

## Evaluation of Prognostic Markers in Neurological Patients with COVID-19

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### ARTICLE INFO

#### Original Article

**Keywords:** Biomarkers, Coagulation markers, COVID-19, Liver markers, Neurological disorders, Renal markers, SpO<sub>2</sub>

**Received:** 19 Jul. 2025

**Received in revised form:** 02 Sep. 2025

**Accepted:** 14 Sep. 2025

**DOI:** 10.61882/JoMMID.14.1.17

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### ABSTRACT

**Introduction:** The interaction between COVID-19 and pre-existing neurological diseases remains poorly understood, highlighting the need to identify key prognostic markers. This study aimed to characterize the clinical and laboratory profiles and identify predictors of peripheral oxygen saturation (SpO<sub>2</sub>) in hospitalized patients with pre-existing neurological disorders and COVID-19. **Methods:** This retrospective cohort study included 128 hospitalized patients with pre-existing neurological disorders and confirmed COVID-19 at Taleghani Educational Hospital in Abadan, Iran, from March 2020 to March 2022. Demographic, clinical, and laboratory data were extracted from the Hospital Information System and patient files. **Results:** On average, patients exhibited elevated levels of key laboratory markers, including renal (creatinine, BUN), hepatic (ALP, direct bilirubin, AST, ALT), coagulation (PT, aPTT, INR), and inflammatory (ESR) markers. The mean peripheral oxygen saturation (SpO<sub>2</sub>) was below the normal range ( $91.71 \pm 7.88\%$ ). Multivariable linear regression revealed that in-hospital mortality ( $B = -5.93$ ;  $P < 0.001$ ), higher respiratory rate ( $B = -0.10$  per breath/min;  $P = 0.03$ ), and intubation ( $B = -5.58$ ;  $P = 0.001$ ) were significantly associated with lower SpO<sub>2</sub> levels. **Conclusion:** In this cohort of neurological patients with COVID-19, dysregulation of renal, hepatic, and coagulation markers was common. Critically, lower SpO<sub>2</sub> levels were observed in patients with adverse outcomes, including mortality, respiratory distress, and the need for mechanical ventilation. These findings underscore the importance of monitoring these specific biomarkers and SpO<sub>2</sub> to improve risk stratification and guide clinical management in this high-risk patient population.

### INTRODUCTION

In December 2019, the novel coronavirus, severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), emerged in Wuhan, China. By March 2020, the World Health Organization (WHO) had declared the resulting disease, coronavirus disease 2019 (COVID-19), a pandemic [1]. The clinical spectrum ranges from mild symptoms to severe multiorgan failure [2] and encompasses a broad array of neurological manifestations, from anosmia and headache to more severe conditions such as encephalitis and stroke. Common symptoms of COVID-19 include fever, dry cough, headache, fatigue, sore throat, and diarrhea, though the severity can vary considerably. Beyond these common presentations, the virus can also lead to systemic

complications, with significant involvement reported in the nervous, digestive, cardiovascular, and renal systems [3].

Neurological disorders are the leading cause of disability-adjusted life years (DALYs) and the second leading cause of death globally [4], underscoring the clinical importance of understanding COVID-19 outcomes in patients with pre-existing neurological conditions. Patients with COVID-19 frequently present with neurological manifestations, especially in severe cases, which may include acute cerebrovascular events, altered consciousness, and skeletal muscle injury. The presence of neurological manifestations in COVID-19 patients is associated with a higher risk of adverse clinical

outcomes and mortality [5, 6]. These observations suggest that SARS-CoV-2 possesses neurotropic potential, enabling it to directly or indirectly affect the nervous system and produce a wide range of neurological complications [7]. Furthermore, COVID-19 has been reported to exacerbate pre-existing neurological conditions, such as Alzheimer's disease, and to trigger new-onset neurological syndromes, including delirium [8, 9].

Beyond its respiratory effects, COVID-19 is characterized by significant systemic inflammation and multiorgan dysregulation, reflected in various laboratory abnormalities. For instance, the disease is frequently associated with lymphopenia and elevated inflammatory markers, such as the erythrocyte sedimentation rate (ESR) [10]. Coagulation pathways are also frequently disrupted, with an elevated prothrombin time (PT) emerging as a key indicator of disease severity in critically ill patients. The activated partial thromboplastin time (aPTT), however, has been reported to be more variable, showing either prolongation or shortening in different clinical contexts [11]. Hepatic injury is also common, with elevated aminotransferases such as aspartate aminotransferase (AST) and alanine aminotransferase (ALT) being frequently observed in COVID-19 patients [12]. Similarly, renal dysfunction has been widely reported, evidenced by elevated serum creatinine and blood urea nitrogen (BUN) levels [13].

