sectional, which is one of its drawbacks. The COVID-19 pandemic has different treatment protocols in each nation. Biomarker levels may be impacted by therapies. Within the countries, there are still more differences between the various socioeconomic levels and areas. Another drawback is that it is impossible to rule out the confounding effects of genetic and environmental risk factors. Pre analytical and analytical factors may have an impact on biomarker levels in laboratory research. On the same admission as COVID-19, the biomarker levels were measured. The COVID-19 phenotype has drastically changed since the research period. The phenotype of COVID-19 has been altered by vaccination, novel variations, efficient treatments, and the worldwide reaction to pandemics.

A pro inflammatory disease called COVID-19 may cause biomarker elevations in multiple important pathways. In this study, we looked at the biomarkers in relation to pneumonia severity and 1-year mortality in COVID-19 patients who were hospitalized. In the initial COVID-19 wave, the one-year death rate was high. The 1-year mortality could only be predicted by the d-dimer parameter. A sensitive and particular biomarker of severe CT-SS was LDH. LDH and CT-SS, however, were not linked to 1-year mortality. To examine the effectiveness of new criteria and algorithms that can enhance COVID-19 results, larger research is required.