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**Short communication**

# Hormonal dysregulation in COVID-19: A comparative analysis of cortisol, growth hormone, and IGF-1 levels across different severities of illness

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## *Abstract*

***Background:*** COVID-19 has multiple effects on the endocrine system, particularly altering the levels of growth hormone (GH), insulin-like growth factor-1 (IGF-1), and cortisol, which may influence disease severity and prognosis. This study aimed to evaluate circulating levels of GH, IGF-1, and cortisol in COVID-19 patients categorized by disease severity, including outpatients, inpatients, and intensive care unit (ICU) patients.

***Methods:*** A total of 118 confirmed COVID-19 patients were divided into three groups: outpatients, inpatients, and ICU patients. Serum levels of GH, IGF-1, and cortisol were measured using ELISA and statistically compared across groups. Correlations among these hormones were also analyzed.

***Results:*** Cortisol levels were highest in inpatients, intermediate in ICU patients, and lowest in outpatients. GH levels were reduced in ICU patients compared to other groups, with no difference between inpatients and outpatients. IGF-1 levels were highest in outpatients, followed by inpatients, and lowest in ICU patients. A significant inverse correlation between cortisol and IGF-1 was observed only in ICU patients ($p = 0.013$).

***Conclusion:*** COVID-19 severity is associated with dysregulation of GH, IGF-1, and cortisol. The inverse cortisol–IGF-1 correlation in ICU patients suggests the impact of physiological stress on hormonal imbalance and impaired recovery. Endocrine interventions may improve clinical outcomes in severe cases.

***Keywords:*** COVID-19, Growth hormone (GH), Insulin-like growth factor-1 (IGF-1), Cortisol, Disease severity.

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A novel virus that swept the globe as a pandemic between December 2019 and May 2023, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), or COVID-19 (1), is still under investigation by scientists. Despite the official end of the pandemic, the virus remains a significant challenge due to recurring waves and long-term complications, underscoring the need for continued research. Growing evidence suggests that dysregulated inflammation is a central mechanism in the progression and severity of COVID-19 (2). Accordingly, immune-targeting anti-inflammatory treatments have been shown to improve survival (3).

Beyond immune dysfunction, COVID-19 has been shown to disrupt several endocrine pathways that are essential for metabolic regulation, stress adaptation, and tissue repair (1). Among these, the growth hormone (GH)/insulin-like growth factor-1 (IGF-1) axis and cortisol regulation have attracted growing attention (4, 5). These hormonal systems are closely integrated with immune and inflammatory responses, and their dysregulation may contribute to disease severity, impaired recovery, and adverse outcomes in COVID-19 patients (6).

The GH/IGF-1 axis is a tightly regulated endocrine pathway that plays a key role in growth, metabolism, tissue repair and immune modulation (7, 8). COVID-19 can disrupt that axis. Several studies have reported reduced circulating IGF-1 levels in patients with severe COVID-19, and lower IGF-1 concentrations have been associated with increased mortality and poor clinical outcomes (9). These alterations are thought to be driven by systemic inflammation, cytokine-mediated suppression of GH signaling, and hepatic dysfunction during acute infection. Reduced IGF-1 levels may contribute to sarcopenia, malnutrition, and delayed recovery, particularly in critically ill patients experiencing prolonged immobilization and catabolic stress (10). Cortisol, the primary glucocorticoid hormone, is a central component of the stress response and is regulated by the hypothalamic–pituitary–adrenal (HPA) axis. During SARS-CoV-2 infection, activation of the HPA axis commonly results in elevated cortisol levels, reflecting the body's response to severe inflammation and physiological stress (11). Elevated cortisol concentrations have been associated with increased disease severity and mortality in COVID-19 (12). While acute cortisol elevation may exert protective anti-inflammatory effects, sustained hypercortisolemia can suppress immune function, worsen metabolic disturbances, and contribute to complications such as hyperglycemia and cardiovascular dysfunction (13). In some patients, cortisol dysregulation resembles a pseudo-Cushing's state, highlighting the complex interaction between inflammation and endocrine regulation during COVID-19 (14). Concerns have also been raised regarding the potential long-term endocrine consequences of COVID-19. Post-COVID-19 condition, or long COVID, is frequently characterized by persistent fatigue, sleep disturbances, and reduced functional capacity, symptoms that may be related to prolonged HPA axis activation and hormonal imbalance (15). However, data regarding persistent alterations in the GH/IGF-1 axis and cortisol levels across different severities of COVID-19 remain limited and inconsistent. Therefore, the objective of the present study is to evaluate alterations in the endocrine system associated with COVID-19, with a particular focus on the GH/IGF-1 axis and cortisol. By comparing hormone levels among ICU, inpatient, and outpatient COVID-19 patients, this study aims to clarify the relationship between endocrine dysregulation and disease severity. Improved understanding of these hormonal changes may provide insight into the pathophysiology of COVID-19 and support the integration of endocrine evaluation into comprehensive patient management strategies.