Given the significant interaction between COVID-19 and neurological diseases, there is an urgent need to elucidate the clinical and laboratory parameters that characterize this high-risk population. A detailed characterization of such markers may contribute to improved risk stratification and inform therapeutic decisions for these patients. However, the combined clinical and laboratory profile of COVID-19 patients with pre-existing neurological disorders, and the relationship of these markers with disease severity indicators such as hypoxemia, remain insufficiently characterized. Therefore, this study aimed to characterize the clinical and laboratory profiles of hospitalized COVID-19 patients with pre-existing neurological disorders and to identify factors associated with hypoxemia in this population.

## MATERIAL AND METHODS

**Study design and setting.** This retrospective cohort study included patients with a pre-existing diagnosis of a neurological disease who were subsequently diagnosed with COVID-19 and hospitalized at Taleghani Educational Hospital in Abadan, southwestern Iran, between March 2020 and March 2022. Eligible neurological conditions included, but were not limited to, cerebrovascular accident (CVA), transient ischemic attack (TIA), Alzheimer's disease, epilepsy, brain tumor, migraine, meningitis, hydrocephalus, motor deficits (*e.g.*, hemiplegia), neuropathy, and encephalitis. All demographic, clinical, and laboratory data were

retrospectively extracted from electronic patient files and the Hospital Information System (HIS).

**Inclusion and exclusion criteria.** Patients were included if they met all of the following criteria: (1) a confirmed diagnosis of a neurological disease by a neurologist; (2) hospitalization at Taleghani Educational Hospital between March 2020 and March 2022; and (3) a confirmed diagnosis of COVID-19, either at the time of admission or during hospitalization. Confirmation of COVID-19 was based on at least one of the following: (a) a positive nasopharyngeal swab for SARS-CoV-2 via reverse transcription polymerase chain reaction (RT-PCR); or (b) chest computed tomography (CT) findings consistent with viral pneumonia and indicative of COVID-19, as interpreted by a radiologist. Patients were excluded if they had missing data for any of the primary demographic (age, sex), clinical (SpO<sub>2</sub>, respiratory rate), or laboratory variables (renal, hepatic, and coagulation markers) required for the primary analysis.

**Statistical analysis.** Descriptive statistics were used to summarize the data. Continuous variables are presented as mean  $\pm$  standard deviation (SD) for normally distributed data, while categorical variables are presented as counts (n) and percentages (%). To identify predictors of peripheral oxygen saturation (SpO<sub>2</sub>), a multivariable linear regression model was constructed with SpO<sub>2</sub> as the continuous dependent variable. As the exclusion criteria ensured complete data for all variables, the regression model included all 128 patients. All statistical analyses were performed using SPSS Statistics for Windows, Version 25.0 (IBM Corp., Armonk, NY, USA). A two-sided *P*-value of less than 0.05 was considered statistically significant for all analyses.

## RESULTS

**Participant demographics.** The final study cohort comprised 128 patients with a confirmed co-diagnosis of a neurological disorder and COVID-19. The mean age of the participants was  $61.87 \pm 19.77$  years, and the majority were male ( $n = 82$ ; 64.1%) (Table 1).

**Clinical and laboratory findings.** The primary clinical comorbidities and laboratory findings for the cohort are summarized in Table 2. The most prevalent comorbidities were cardiovascular disease ( $n = 67$ ; 52.3%) and diabetes ( $n = 46$ ; 35.9%). Notably, the mean values for several key laboratory markers were above their normal reference ranges, suggesting possible organ dysfunction and inflammation. Specifically, markers of renal function (creatinine:  $1.65 \pm 1.56$  mg/dL; BUN:  $33.08 \pm 24.57$  mg/dL), hepatic injury (ALP:  $259.44 \pm 124.47$  IU/L; direct bilirubin:  $0.37 \pm 0.33$  mg/dL; AST:  $54.13 \pm 39.59$  IU/L; ALT:  $46.64 \pm 66.50$  IU/L), and coagulation (PT:  $14.71 \pm 4.52$  s; aPTT:  $39.71 \pm 12.66$  s; INR:  $1.34 \pm 0.28$ ) were, on average, elevated above their normal reference ranges. Furthermore, the mean erythrocyte sedimentation rate (ESR) was high at  $47.11 \pm 35.80$  mm/h. Clinically, patients exhibited tachypnea, with an average respiratory

rate of  $21.67 \pm 3.82$  breaths/min, and hypoxemia, with a mean peripheral oxygen saturation of  $91.71 \pm 7.88\%$ .

