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Exploring the Link Between SARS-CoV-2 RT-PCR Cycle Threshold, Clinical Characteristics, and Laboratory Results in Hospitalized COVID-19 Patients With Neurological and Autoimmune Conditions

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Abstract

Background: Coronavirus disease-2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), is having a significant impact on global health and the global economy. Notably, the virus can persist in various organs postinfection, potentially triggering chronic inflammation and long-term health issues, including neurological and autoimmune disorders. This study examines the association between SARS-CoV-2 viral load (measured by reverse transcription polymerase chain reaction [RT-PCR] cycle threshold [Ct] values) and clinical/laboratory parameters in hospitalized COVID-19 patients with preexisting neurological or autoimmune disorders.

Methods: This cross-sectional study included 86 COVID-19 patients hospitalized in Kowsar and Amir-Al-Momenin hospitals, affiliated with Semnan University of Medical Sciences, from September 2022 to March 2023. Nasopharyngeal and oropharyngeal swab samples were collected and analyzed using RT-PCR. Clinical data, including demographic information, clinical manifestations, and laboratory parameters, were statistically analyzed using SPSS (version 26). Variables with $P \leq 0.05$ were considered significant.

Results: This cohort study comprised 38 men (44.2%) and 48 women (55.8%), with a median age of 61 years. Neurological diseases were present in 12 patients (14%), and autoimmune diseases were observed in 6 patients (7%). The mean age of patients with neurological diseases was significantly higher than that of patients without neurological diseases (74.75 vs 66.24 years, $P = 0.045$). At the same time, no significant differences were found in clinical parameters, including fasting blood glucose (FBG), white blood cell (WBC) count, and liver function tests (AST, ALT), between patients with and without neurological or autoimmune diseases. Survival analysis indicated a marginally significant association between neurological diseases and survival ($P = 0.050$), whereas no significant association was observed for autoimmune diseases.

Conclusion: This study did not find significant associations between SARS-CoV-2 RT-PCR Ct values and the presence of neurological or autoimmune diseases in COVID-19 patients. While Ct values reflect viral load, other factors, such as pre-existing conditions, genetic predisposition, and immune responses, may play a more critical role in the development of these conditions. Further prospective studies are necessary to elucidate these complex relationships and improve patient treatment strategies.

Keywords: COVID-19; SARS-CoV-2, Cycle threshold, Neurological diseases, Autoimmune diseases, RT-PCR, Viral load

Conflicts of Interest: *None declared*
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↑What is "already known" in this topic:
COVID-19 can persist in various organs postinfection, potentially triggering chronic inflammation and long-term health issues, including neurological and autoimmune disorders.

→What this article adds:
Our research did not find significant associations between SARS-CoV-2 RT-PCR Ct values and the presence of neurological or autoimmune diseases in COVID-19 patients.

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Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) is the infectious agent that causes coronavirus disease (COVID-19). The COVID-19 pandemic has complicated the daily lives of people around the world and placed a heavy burden on families. Many people have died, and the number is increasing every day (1). The economy and healthcare have been severely affected by this pandemic (2, 3).

A meta-analysis study shows that chronic hypertension, obstructive pulmonary disease (COPD), diabetes, cardiovascular disease, and cerebrovascular disease are notable risk factors for patients diagnosed with COVID-19 (4). SARS-CoV-2 particles persist in various organs even after symptoms have disappeared or a strong immune response has developed following acute COVID-19 infection. It is essential to understand that the presence of an undetectable virus does not necessarily indicate eradication. Persistence of viral particles continues to trigger the immune system, leading to chronic inflammation and local tissue damage (5).

Some studies show that SARS-CoV-2 RNA was detected throughout the body, including brain tissue, up to 230 days after symptom onset, highlighting the possibility of long-term viral persistence and disease progression (6). According to clinical and animal research, long-term stress increases oxidative stress and proinflammatory cytokine production, which are the root causes of cognitive and psychological issues (7, 8). COVID-19 can cause cytokine dysregulation, or a cytokine storm [9]. Inflammatory responses triggered by the virus are believed to play a role in contributing to neurodegeneration (8). Also, patients hospitalized with COVID-19 face myriad psychological stressors that make them vulnerable to post-traumatic stress disorder (PTSD). Social isolation measures, restrictive visitation policies, and limited contact with relatives increase feelings of loneliness and emotional stress. In addition, the physical discomfort, invasive medical procedures, and fear of death associated with the symptoms of COVID-19 further exacerbate the traumatic hospitalization experience. New evidence suggests a notable association between COVID-19 hospitalization and PTSD symptoms, with studies reporting increased levels of PTSD-related symptoms such as intrusive thoughts, avoidance behaviors, and hypervigilance in hospitalized patients (10, 11). The hypothalamus is critical in initiating hormonal responses to PTSD via the hypothalamic–pituitary–adrenal axis. Furthermore, the hypothalamus can affect almost all parts of the body through regulating endocrine and autonomic systems as well as somatic behaviors (12).