## Methods

**Study population:** This study was conducted on patients with COVID-19 at a designated hospital at Arak University of Medical Sciences (Iran) between October and December 2021. 118 patients with COVID-19 were recruited for the study. The patients were divided into three groups: Group I: Mild COVID-19, diagnosed based on mild clinical symptoms without pneumonia. This group included outpatients referred to the hospital clinic. Group II: Moderate COVID-19 was diagnosed with respiratory symptoms, fever, and pneumonia. These patients were admitted to the hospital's internal medicine ward. This group was referred to as Inpatients. Group III: Severe or critical COVID-19 was diagnosed with resting oxygen saturation of less than 90% or respiratory failure and pulmonary lesions on imaging. These patients were admitted to the hospital's ICU. This group was referred to the ICU patients.

In all groups, SARS-CoV-2 infection was verified by real-time reverse transcription polymerase chain reaction (RT-PCR) assays performed on nasopharyngeal and/or oropharyngeal swab specimens. A diagnosis of COVID-19 was established based on positive RT-PCR findings. In groups II and III, Chest CT imaging was also performed. The exclusion criteria were: 1. patients with BMI ≥ 35, 2. diabetic patients, 3. false-negative RT-PCR results for SARS-CoV-2 infection (patients with outward symptoms), 4. Age less than 30 years, 5. Chronic disorder of the adrenal or pituitary glands.

**Analytical methods:** The participants' laboratory tests were performed in the morning under fasting conditions. Blood samples were collected and immediately centrifuged at 2500 rpm; the resulting serum was aliquoted and stored at −80 °C until analysis. Serum cortisol, GH, and IGF-1 were measured by ELISA using commercially available kits (Diazist, Iran; DiaMetra, Italy). Levels of hormones were reported in ng/mL.

**Statistical analysis:** In this study, laboratory parameters were compared between patients with severe and non-severe forms of COVID-19. The Shapiro–Wilk test was applied to examine the normality of the parameters. Group comparisons were performed using the Kruskal–Wallis test, and post hoc analyses were carried out with the Mann–Whitney U test. Correlations between the studied variables were evaluated through Spearman's correlation test. Statistical analyses were performed using SPSS software, Version 23 (SPSS Inc., Chicago), and $p < 0.05$ was considered statistically significant.

## Results

We included 118 patients with positive RT-PCR results for SARS-CoV-2 in our study. Participants included 68 women (57.6%) and 50 men (42.4%). The patients were categorized as outpatients, inpatients, and ICU patients. The median age of the patients was 57 years (range: 35–81 years). Among the 118 patients, 52 (44.1%) were outpatients, 42 (35.6%) were inpatients, and 24 (20.3%) were admitted to the ICU. There were no significant differences among the three groups in gender, BMI, or age ($p > 0.05$).

**Serum hormone profiles in the patients with COVID-19**

**Cortisol:** Analysis of results showed that the mean cortisol level was significantly lower in outpatients compared to inpatients (154.8±13 vs. 286.89±25, P= 0.001, mean±SEM) and ICU patients (154.8±13 vs. 228±28, P= 0.016, mean±SEM). Interestingly, the mean cortisol level was not statistically significant in inpatients than in ICU patients (286.89±25 vs. 228±28, P= 0.24, mean±SEM). The results are depicted in figure 1A.

