SHORT COMMUNICATION

Assessment of hepcidin levels in COVID-19 patients entering the intensive care unit

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ABSTRACT

The COVID-19 coronavirus disease's clinical spectrum ranges from a minor illness to a major illness. Multi-organ failure brought on by the so-called "cytokine storm," respiratory failure, septic shock, and/or septic shock can be seen in patients with severe diseases. The main way that inflammatory cytokines affect iron metabolism is through increasing the production of the rarely measured hormone peptide hepcidin. High hepcidin levels have been associated with the severity of COVID-19. In this study, a sample of COVID-19 patients who had been admitted to the Intensive Care Unit (ICU) at the Policlinico Tor Vergata in Rome, Italy, had their levels of hepcidin retrospectively examined. 38 people were included in the trial between November 2020 and May 2021. Based on the clinical outcome, patients were split into two groups: survivors and non-survivors. A number of common laboratory parameters were also evaluated while the patients were in the ICU, and the levels of these indicators were connected to the outcomes. There were statistically significant differences in the levels of hepcidin, D-dimer, IL-6, LDH, NLR, neutrophils, CRP, TNF-, and transferrin across the groups. Particularly, hepcidin levels revealed significantly different median values (88 ng/mL *vs* 146 ng/mL) between survivors and non-survivors. Using ROC curves analysis, it was discovered that hepcidin was an effective biomarker for predicting the severity and mortality of COVID-19 in ICU patients, with sensitivity and specificity values of 74% and 76%, respectively, at a cut-off of 127 (ng/mL).

Key Words: *COVID-19; Hepcidin; ICU*

INTRODUCTION

The 2019 Coronavirus Disease (COVID-19), which is caused by the recently identified human coronavirus SARS-CoV-2 [1], can present as a non-life-threatening sickness or as a life-threatening illness, depending on its clinical characteristics. Patients with critical illnesses may experience respiratory failure, septic shock, and/or multi-organ failure as a result of the alleged "cytokine storm." This heightened inflammatory response is indicated by elevated levels of many cytokines, including IL-2, IL-6, IL-7, interferon-c inducible protein-10, macrophage inflammatory protein 1, Tumour Necrosis Factor (TNF), monocyte chemoattractant protein 1, and granulocyte colony-stimulating factor. The 25-amino acid peptide hepcidin is the main regulator of iron absorption and distribution to tissues produced mostly by inflammatory cytokines in liver cells. Hepcidin stimulates the synthesis of iron, which is essential for iron metabolism.

Serum ferritin is a critical measure of inflammation and a sign of iron status. Elevated ferritin levels are linked to significant mortality in COVID-19 patients, which is reflected in aberrant iron metabolism [1]. Hepcidin controls the export of cellular iron to plasma and extracellular fluid through ferroportin, a hepcidin receptor and cellular iron exporter in vertebrates. The homeostatic regulation of hepcidin levels is regulated by iron and erythropoietic activity. Hepcidin, which is created when the body has too much iron, stops the intestines from absorbing iron. Instead, iron deficiency causes hepcidin suppression, which aids in iron absorption from food and replenishing iron stores. The host's defence mechanism to remove iron from the infecting microorganisms is likely responsible for the spike in hepcidin levels that occur during inflammation and infection. Hepcidin uses macrophages to sequester the majority of the iron. This phenomenon is well known in bacterial infections, but its relevance in viral infections is unclear. Iron metabolism has an effect on the immune system's functionality. Lymphocytes are primed by contact with antigen-presenting cells, and they need iron to produce a strong and effective cellular and humoral response. Despite playing a crucial role in the control of iron metabolism, hepcidin is rarely examined in clinical laboratories. The severity of the disease is connected with higher hepcidin levels in COVID-19 patients, according to early studies. Low serum iron levels and severe hypoxemia in Intensive Care Unit (ICU) patients have both been associated with COVID-19 severity and death

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[2]. In light of this, the goal of the study was to retrospectively evaluate the levels of hepcidin in a group of COVID-19 patients who had been admitted to the ICU at the Policlinico Tor Vergata in Rome, Italy.