Additionally, patients infected with SARS-CoV-2 were 25% more likely to get an autoimmune inflammatory rheumatic disease later on (13). The associated inflammatory response in some COVID-19 patients leads to increased amounts of pro-inflammatory cytokines and is being studied as a prognostic factor. Therefore, the immunogenic changes observed in patients with Parkinson's disease and Alzheimer's disease also include their pathogenic role (14).

This study aims to investigate the association among SARS-CoV-2 RT-PCR Ct values, clinical manifestations, and laboratory findings in hospitalized COVID-19 patients with preexisting neurological or autoimmune disorders. This addresses viral load dynamics during active infection in vulnerable populations, rather than post-infection autoimmune risk.

Methods

Study Design and Setting

A hospital-based cross-sectional study was conducted at Kausar and Amir-Al-Momenin hospitals (Semnan University of Medical Sciences, Iran) from September 2022 to March 2023. The study received ethical approval (Certificate No: IR.SEMUMS.REC.1402.055).

Participants

Inclusion Criteria

1. Hospitalized adults (≥18 years) with laboratory-confirmed SARS-CoV-2 infection (RT-PCR-positive nasopharyngeal/oropharyngeal swab)
2. Preexisting neurological/autoimmune disorders diagnosed by specialists before COVID-19 admission
3. Symptomatic COVID-19 requiring hospitalization

Table 1. Variables and Data Collection

Variable Category	Specific Measures	Operational Definition
Primary Exposure	Ct value	Mean Ct of SARS-CoV-2 E/N/RdRp genes
Clinical Outcomes	Disease severity	WHO Clinical Progression Scale (1=mild; 8=death)
	SPO_2 (%)	Initial oxygen saturation on admission
	Mortality	In-hospital death
Laboratory Parameters	CRP	C-reactive protein (mg/L)
	WBC	White blood cells ($\times 10^3/\mu L$)
	AST/ALT	Liver enzymes (U/L)
	Creatinine	Renal function (mg/dL)
Confounders	Age	Continuous (years)
	Vaccination status	Unvaccinated/Partially/Fully
	Immunosuppressants	Current use (yes/no)
	Comorbidities	Hypertension, diabetes, etc.

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Exclusion Criteria

1. Asymptomatic COVID-19 cases
2. Incomplete medical records (>20% data missing)

Laboratory Methods

1. Sample Collection: Trained staff collected nasopharyngeal/oropharyngeal swabs using synthetic fiber swabs (Copan, Italy), stored in viral transport medium at 4°C for ≤48h.

2. RNA Extraction: Viral RNA extracted with BehPrep Viral RNA Extraction Kit (Cat. No: PR004, Behsam, Iran) following the manufacturer's protocol.

3. RT-PCR: Performed using COVID-19 One-Step RT-PCR Kit (COVITECH, Iran) on QIAquant 5-Plex (Qiagen). Cycling conditions: 50°C/20 min; 95°C/3 min; 45 cycles of 95°C/15 sec → 60°C/30 sec.

4. Quality Control: Internal control (human RNase P) confirmed RNA integrity. Ct values represented as mean of triplicate runs; samples with Ct of >40 were considered negative.

Statistical Analysis

The mean and standard deviation for normally distributed continuous variables and the median with interquartile range for non-normally distributed continuous variables were used to summarize the data. For these variables (Table 1), group comparisons used Mann-Whitney U tests for nonparametric distributions and independent t tests for parametric data. Frequencies with percentages were used to represent categorical variables, and Fisher's exact tests or Pearson's chi-square tests were used for analysis as necessary.

Viral load was categorically stratified as "high" (Ct < 25) versus "low" (Ct ≥ 25) based on the World Health Organization guidelines for SARS-CoV-2 viral load quantification. To assess independent predictors of viral load, multivariable linear regression analysis was performed with Ct value as the primary outcome variable. This model adjusted for clinically relevant covariates, including age, biological sex, and COVID-19 vaccination status. Statistical significance was defined a priori as a two-tailed P < .05. All analyses were conducted using SPSS Statistics version 26 (IBM Corp).