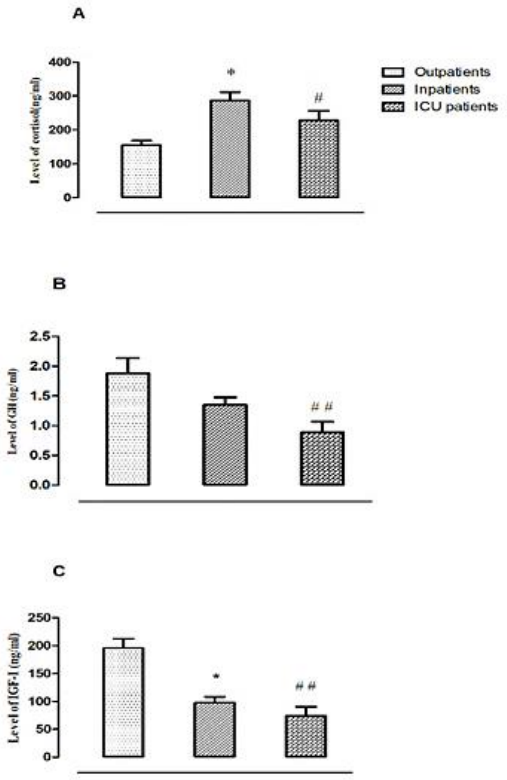
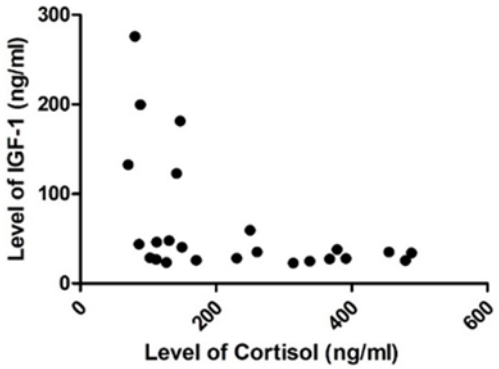



**Figure 1. Comparison of the levels of cortisol (A), GH (B), and IGF-1 (C) hormones between the outpatient, inpatient, and ICU patients with COVID-19. Data are presented in mean ± SEM**.* shows a significant difference between the inpatient and outpatient groups. #shows a significant difference between the ICU and outpatient groups. ##shows a significant difference between ICU patients and outpatient and inpatient groups.

**Growth hormone (GH):** Our results showed that the mean GH level was significantly lower in ICU patients compared to outpatients (0.89±0.18 vs. 1.88±0.26, P=0.001, mean±SEM) and inpatients (0.89±0.18 vs. 1.35±0.13, P=0.008, mean±SEM). However, no significant difference was observed in GH between inpatients and outpatients (1.88±0.26 vs. 1.35±0.13, P=0.37, mean±SEM). The results are depicted in figure 1B.

**Insulin growth factor (IGF):** Analysis of results revealed that the mean IGF-1 level significantly increased in outpatients compared with inpatients (195±17.06 vs. 97.63±10.45, P =0.001, mean±SEM) and ICU patients (195±17.06 vs. 74.17±16.29, P=0.001, mean±SEM). On the other hand, the mean IGF-1 level was significantly higher in inpatients than in ICU patients (97.63±10.45 vs. 74.17±16, P=0.006, mean±SEM). The results are depicted in figure 1C.

**Correlation between hormone levels by group:** Based on the Spearman's correlation analysis, cortisol, GH, and IGF-1 showed no significant correlations in both the outpatient and inpatient groups ($p > 0.05$). In ICU patients, a significant negative correlation was detected between cortisol and IGF-1 ($p < 0.013$), whereas no significant associations were identified between cortisol and GH ($p > 0.05$) or between GH and IGF-1 ($p > 0.05$), figure 2.




**Figurer 2 .Scatter plot of inverse correlation between cortisol and IGF-1 in ICU patients**

## Discussion

SARS-CoV-2, the causative agent of the global COVID-19 pandemic, remains the subject of extensive research in the scientific community. The virus' impact is not limited to the respiratory system, and increasing attention is being paid to its effects on endocrine function. This study examined hormonal changes, focusing on cortisol, GH, and IGF-1 levels. The levels of cortisol, GH, and IGF-1, along with their correlation with COVID-19 severity, were

evaluated in 118 patients at different stages of disease progression. The levels of cortisol, GH, and IGF-1 differed significantly among three distinct groups: outpatients, inpatients, and ICU patients. Elevated cortisol levels in COVID-19 patients significantly modulate inflammatory responses through various mechanisms. Cortisol, a glucocorticoid hormone, is often elevated due to the stress response triggered by SARS-CoV-2 infection, leading to a state of relative hypercortisolism (RHC) that exacerbates inflammation and immune dysregulation, thereby reducing the ability of the immune system to effectively clear the virus (5). Continued high levels of cortisol may increase inflammation and worsen the eventual course, leading to outcomes such as hyperglycemia, cardiovascular complications, and pulmonary complications. Modulation of cortisol levels may help balance these effects and improve therapeutic strategies (12).

