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Original Article

SIADH-associated hyponatremia in hospitalized COVID-19 patients: relationship with inflammatory and stress response markers

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ABSTRACT

Aims: Hyponatremia observed during the course of COVID-19 infection is closely associated with disease prognosis. The objective of this study is to determine the prevalence of syndrome of inappropriate antidiuretic hormone secretion (SIADH) in patients diagnosed with COVID-19 and to analyze the relationship between this condition and inflammatory markers and stress response parameters.

Methods: The study included 50 patients hospitalized with a diagnosis of COVID-19 who were found to have hyponatremia (Na<135 mEq/L). The diagnosis of SIADH was based on the criteria of clinical euvolemia, low plasma osmolarity (<275 mOsm/kg), and high urinary sodium (>40 mEq/L). TSH and cortisol levels were evaluated to rule out secondary causes in the differential diagnosis. Patients were divided into two groups: SIADH-positive (n=37) and non-SIADH (n=13). The Mann-Whitney U test was used for intergroup comparisons, and the Spearman test was used for correlation analyses.

Results: The mean age of the patients was 62.1±12.3 years, and a higher proportion of female patients was observed in the SIADH group (p=0.038). Serum sodium levels (median: 130.0 mEq/L) were significantly lower in the SIADH (+) group compared to the Non-SIADH group (p<0.01). Among inflammatory markers, ferritin levels were found to be significantly higher in the SIADH group (741.0 vs 335.0 ng/ml; p=0.029), and serum cortisol levels were also found to be markedly elevated in this group (7.5 vs 1.6 µg/dl; p=0.017). In the correlation analysis, a significant negative correlation was found between serum sodium levels and cortisol (r=-0.325, p=0.021) and ferritin (r=-0.284, p=0.046).

Conclusion: The primary etiological factor for COVID-19-associated hyponatremia is SIADH. Elevated ferritin and cortisol levels accompanying this condition indicate that systemic inflammation and the stress response are associated with the severity of hyponatremia. Preserved urinary sodium and elevated cortisol levels are key clinical clues supporting the diagnosis of SIADH in the course of COVID-19.

Keywords: COVID-19, SIADH, hyponatremia, ferritin, cortisol

INTRODUCTION

COVID-19 is a viral infection that primarily affects the respiratory system but can also involve multiple organ systems.[1,2] Electrolyte imbalances, along with hematological, inflammatory, and metabolic abnormalities, are commonly observed during the course of the disease.[3] The most common of these electrolyte abnormalities is hyponatremia; it has been reported that hyponatremia develops in approximately 20-35% of patients hospitalized for COVID-19 at the time of admission or during the course of the illness.[4,5]

Hyponatremia has been associated with increased mortality, prolonged hospital stay, and poor clinical prognosis in COVID-19 patients.[6,7] Although the etiology of COVID-19-associated hyponatremia is multifactorial, inappropriate antidiuretic hormone secretion syndrome (SIADH) stands out as a key mechanism alongside gastrointestinal fluid losses, inadequate oral intake, cardiac dysfunction, and renal causes.[8,9]

SIADH is a condition characterized by the inability to suppress antidiuretic hormone secretion despite low serum osmolality, the formation of inappropriately concentrated urine, and ongoing renal sodium excretion.[10,11] The primary mechanism responsible for the development of SIADH in COVID-19 is thought to be pulmonary inflammation and cytokine-mediated neurohormonal activation. It has been reported that proinflammatory cytokines, particularly interleukin-6 (IL-6), can stimulate ADH secretion.[12,13] This mechanism may explain the relationship between increased levels of inflammatory markers, such as ferritin, and hyponatremia. Additionally, it has been demonstrated that the marked increase in ferritin levels in severe COVID-19 infection serves as an indicator of systemic inflammation.[14] In patients with euvolemic hyponatremia, secondary causes such as adrenal insufficiency and hypothyroidism must be ruled out before diagnosing SIADH. Therefore, serum cortisol levels are important not only for assessing adrenal function but also as an indicator of the systemic stress response.[15]

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The aim of this study is to evaluate the relationship between serum sodium levels and inflammatory burden (ferritin) and stress response (cortisol) in patients with COVID-19 pneumonia who present with hyponatremia consistent with SIADH.