Bias Mitigation

Three primary strategies were implemented to address potential biases. Measurement bias was minimized through standardized RT-PCR protocols with internal quality controls (human RNase P) and triplicate testing of each sample. Selection bias was mitigated by enrolling all eligible hospitalized COVID-19 patients who met the inclusion criteria consecutively during the study period. Confounding bias was addressed through multivariable regression modeling that incorporated key demographic and clinical covariates identified a priori in the analytical plan.

Results

A total of 86 COVID-19 patients hospitalized at Kowsar and Amir-Al-Momenin in Semnan participated in this study. Of these, 38 were male (44.2%), and 48 were female (55.8%), yielding a male-to-female ratio of 0.79. The patient age range was 18 to 70 years. Based on the results, 5.8% of patients are single, and 91.9% are married. Also, the Mortality rate was 9.3% (Table 2).

Table 2. Patient Demographics With a COVID-19 Diagnosis

Characteristic	Category	Frequency	Percent
Sex	Male	38	44.2
	Female	48	55.8
Age	<20	2	2.3
	21-30	4	4.7
	31-40	2	2.3
	41-50	4	4.7
	51-60	12	14.0
	>61	62	72.1
Status	Alive	78	90.7
	Dead	8	9.3

The results of the clinical manifestations study revealed that the most common neurological symptom was headache. Additionally, among the 86 individuals diagnosed with the novel coronavirus, there were 12 cases of neurological disease (14%: 4 Parkinson's, 8 Alzheimer's), 6 cases of autoimmune disease (7%: 4 rheumatoid arthritis [RA], 1 multiple sclerosis [MS], and 1 autoimmune nephropathy).

Among COVID-19-positive patients, those with neurological disease (n = 12) had a mean age of 74.75 years (SD = 11.49), while those without neurological disease (n = 74) had a mean age of 66.24 years (SD = 19.19). Under the equal variances assumption—the more appropriate approach given the result of Levene's test—the independent samples t test revealed that the age difference (a mean difference of 8.51 years) was not statistically significant (P = 0.140). Notably, if the assumption of unequal variances is applied, the t-test yields a significant result (P = 0.045); however, this result is less appropriate given that Levene's test supports homogeneity of variances (Table 3).

Furthermore, among COVID-19-positive patients, those with autoimmune diseases (n = 6) had a mean age of 61.33 years (SD = 16.67), compared with a mean age of 67.89 years (SD = 18.64) for patients without autoimmune diseases (n = 80). The independent-samples t test, assuming equal variances, indicated no statistically significant difference in mean age (P = 0.406). A similar nonsignificant result was observed when assuming unequal variances (P = 0.393) (Table 4).

In summary, COVID-19-positive patients with neurological diseases tend to be, on average, 8.51 years older than those without neurological diseases. However, this difference does not reach statistical significance under the equal-variance assumption. On the other hand, there were no significant differences in age between COVID-19-positive patients with and without autoimmune disorders.

SARS-CoV-2 Viral Load Distribution in Hospitalized COVID-19 Patients With Preexisting Neurological and Autoimmune Disorders

Analysis of SARS-CoV-2 cycle threshold (Ct) values

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Table 3. Comparison of Mean Age in COVID-19 Positive Patients With and Without Neurological Diseases

Group	N	Mean Age	Standard Deviation
Neurological Disease	12	74.75	11.490
No Neurological Disease	74	66.24	19.189

Table 4. Comparison of Mean Age in COVID-19 Positive Patients With and Without Autoimmune Diseases

Group	N	Mean Age	Standard Deviation
Autoimmune Disease	6	61.33	16.669
No Autoimmune Disease	80	67.89	18.640

Table 5. Stratified Analysis of SARS-CoV-2 Viral Load by Comorbidity Status in Hospitalized COVID-19 Patients With Neurological and Autoimmune Disorders

Group	High Viral Load (Ct<25)	Low Viral Load (Ct≥25)	*P*-value
Neurological Disorder	9 (75.0%)	3 (25.0%)	0.233
No Neurological Disorder	42 (56.8%)	32 (43.2%)	(ref)
Autoimmune Disorder	1 (16.7%)	5 (83.3%)	0.106†
No Autoimmune Disorder	50 (62.5%)	30 (37.5%)	(ref)

Chi-square test; †Fisher's exact test.

revealed no statistically significant difference in viral load between COVID-19 patients with pre-existing neurological disorders and those without neurological conditions ($P = 0.233$ by independent t-test/Mann-Whitney U test). This indicates that baseline neurological status did not significantly influence SARS-CoV-2 replication kinetics during acute infection.