**Table 1.** Demographic characteristics of the study cohort (N = 128)

Variable	Category	Count (n)	Percent (%)
Age, years	≤ 50	29	22.7
	51-70	47	36.7
	≥ 71	52	40.6
Sex	Male	82	64.1
	Female	46	35.9
Marital Status	Married	109	85.2
	Single	19	14.8
Total		128	100

**Table 2.** Baseline clinical and laboratory characteristics of the study cohort (N = 128)

Variable	Value	Reference range
<b>Comorbidities, n (%)</b>		
History of cardiovascular disease	67 (52.3)	—
History of diabetes	46 (35.9)	—
History of respiratory disease	23 (18.0)	—
History of renal disease	21 (16.4)	—
History of seizure	15 (11.7)	—
<b>Laboratory Findings, Mean ± SD</b>		
BUN, mg/dL	33.08 ± 24.57	8–23
Creatinine, mg/dL	1.65 ± 1.56	0.6–1.2
Na, mEq/L	138.65 ± 13.20	136–142
K, mEq/L	5.18 ± 0.96	3.5–5.0
ALP, IU/L	259.44 ± 124.47	30–120
Total bilirubin, mg/dL	1.05 ± 1.63	0.3–1.2
Direct bilirubin, mg/dL	0.37 ± 0.33	0.1–0.3
AST, IU/L	54.13 ± 39.59	10–30
ALT, IU/L	46.64 ± 66.50	10–40
PT, s	14.71 ± 4.52	9–13
aPTT, s	39.71 ± 12.66	25–35
INR	1.34 ± 0.28	0.8–1.2
ESR, mm/h	47.11 ± 35.80	M: ≤ 15; F: ≤ 20
WBC, $10^3/\mu\text{L}$	10.34 ± 6.29	4.5–11.0
RBC, $10^6/\mu\text{L}$	4.42 ± 0.82	M: 4.5–5.9; F: 4.1–5.1
MCV, fL	83.85 ± 10.32	80–100
MCH, pg	27.53 ± 3.32	26–34
PLT, $10^3/\mu\text{L}$	218.10 ± 105.39	150–450
<b>Clinical Measurements, Mean ± SD</b>		
RR, breaths/min	21.67 ± 3.82	12–20
SpO <sub>2</sub> , %	91.71 ± 7.88	≥ 95
SBP, mm Hg	118.48 ± 27.11	90–120

**Abbreviations:** SD, standard deviation; M, male; F, female; BUN, blood urea nitrogen; Na, sodium; K, potassium; ALP, alkaline phosphatase; AST, aspartate aminotransferase; ALT, alanine aminotransferase; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio; ESR, erythrocyte sedimentation rate; WBC, white blood cell count; RBC, red blood cell count; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; PLT, platelet count; RR, respiratory rate; SpO<sub>2</sub>, peripheral oxygen saturation; SBP, systolic blood pressure

**Factors associated with SpO<sub>2</sub> levels in multivariable analysis.** The results of the multivariable linear regression analysis are presented in Table 3. After adjusting for all variables in the model, three factors remained significantly associated with lower SpO<sub>2</sub> levels: in-hospital mortality ( $B = -5.93$ , 95% CI  $[-8.96, -2.90]$ ;  $P <$

$0.001$ ), respiratory rate ( $B = -0.10$  per breath/min, 95% CI  $[-0.19, -0.01]$ ;  $P = 0.03$ ), and the need for mechanical ventilation (intubation) ( $B = -5.58$ , 95% CI  $[-8.97, -2.20]$ ;  $P = 0.001$ ). No other demographic, clinical, or laboratory variables showed a statistically significant independent association with SpO<sub>2</sub> levels in this model.