Peak levels of cortisol were noted in inpatients. Conversely, the lowest cortisol concentration was observed in outpatients. Cortisol is a stress-response hormone and may play a role in disease severity. According to several studies, elevated cortisol levels are correlated with higher case-fatality rates, longer hospital stays, and an increased need for ICU care among COVID-19 patients (16 ,13). The highest cortisol concentration observed in our study was 665.04 ng/mL, mainly in inpatients. The average cortisol level among inpatients was 286.88 ng/mL. In this regard, our result was similar to that obtained by Sengel et al. (2023), who found that patients with COVID-19 had a higher baseline cortisol level than healthy controls (17). This finding is also supported by previous studies, including one reporting significantly higher cortisol levels in COVID-19 patients, that were associated with increased mortality and disease severity (18). Several physiological and clinical mechanisms may explain why cortisol levels were higher in inpatients compared with ICU patients. In critically ill individuals, prolonged systemic stress and inflammation may lead to dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis. While cortisol levels typically increase during the early acute phase of severe infection, sustained critical illness may impair adrenal responsiveness, a condition described as relative adrenal insufficiency or critical illness–related corticosteroid insufficiency (CIRCI), resulting in an inadequate cortisol response despite greater disease severity (19, 20). In addition, the frequent use of exogenous corticosteroids in ICU patients may suppress endogenous cortisol production through negative feedback inhibition of the HPA axis, leading to lower measured serum cortisol levels (21). Timing-related differences in hormonal stress responses may also contribute, as cortisol secretion peaks early during acute stress and may attenuate during prolonged critical illness (22). Furthermore, systemic inflammation is associated with reduced circulating cortisol-binding globulin (CBG), which can lower measured total serum cortisol concentrations even when biologically active free cortisol is preserved. Consequently, this phenomenon can complicate comparisons of cortisol levels across different disease-severity groups (23). Collectively, these physiological, therapeutic, and timing-related factors may help explain the observed pattern of higher cortisol levels in inpatients compared with ICU patients.

The lowest mean GH concentration observed in our study was 0.89 ng/mL in ICU patients. In this regard, the aggressive nature of coronavirus disease may lead to a significant decrease in GH levels, consistent with the scientific literature. According to Baykan et al. (2022), patients with severe COVID-19, characterized by pulmonary involvement, exhibit significantly lower serum GH levels than those without lung involvement. Therefore, GH deficiency is common among groups at higher risk of severe COVID-19, including the elderly, males, individuals with obesity, diabetes, or immunocompromised conditions (7). The highest GH levels were measured in outpatients, while the lowest GH levels were measured in ICU patients. Several studies also suggested a possible association between GH and COVID-19 severity. Lubrano (2020) suggested that GH deficiency, a common characteristic of older men with obesity, may be associated with a worse COVID-19 outcome (24). Yuen (2020) reported that the use of GH therapy may be of importance in COVID-19 patients with low GH levels (16). In outpatients, the highest levels of GH were observed, whereas the lowest GH levels were recorded in ICU patients. Research conducted by Baykan et al. has indicated that in COVID-19 patients, lower levels of GH and IGF-1 are associated with lung involvement (7). Another study by Ilias et al. (2021) found that low IGF-1 levels were associated with poor COVID-19 outcomes; therefore, IGF-1 concentration appears to be inversely related to ICU admission rates, disease severity, and mortality (9). The highest IGF-1 value measured in the current study, 611.39 ng/mL, was observed in outpatients. The result is consistent with previous reports by Li et al. (2022), in which higher genetically predicted IGF-1 levels were associated with reduced risk of COVID-19 susceptibility and hospitalization (10). No significant correlations among cortisol, GH, and IGF-1 levels were found in any of the groups, except in ICU patients, in whom a significant negative correlation between cortisol and IGF-1 levels was observed. Scientific literature supports this