However, a clinically notable trend emerged in the autoimmune subgroup. Patients with autoimmune disorders demonstrated lower viral loads (higher Ct values), with 83.3% (5/6) exhibiting Ct values ≥25 (categorized as "Low Viral Load") compared to only 37.5% (30/80) in the non-autoimmune group ($P = 0.106$ by Fisher's exact test). This represents a 2.2-fold higher prevalence of low viral load states among autoimmune patients, suggesting potential modulation of viral replication by immunological factors characteristic of these conditions, though statistical significance was not reached due to limited sample size (Table 5).

Determining the Relationship between SARS-CoV-2 RT-PCR CT, Clinical Characteristics, and Laboratory Data in Neurological Disease Patients

A retrospective analysis was performed using data collected from 86 COVID-19 patients with a known neurological condition. CT values are classified as "serious" based on established thresholds.

The analysis revealed that out of a cohort of 86 COVID-19 patients, 12 had neurological disease and 74 did not. In the neurological disease group, 9 cases were classified as "severe" based on CT values, while 3 cases fell into the "mild" category. In contrast, in the absence of neurological disease, 42 cases were classified as "severe" and 32 as "mild". The chi-square test yielded nonsignificant results, indicating that Ct values were not associated with the development of neurological disorders (Pearson's chi-square: $P = 0.233$).

Our analysis revealed no statistically significant association between Ct values and neurological diseases ($P > 0.05$). Similarly, there was no significant difference in initial SPO_2 levels between patients with and without neurological diseases ($P > 0.05$). Survival rates in COVID-19 positive patients with neurological diseases showed a borderline significant association ($P = 0.05$), with older age observed in patients with neurological diseases compared to those without. However, no significant difference in mean age was found between the two groups. Clinical parameters—including fasting blood sugar (FBS), mean arterial pressure, white blood cell (WBC) count, C-reactive protein (CRP), aspartate aminotransferase (AST), alanine aminotransferase (ALT), blood urea nitrogen (BUN), and creatinine—did not differ significantly between patients with and without neurological disease (Table 6).

Among those with neurological diseases, 9 patients were alive, and 3 patients were deceased. Among those without neurological diseases, 66 patients were alive, and 5 patients were deceased. The Pearson chi-square test revealed a marginally significant association between neurological diseases and survival rates among COVID-19-positive patients ($P = 0.05$).

The association between SARS-CoV-2 RT-PCR CT values, clinical characteristics, and laboratory data in patients with Autoimmune Disorders

The analysis aimed to determine the connection between Ct results and the prevalence of autoimmune conditions, such as autoimmune nephropathy, multiple sclerosis (MS), and rheumatoid arthritis (RA). However, the study showed no statistically significant association between Ct values and autoimmune diseases in the study population. Autoimmune diseases were identified in 6 out of 86 individuals. Of these, 5 were classified as mild, and 1 as severe. However, there was no significant difference in the distribution of Ct values (positive or negative) between patients with mild and severe autoimmune diseases.

Our analysis showed no statistically significant association between Ct values and autoimmune diseases ($P > 0.05$). Similarly, initial SPO_2 levels in individuals with and without autoimmune diseases did not differ significantly ($P > 0.05$). Survival rates in COVID-19 positive patients with autoimmune disorders did not show significant differences compared to those without autoimmune disorders ($P > 0.05$). The mean age also did not differ significantly between the two groups. Clinical parameters, including FBS levels, mean blood pressure, WBC count,

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Table 6. Comparison of Clinical Parameters between Neurological, Autoimmune, and Non-Neurological/Non-Autoimmune Disease Groups (Mean ± SD)