**Table 3.** Multivariable linear regression analysis of factors associated with SpO<sub>2</sub> (N = 128).

Predictor variable	Unstandardized coefficient (B)	Std. error (SE)	95% confidence interval (CI)	Standardized coefficient ( $\beta$ )	t-statistic	P-value
<b>Demographics</b>						
Age, per year	-0.046	0.040	[-0.125, 0.034]	-0.106	-1.138	0.257
Sex (Male vs. Female)	1.990	1.532	[-1.045, 5.024]	0.120	1.299	0.197
Marital status (Single vs. Married)	-1.473	2.126	[-5.685, 2.738]	-0.064	-0.693	0.490
<b>Clinical Outcomes &amp; Comorbidities</b>						
In-hospital mortality (Death vs. Survival)	-5.928	1.528	[-8.956, -2.900]	-0.350	-3.881	< 0.001
History of diabetes (Yes vs. No)	-1.902	1.501	[-4.875, 1.071]	-0.117	-1.267	0.208
History of renal disease (Yes vs. No)	-2.480	2.016	[-6.472, 1.513]	-0.114	-1.230	0.221
History of cardiovascular disease (Yes vs. No)	1.035	1.470	[-1.877, 3.946]	0.065	0.704	0.483
History of respiratory disease (Yes vs. No)	-1.769	1.866	[-5.466, 1.927]	-0.088	-0.948	0.345
History of seizure (Yes vs. No)	-0.157	2.255	[-4.624, 4.311]	-0.006	-0.069	0.945
<b>Laboratory Parameters (per unit increase)</b>						
BUN, mg/dL	-0.038	0.029	[-0.095, 0.019]	-0.127	-1.328	0.187
Creatinine, mg/dL	0.001	0.019	[-0.038, 0.038]	0.003	0.035	0.972
Sodium, mEq/L	-0.013	0.016	[-0.045, 0.018]	-0.079	-0.841	0.402
Potassium, mEq/L	0.091	0.101	[-0.110, 0.291]	0.086	0.897	0.372
Alkaline phosphatase, IU/L	-0.002	0.016	[-0.034, 0.029]	-0.017	-0.153	0.878
Total bilirubin, mg/dL	0.006	0.022	[-0.036, 0.049]	0.035	0.299	0.766
Direct bilirubin, mg/dL	0.003	0.004	[-0.006, 0.012]	0.081	0.686	0.495
AST, IU/L	-0.008	0.006	[-0.020, 0.003]	-0.148	-1.407	0.163
ALT, IU/L	-0.005	0.010	[-0.025, 0.015]	-0.050	-0.476	0.635
Prothrombin time, s	0.021	0.056	[-0.091, 0.133]	0.036	0.372	0.711
aPTT, s	-0.008	0.016	[-0.041, 0.024]	-0.050	-0.507	0.613
INR	-0.016	0.017	[-0.049, 0.017]	-0.093	-0.938	0.351
ESR, mm/h	0.010	0.008	[-0.005, 0.025]	0.160	1.268	0.209
WBC count, 10 <sup>3</sup> /μL	0.114	2.161	[-4.167, 4.395]	0.005	0.053	0.958
RBC count, 10 <sup>6</sup> /μL	-0.016	0.010	[-0.035, 0.003]	-0.155	-1.664	0.099
MCV, fL	0.045	0.127	[-0.207, 0.297]	0.033	0.351	0.726
MCH, pg	0.051	0.040	[-0.029, 0.131]	0.118	1.261	0.210
Platelet count, 10 <sup>3</sup> /μL	0.001	0.001	[-0.001, 0.004]	0.101	1.046	0.298
<b>Clinical Variables &amp; Symptoms</b>						
Respiratory rate, per breath/min	-0.101	0.046	[-0.191, -0.010]	-0.208	-2.196	0.030
Systolic blood pressure, mm Hg	-0.007	0.034	[-0.075, 0.061]	-0.020	-0.195	0.846
Fever (Yes vs. No)	1.015	1.560	[-2.076, 4.106]	0.061	0.651	0.517
Cough (Yes vs. No)	-2.073	1.460	[-4.966, 0.820]	-0.131	-1.419	0.158
Shortness of breath (Yes vs. No)	-2.228	1.456	[-5.112, 0.656]	-0.141	-1.531	0.129
Intubation (Yes vs. No)	-5.582	1.708	[-8.966, -2.198]	-0.291	-3.268	0.001
ICU care (Yes vs. No)	-2.218	1.539	[-5.266, 0.831]	-0.133	-1.441	0.152
ICU duration, per day	-0.134	0.146	[-0.423, 0.154]	-0.086	-0.918	0.361