finding, and in most studies, cortisol and IGF-1 levels show an inverse correlation (25). This issue is especially critical for patients with COVID-19, as they are exposed to immune system dysregulation and psychological stress. In stressed patients such as those with COVID-19, the cortisol-IGF-1 balance is disturbed, which can, in turn, interfere with tissue repair. This disruption may lead to prolonged inflammation and hinder recovery, complicating the overall healing process (26). Generally, IGF-1 supports immune function, whereas cortisol has immunosuppressive actions. The balance between these two determines individual variability in host defense against infections and tissue repair (27). Several theories exist regarding the interaction mechanisms between these two hormones. One such theory suggests that cortisol, as part of the HPA axis, can suppress the production of GH by the pituitary gland. Since GH stimulates the production of IGF-1, this ultimately leads to a decrease in IGF-1 levels (27).

The significant inverse correlation between cortisol and IGF-1 observed in ICU patients highlights the profound impact of physiological stress on hormonal regulation. Cortisol, a catabolic hormone, increases during the stress response, while IGF-1, an anabolic hormone, decreases, reflecting a metabolic shift toward catabolism in critically ill patients, consistent with endocrine alterations observed during systemic inflammation (Mehdi et al., 2025). (28), and by Losef et al. (2024), who reported elevated cortisol and reduced IGF-1 levels in high-stress ICU scenarios (5). Clinically, these insights underscore the potential of stress management strategies to modulate cortisol levels and potentially improve endocrine balance (Rogerson et al., 2024) (29). Further research should investigate individualized interventions to address these hormonal changes and enhance recovery outcomes in ICU settings.

Our study has several limitations that should be acknowledged. First, the number of patients in the ICU group was relatively small, partly due to patient mortality during the study period; therefore, the findings should be interpreted with caution. Second, hormone levels were measured only once during the acute phase of COVID-19. Due to clinical and logistical constraints—particularly in hospitalized and critically ill patients—repeated sampling was not feasible. Consequently, this study does not provide information on the dynamic changes in cortisol, GH, or IGF-1 over time. Longitudinal follow-up studies are warranted to assess the trajectory of endocrine alterations during recovery and to determine their potential role in post-COVID or long COVID conditions. A key limitation of this study is that the exclusion of patients with diabetes, severe obesity (BMI ≥ 35), and endocrine disorders resulted in a cohort that is not representative of the typical high-risk COVID-19 population. Consequently, our findings should be interpreted within the context of non-diabetic, non-obese patients without known endocrine disorders and should not be directly extrapolated to high-risk groups. Our study provides new insights into the complex interplay among cortisol, GH, and IGF-1 in relation to COVID-19 severity. Compared with previous studies, our current research has identified hormonal response profiles across three stages of COVID-19 severity: outpatients, inpatients, and ICU patients. Our study found markedly elevated cortisol levels in inpatients and a noticeable inverse correlation between cortisol and IGF-1 in ICU patients, underscoring the potential role of cortisol in exacerbating disease severity and impairing IGF-1, which is important for immune function and recovery. The study not only confirms but also extends the current understanding of differential hormonal profiles associated with various phases of disease progression, adding new insights into hormonal dynamics that may influence treatment strategies and patient management in COVID-19.

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**Ethics approval:** The study protocol was approved by the Institutional Ethics Committee of Arak University of Medical Sciences, Arak, Iran (Ethics Code: IR.ARAKMU.REC.1400.228). Written informed consent was obtained from all participants prior to their inclusion in the study.

**Conflicts of interest**: The authors declare that they have no conflict of interest.

**Authors' contribution:** Farideh jalali mashayekhi: Conceptualization, Supervision, Investigation, Methodology, Writing original draft and review and Editing. Mohammad Hosseinzadeh: Conceptualization, Investigation, Methodology, Sample collection, data collection, Writing original draft and review and Editing. Mohammad milad Shirvandehi: Investigation, Methodology, data collection, writing original draft. Navid Sehat: Sample collection, data collection, writing original draft. Azam Moslemi: Statistical analysis, Data interpretation, writing original draft. Ehsanollah Ghaznavi-

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