Parameter	Neurological (n=12)	No Neurological (n=74)	*P*-value	Autoimmune (n=6)	No Autoimmune (n=80)	*P*-value
SPO_2 (%)	89.2 ± 5.1	90.5 ± 6.3	0.512	91.0 ± 4.9	90.1 ± 6.2	0.741
Mortality	25.0%	6.8%	0.050	0%	10.0%	0.999
FBS (mg/dL)	185.0 ± 126.10	148.96 ± 65.92	0.135	192.00 ± 49.65	151.68 ± 78.25	0.148
AST (U/L)	40.70 ± 21.96	56.38 ± 134.64	0.027	27.20 ± 20.08	56.21 ± 129.16	0.266
ALT (U/L)	24.83 ± 14.35	60.53 ± 146.95	0.762	21.67 ± 4.72	58.72 ± 142.98	0.266
CRP (mg/L)	82.00 ± 49.82	49.75 ± 40.64	0.027	82.00 ± 49.82	49.75 ± 40.64	0.266
WBC ($\times 10^3/\mu L$)	10.2622 ± 5.39	10.5054 ± 4.68	0.892	10.5898 ± 4.78	10.5898 ± 4.78	0.956
HB (g/dL)	11.4111 ± 1.70	11.5418 ± 2.56	0.850	14.1500 ± 1.06	11.4296 ± 2.43	0.004
BUN (mg/dL)	32.167 ±17.66	24.944 ± 11.76	0.180	22.400 ± 10.62	26.203 ± 13.04	0.936
Creatinine (mg/dL)	1.5750 ± .91	1.3278 ± .95	0.278	1.2000 ± .800	1.3734 ± .95	0.936

Mann-Whitney U test (non-normal distributions)

CRP levels, AST, ALT, BUN, and patients with and without autoimmune diseases, did not significantly differ in creatinine levels (Table 6).

Of the 86 COVID-19 positive patients, 6 had autoimmune diseases, and 77 did not. All 6 patients with autoimmune diseases survived, whereas among those without autoimmune diseases, 69 survived, and 8 did not. The Pearson Chi-Square test indicated no significant association between the presence of autoimmune diseases and survival in COVID-19-positive patients ($\chi^2 = 0.690$, df = 1, $P = 0.406$). There was no significant association between autoimmune diseases and survival in COVID-19-positive patients. The *P*-value of 0.406 indicates insufficient statistical evidence for a relationship between autoimmune diseases and survival outcomes in this population.

However, it is important to consider that the small sample size of patients with autoimmune diseases (n = 6) may have limited the analysis's statistical power. A low sample size increases the likelihood of a type II error, meaning that a true association, if present, may not have been detected. Future studies with larger cohorts may be necessary to assess better the potential relationship between autoimmune diseases and COVID-19 survival outcomes.

Discussion

The emergence of COVID-19 has led to a myriad of problems beyond the acute infection, including mental health and immune system complications during the post-recovery phase. This study explores the multifaceted impacts of the virus on mental well-being, encompassing PTSD, anxiety, depression, and cognitive impairment. Furthermore, it examines the complex interplay between the immune system and mental health, elucidating the immunological mechanisms underlying neuropsychiatric symptoms. The study evaluated a range of clinical factors in individuals with COVID-19 who also had immunological and neurological diseases. The SARS-CoV-2 RT-PCR cycle threshold, clinical characteristics, and laboratory data in patients with neurological and autoimmune diseases did not change significantly, according to the study.

Studies show that those who contracted SARS-CoV-2 were 25% more likely to be diagnosed with an autoimmune inflammatory disease in the future (13). The presence of other risk factors, such as hypertension, cardiovascular disease, and diabetes, is often associated with increased susceptibility to COVID-19 (15, 17). Although respiratory disease is the primary clinical manifestation, neurologic manifestations are also receiving increasing attention (18). On the other hand, some studies have shown a positive correlation between symptoms of depression/anxiety/stress and levels of blood markers of inflammation, including the distribution width of platelets and red blood cells. The study's findings provide insights into mental health and physiological markers, highlighting the potential impact of inflammation on mood disorders (19). The results of our investigations showed that there may be no significant correlation between Ct values in COVID-19 patients and the development of neurological diseases. Although this retrospective study provides insight into the relationship between Ct values and neurological disease in COVID-19 patients, further studies are needed to confirm these findings. To learn more about the impact of viral load on the onset and progression of neurological disorders linked to COVID-19, further research with larger sample sizes and prospective designs is required.