**Note:** The dependent variable is peripheral oxygen saturation (SpO<sub>2</sub>), measured in percent (%).

**Abbreviations:** BUN, blood urea nitrogen; AST, aspartate aminotransferase; ALT, alanine aminotransferase; PT, prothrombin time; aPTT, activated partial thromboplastin time; INR, international normalized ratio; ESR, erythrocyte sedimentation rate; WBC, white blood cell; RBC, red blood cell; MCV, mean corpuscular volume; MCH, mean corpuscular hemoglobin; ICU, intensive care unit; IU/L, international units per liter.

## DISCUSSION

This study sought to characterize the clinical and laboratory profiles and identify factors associated with hypoxemia in a cohort of hospitalized patients with comorbid neurological disorders and confirmed COVID-19. Using SpO<sub>2</sub> as a primary indicator of respiratory function, our multivariable analysis revealed several critical associations. The central finding of this study is that lower SpO<sub>2</sub> levels were significantly observed in patients with adverse clinical outcomes. Specifically, patients who experienced in-hospital mortality, had higher respiratory rates, significantly lower SpO<sub>2</sub> levels,

and required mechanical ventilation (intubation). While these associations do not establish a causal or predictive direction, they highlight the clinical relevance of SpO<sub>2</sub> monitoring in this population.

This strong association between hypoxemia and adverse outcomes is consistent with the systemic impact of hypoxia, particularly given the brain's profound vulnerability to oxygen deprivation. The brain's high metabolic rate requires a constant supply of oxygen, and when this supply is compromised, cellular hypoxia ensues. This initially leads to reduced synaptic activity, but prolonged or severe hypoxia can cause irreversible

neuronal cell loss and death [14]. Although our study measured peripheral SpO<sub>2</sub> rather than cerebral oxygenation directly, peripheral hypoxemia may serve as a surrogate indicator of compromised oxygen delivery to the brain. Our findings parallel recent research by Adingupu *et al.* (2023), who found that 24% of individuals in the post-COVID-19 phase exhibited decreased cerebral tissue oxygen saturation [15]. Although that study examined post-acute sequelae rather than acute illness, both findings highlight the vulnerability of the nervous system to COVID-19-related hypoxia. Notably, this cerebral hypoxia was directly associated with diminished neurological function and a lower quality of life [15].

Our observation of an elevated mean ESR, a key inflammatory marker, highlights the pronounced systemic inflammatory response in this patient population. This result corroborates previous findings that ESR levels are frequently elevated in hospitalized patients with confirmed COVID-19 who develop acute neurological symptoms [16]. Beyond inflammation, our study also revealed that the mean levels of markers for renal dysfunction (creatinine, BUN) and coagulopathy (PT, aPTT) were elevated. This pattern of multiorgan dysregulation is consistent with the findings of Alshehri *et al.* (2023), who reported that elevated creatinine, blood urea nitrogen, and aPTT, among other markers, were significantly associated with mortality in COVID-19 patients with neurological manifestations [17]. Taken together, these findings suggest that the derangements in renal and coagulation pathways observed in our cohort are not merely incidental but may be key contributors to disease severity and poor outcomes in neurological patients with COVID-19.

Our finding of elevated liver enzymes is consistent with a growing body of evidence indicating that hepatic dysfunction is a common feature of COVID-19. Previous meta-analyses have confirmed that high levels of liver enzymes, including ALT, AST, and ALP, are frequently reported, typically indicating mild liver injury [18]. Crucially, such elevations are not benign; elevated total bilirubin, AST, and ALT have been identified as significant predictors of in-hospital mortality in patients with COVID-19 pneumonia [19]. This connection is potentially relevant for our cohort, as liver dysfunction can precipitate or exacerbate neurological manifestations through mechanisms such as hyperammonemia [20], although ammonia levels were not measured in the present study. Overall, the elevated hepatic markers observed in our study may signal concomitant liver injury, but their specific contribution to neurological outcomes was not demonstrated in our analysis and warrants further investigation.