METHODS

Ethics

This study was conducted with the approval of the Non-interventional Clinical Researches Ethics Committee at Van Yüzüncü Yıl University (Date: 12.02.2021, Decision No: 2021/02-18). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Design and Patient Selection

This retrospective observational study included a total of 50 patients who were hospitalized for COVID-19 pneumonia at a tertiary care center between January 2021 and December 2021 and in whom hyponatremia (serum sodium <135 mEq/L) was detected during follow-up. SARS-CoV-2 infection was confirmed by real-time reverse transcription polymerase chain reaction (RT-PCR) performed in the microbiology laboratory of our hospital using the Bio-Speedy® COVID-19 RT-qPCR Detection Kit (Bioeksen R&D Technologies, Istanbul, Türkiye) on nasopharyngeal swab specimens. Pneumonia was confirmed by chest computed tomography (CT) demonstrating findings compatible with COVID-19 pneumonia.

Diagnostic Criteria and Classification

The diagnosis of SIADH was established based on the presence of hypotonic hyponatremia (calculated plasma osmolality <275 mOsm/kg), clinically euvolemic status, urinary sodium concentration >40 mEq/L, normal thyroid function tests, and the exclusion of adrenal insufficiency, renal failure, and other secondary causes of hyponatremia. In addition, a urine specific gravity >1.010 was considered a supportive finding for the diagnosis of SIADH.

Patients meeting these criteria were classified into the SIADH (+) group (n=37), while those who did not meet the criteria or whose hyponatremia could be explained by alternative causes were classified into the Non-SIADH group (n=13).

Data Collection

Demographic data, comorbid conditions, and laboratory parameters were retrospectively obtained from the hospital information management system. Laboratory assessments included serum sodium, potassium, creatinine, CRP, ferritin, LDH, TSH, free T4, and morning serum cortisol levels.

Plasma osmolarity was calculated using the following formula:

Plasma osmolarity=2×Na+glucose/18+BUN/2.8

Statistical Analysis

The date were performed using IBM SPSS Statistics version 31.0 (IBM Corp., Armonk, NY, USA). Data distribution was assessed using the Shapiro-Wilk test. The Mann-Whitney U test was used to compare continuous variables, while the Chi-square or Fisher's exact test was used to analyze categorical variables. Continuous variables were expressed as the median (interquartile range, IQR), while categorical variables were expressed as counts and percentages. Relationships between variables were assessed using Spearman's correlation analysis. A p-value of <0.05 was considered statistically significant.

RESULTS

The demographic and baseline clinical characteristics of the 50 patients included in the study are presented in **Table 1**. The median age of the patients was 64.0 years in the SIADH (+) group and 58.0 years in the Non-SIADH group; no statistically significant difference in age was observed between the groups (p=0.166). When gender distribution was examined, the proportion of female patients in the SIADH (+) group (56.7%) was found to be significantly higher than in the Non-SIADH group (23.1%) (p=0.038).

All patients (100%) had radiologically confirmed COVID-19 pneumonia (lung involvement). In the clinical volume assessment, all patients (100%) in the SIADH group were euvolemic, whereas this rate was 69.2% in the Non-SIADH group (p=0.004). No significant difference was observed between the groups in terms of baseline renal function markers (serum creatinine; p=0.194) or the nutritional/inflammatory marker albumin (p=0.312). The mean length of hospital stay was 11.5 days in the SIADH group and 9.0 days in the control group, showing statistical similarity (p=0.214). A total of 5 patients (10%) died during the study period, and mortality rates were found to be similar between the groups (p=0.741) (**Table 1**).