DISCUSSION

Since COVID-19 initially appeared at the end of 2019, millions of people have perished as a result of it. Despite the fact that the majority of patients have mild symptoms and a good prognosis, a small number of patients encounter more severe clinical circumstances, such as severe pneumonia, acute respiratory distress, or multiorgan failure with mortality [3]. These critically ill patients exhibit a "cytokine storm," or a severe inflammatory response accompanied by the release of many cytokines. This inflammatory response alters several haematological and biochemical markers. Recent studies suggest that the essential iron metabolism regulator hepcidin may be able to predict the severity of the illness and mortality in COVID-19 patients. Patients with severe illnesses or poor outcomes did, in fact, have higher serum hepcidin levels. In this study, we evaluated the typical haematological, biochemical, and hepcidin profiles in a group of COVID-19 patients who were admitted to the ICU with severe pneumonia. Our results show statistically significant differences in the level of several markers between survivors and non-survivors. The non-survivors had higher levels of D-dimer, IL-6, LDH, NLR, CRP, TNF-, and transferrin than the survivors did. Elevated D-dimer levels are linked to a poor prognosis and the need for intensive care unit care in COVID-19 patients [4]. According to a recent meta-analysis, patients who died had higher D-dimer levels than those who survived, which is consistent with our findings. LDH is a recognisable marker of the severity and mortality of COVID-19. This conclusion was validated by a second meta-analysis that also revealed that LDH is considerably higher in ICU patients compared to non-ICU patients and in non-survival patients compared to survival patients. It can therefore be used to forecast survival. The significance of the prognostic value of LDH level in COVID-19 patients was confirmed by a sizeable retrospective analysis that was published at the beginning of 2022. The higher level of LDH in the non-survivors' group in our study compared to the survivors' group supports the latter claim. Numerous academic articles have addressed the connection between IL-6 and TNF- and the severity and mortality of COVID-19. Elevated levels of these two biomarkers significantly increase the risk of illness severity and mortality, as was recently assessed in a study with a large sample size. A considerably higher level of IL-6 was present in patients who did not survive compared to those who did. The TNF level displayed a similar pattern. Our results provide credence to the idea that elevated IL-6 and TNF- levels are associated with a bad prognosis. In addition, elevated CRP levels, raised neutrophil and NLR numbers, and a dismal prognosis are all signs of a severe disease. These observations are supported by the greater levels of these laboratory indices in the non-survivors' group in our data. Lower levels of transferrin have been associated with increased inflammation and the progression of the disease. Our study found that group survivors had median higher transferrin levels than group non-survivors, suggesting that low transferrin levels may be a sign of a more serious illness. Recent studieshave looked at hepcidin as a potential indicator of mortality and the severity of the disease in COVID-19 patients. All of the patients in our study who required invasive mechanical ventilation and were admitted to the ICU due to severe pneumonia had elevated hepcidin levels, and these levels were significantly greater in the patients who did not survive. Finally, ROC curve research showed that IL-6 and LDH have the best sensitivity and specificity. Hepcidin measurement, however, exhibited a sensitivity of 74% and a specificity of 76% at a cut-off value of >127 ng/mL, demonstrating that it is a useful biomarker for predicting the severity and outcome of COVID-19 in ICU patients. Furthermore, hepcidin could be used with Serum Amyloid Protein (SAA) and Mid-Regional Proadrenomedullin (MR-proADM), two recently discovered new inflammatory biomarkers that have been investigated for predicting death in COVID-19 patients.

Create a formula [5]. SAA levels were higher in deceased patients than in living ones. Similar to this, people who had MR-proADM levels greater than 1105 nmol/L had a threefold increased risk of passing away. Despite the fact that these indicators of disease severity are still important, our research did not uncover any statistically significant changes in the levels of fibrinogen, AST, ferritin, leucocytes, lymphocytes, or sideremia between the groups of survivors and non-survivors. One of the study's weaknesses is the small sample size, which may reduce the validity of the results. However, this study confirms the effectiveness of particular biomarkers in predicting the severity and death of COVID-19 patients admitted to the ICU and gives critical information about the utility of hepcidin measurement in these patients. All patients with elevated hepcidin levels had a bad prognosis.

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