The association between lower PCR Ct values, indicative of higher SARS-CoV-2 viral load, and adverse outcomes in hospitalized COVID-19 patients is a critical area of investigation. Several studies have consistently shown a strong correlation between Ct values and disease severity, mortality, and various adverse clinical outcomes (20-24). In our study, non-significant results indicate that there is no statistically significant relationship between Ct and the development of neurological diseases in COVID-19 patients. This finding is relevant to understanding the potential relationship between SARS-CoV-2 viral load, as indicated by Ct values, and the development of neurological conditions such as epilepsy, Alzheimer's disease, and Parkinson's disease in individuals infected with COVID-19. The lack of significance shows that Ct values alone may not be sufficient to predict or explain the development of neurological disease in COVID-19 patients. Although Ct values provide valuable information on viral load and disease severity, other factors may play a greater role in the pathogenesis of neurologic disease in this population. These factors may include preexisting neurological comorbidities, genetic predisposition, immune response, and neuroinflammatory processes caused by viral infec-

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tions. Also, Findings revealed no statistically significant association between Ct values and autoimmune diseases within the study cohort. Of 86 individuals assessed, autoimmune diseases were identified in 6 cases. Among these, five cases were categorized as serious, and one as severe. Nonetheless, no significant disparity was observed in the distribution of CT values (positive or negative) among patients with severe or mild autoimmune diseases.

COVID-19 primarily affects pulmonary tissues, disrupting gas exchange and inducing systemic hypoxia. Given the constant oxygen requirement for proper neuronal function, neurons are particularly susceptible to changes in oxygen saturation, which can result in neuronal injury, with or without neuroinflammation. Our hypothesis posits that hypoxia is a significant clinical manifestation of severe SARS-CoV-2 infection, directly or indirectly fostering premature neuronal aging, neuroinflammation, and neurodegeneration by modulating the expression of genes crucial to cell survival. Recent studies underscore the intricate interplay among hypoxia, early neuronal aging, COVID-19 infection, and neurodegenerative disorders, offering valuable insights into the molecular processes of neurodegeneration (25, 26). Although SPO_2 levels differed between individuals with and without neurological and autoimmune diseases, the analysis found no statistically significant association. These results suggest that baseline SPO_2 levels may not reliably predict neurological and autoimmune disease in the study population. Further studies with larger sample sizes and consideration of other variables are needed to clarify the relationship between baseline SPO_2 levels and the development of neurological and autoimmune diseases**.**

According to several studies, a low platelet count has been identified as a possible marker to differentiate between severe and moderate COVID-19 infection, and it is associated with increased mortality and more severe COVID-19 disease (27). Up to 85% of severe cases of COVID-19 result in lymphopenia, a serious hematologic condition. Clinical outcome is correlated with lymphopenia severity. Conversely, when COVID-19 worsens, the number of neutrophils in the blood steadily increases, leading to neutrophilia, which is associated with severe respiratory disease and a worse prognosis. Studies show that severe COVID-19 cases have significantly elevated levels of white blood cells and neutrophils compared to mild cases. In addition, as the disease progresses, the number of white blood cells, including neutrophils, increases in severe cases (28-30). Systematic reviews and meta-analyses suggest that the most common laboratory findings in these patients are elevated CRP and ESR (31). Some other Studies have indicated that severe or fatal cases of COVID-19 exhibit several hematological and biochemical abnormalities. These include significantly elevated WBCs, as well as markers of liver and kidney function such as BUN, creatinine, CRP, and lactate dehydrogenase (LDH). These findings underscore the importance of monitoring these parameters to assess disease severity and predict outcomes in COVID-19 patients (32-35).

Comparing clinical parameters in COVID-19 patients with neurological and autoimmune conditions provides valuable insights into potential associations and implications for patient management. One notable limitation of this study is its retrospective design, which could introduce bias and constrain the capacity to establish causal relationships. Furthermore, the relatively small sample size of patients with neurological diseases may have reduced the statistical power necessary to identify significant associations.

Conclusion

The findings of this study suggest that there is no significant association between CT values of SARS-CoV-2 RT-PCR and neurological or autoimmune diseases in COVID-19 patients. Clinical parameters also did not significantly differ between patients with and without these conditions. Additional research with larger sample sizes and prospective study designs is necessary to validate these findings and investigate potential mechanisms underlying the observed associations.

Authors' Contributions

MG and RN contributed to conceptualization, methodology, investigation, and writing the original draft. AG and MT and PP contributed to data curation and formal analysis. ME and HG contributed to supervision, validation, and reviewing and editing the manuscript. All authors have read and approved the final manuscript.

Ethical Considerations

All procedures performed in this work followed the ethical standards of the committee of the Semnan University of Medical Sciences (Ethical Code: IR.SEMUMS.REC.1402.055).

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Conflict of Interests

The authors declare that they have no competing interests.

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