In addition to organ-specific dysfunction, our study's finding of elevated coagulation markers aligns with the well-established link between severe COVID-19 and a prothrombotic state, or coagulopathy. Indeed, previous

research by Qiu *et al.* (2021) demonstrated that coagulation parameters, including PT, INR, and aPTT, were significantly higher in patients with severe COVID-19 [21]. This hypercoagulable state is of profound neurological relevance, as coagulopathy has been specifically linked to an increased risk of acute cerebrovascular events such as stroke [22]. Therefore, the elevated coagulation markers observed in our cohort may reflect a clinically significant systemic coagulopathy that warrants further investigation in neurological patients.

This study has several limitations that should be acknowledged. First, the relatively small sample size (n = 128), derived from a single center, may limit the statistical power to detect more subtle associations and constrain the generalizability of our findings. The single-center design may also introduce selection bias, as the patient population may not be representative of other geographic regions or healthcare settings. Second, due to the retrospective design, we were unable to control all potential confounding variables, such as specific medication regimens (*e.g.*, corticosteroids, anticoagulants) or the precise severity of the underlying neurological disease. These unmeasured factors could influence both laboratory values and clinical outcomes. Third, the inclusion of patients diagnosed with COVID-19 based on CT findings alone, without RT-PCR confirmation, introduces a potential for misclassification bias, although this approach was a common clinical practice during the peak of the pandemic.

In conclusion, this study of hospitalized neurological patients with COVID-19 reveals a clinical profile characterized by a high burden of cardiovascular comorbidities and significant multisystemic dysregulation. Our findings demonstrate a clear pattern of renal, hepatic, and coagulation pathway derangements, accompanied by a pronounced inflammatory state. Critically, we found that lower SpO<sub>2</sub> levels were strongly associated with in-hospital mortality and the need for mechanical ventilation, suggesting that hypoxemia is a key marker of disease severity in this population. These results underscore the necessity for clinicians to vigilantly monitor these laboratory parameters and, most importantly, peripheral oxygen saturation, to facilitate early risk stratification and guide timely interventions in this vulnerable patient population.

Building upon our findings, future research should employ prospective, multicenter, and longitudinal designs. Such studies would be ideally positioned to compare clinical trajectories and laboratory parameters across distinct cohorts, including hospitalized patients with neurological disease alone, COVID-19 alone, both conditions, and matched controls without either condition. Furthermore, the application of advanced statistical models, such as propensity score matching or mixed-effects models, would be crucial. These methods could more robustly disentangle the independent and synergistic

effects of each condition on disease severity and treatment outcomes.

### CONFLICTS OF INTEREST

The authors declare that there are no conflicts of interest associated with this manuscript.

### ACKNOWLEDGMENT

The authors wish to thank the Clinical Research Development Unit at Taleghani Educational Hospital, Abadan University of Medical Sciences, for facilitating data access.

### DATA AVAILABILITY

The dataset generated and analyzed for this study is not publicly available to protect patient privacy and confidentiality. However, de-identified data can be made available from the corresponding author upon reasonable request.

### AUTHORS' CONTRIBUTIONS

TEF: Investigation, Methodology, Data Curation, Writing–Original Draft. NK: Investigation, Methodology, Formal Analysis, Writing–Original Draft. MMSH: Investigation, Methodology, Data Curation. FKk: Data Curation. FShD: Data Curation. KhK: Investigation, Methodology, Data Curation. AZ: Investigation, Methodology. SM: Investigation, Methodology. ER: Conceptualization, Project Administration, Investigation, Methodology, Data Curation, Writing–Original Draft.

### FUNDING

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

### AI DISCLOSURE

No artificial intelligence (AI) or AI-assisted technologies were utilized in the writing or editing of this manuscript.

### ETHICS STATEMENT

The study protocol was reviewed and approved by the Institutional Review Board (IRB) / Ethics Committee of Abadan University of Medical Sciences (Approval code: IR.ABADANUMS.REC.1399.186). The research was conducted in accordance with the principles of the Declaration of Helsinki. Due to the retrospective nature of the study and the use of fully anonymized data, the Ethics Committee granted a waiver for informed consent. Patient confidentiality was rigorously maintained throughout the study by de-identifying all personal information prior to analysis.

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**Cite this article:**

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Foumani TE, Kamyari N, Shadravan MM, Khishvand F, Shahroud Dezful F, Kanani K, Zahedi A, Mobarak S, Radmanesh E. Evaluation of Prognostic Markers in Neurological Patients with COVID-19. *J Med Microbiol Infect Dis,* 2026; 14 (1): 17-23. DOI: 10.61882/JoMMID.14.1.17.