Table 1. Demographic and clinical characteristics of the patients

Variable	SIADH (+) (n=37)	Non-SIADH (n=13)	p-value
Age (years), median (IQR)	64.0 (57-72)	58.0 (49-62)	0.166
Gender (female), n (%)	21 (56.7)	3 (23.1)	0.038
Clinical findings, n (%)			
Lung involvement (pneumonia)	37 (100)	13 (100)	1.000
Volume status (euvolemia)	37 (100)	9 (69.2)	0.004
Baseline laboratory data, median (IQR)			
Serum creatinine (mg/dl)	0.81 (0.6-0.9)	0.92 (0.7-1.2)	0.194
Serum albumin (g/dl)	3.2 (2.9-3.6)	3.4 (3.1-3.8)	0.312
Length of hospital stay (days)	11.5 (8-16)	9.0 (7-14)	0.214
Mortality (exitus), n (%)	4 (10.8)	1 (7.6)	0.741

Data are presented as median (interquartile range, IQR) or number (%). Statistical comparisons were performed using the Mann-Whitney U test, Chi-square test, or Fisher's exact test where appropriate. SIADH: Syndrome of inappropriate antidiuretic hormone secretion, IQR: Interquartile range

The laboratory parameters of the groups included in the study are presented in a comparative format in **Table 2**. While the median serum sodium level in the SIADH (+) group was 130.0 mEq/L (IQR: 127.0-132.0), it was 133.0 mEq/L (IQR: 131.0-134.0), and the difference between the two was found to be highly statistically significant (p<0.001). Similarly, plasma osmolarity levels were also significantly lower in the SIADH group (268.5 vs 276.0 mOsm/kg; p=0.002).

When examining the stress and inflammation parameters, which were the focus of the study, it was found that serum cortisol levels in the SIADH group (median: 7.5 µg/dl) were approximately 4.5 times higher than in the control group (median: 1.6 µg/dl), and this difference was statistically significant (p=0.017). Serum ferritin levels, an inflammatory

marker, were also found to be statistically significantly higher in the SIADH group compared to the Non-SIADH group (741.0 vs 335.0 ng/ml; p=0.029). However, no significant differences were observed between the groups regarding CRP, LDH, TSH, and complete blood count parameters (WBC, lymphocytes) (p>0.05) (Table 2).

Table 2. Comparison of laboratory parameters between groups

Parameter	SIADH (+) (n=37)	Non-SIADH (n=13)	p-value
Serum sodium (mEq/L)	130.0 (127.0-132.0)	133.0 (131.0-134.0)	<0.001
Serum cortisol (µg/dl)	7.5 (4.2-13.6)	1.6 (1.1-4.3)	0.017
Serum ferritin (ng/ml)	741.0 (489.0-1250.0)	335.0 (274.0-511.0)	0.029
Plasma osmolarity (mOsm/kg)	268.5 (262.0-272.5)	276.0 (273.5-281.0)	0.002
C-reactive protein (mg/L)	102.0 (63.0-161.0)	96.0 (47.0-129.0)	0.682
LDH (U/L)	464.0 (318.0-618.0)	419.0 (327.0-642.0)	0.706
WBC ($\times10^3$/µL)	9.0 (7.0-12.3)	7.9 (6.7-9.0)	0.388
Lymphocyte count ($\times10^3$/µL)	630 (460-970)	620 (500-700)	0.674
TSH (mU/L)	0.8 (0.4-1.5)	0.9 (0.6-1.3)	0.535

Continuous variables are expressed as median (IQR). p-values were calculated using the Mann-Whitney U test. SIADH: Syndrome of inappropriate antidiuretic hormone secretion, mOsm: Milliosmole, LDH: Lactate dehydrogenase, WBC: White blood cell, TSH: Thyroid-stimulating hormone

The results of the Spearman correlation analysis conducted to determine the relationship between serum sodium levels and various clinical and laboratory parameters are presented in Table 3. The analysis revealed a moderate, negative, and statistically significant correlation between serum sodium levels and serum cortisol levels (r=-0.325, p=0.021). Similarly, a significant negative relationship was also found between sodium levels and ferritin levels (r=-0.284, p=0.046).

These findings indicate that as the severity of hyponatremia increases (as sodium levels decrease), both the systemic inflammatory burden (ferritin) and the physiological stress response (cortisol) increase. Furthermore, the expected strong positive correlation between sodium levels and plasma osmolarity was confirmed (r=+0.642, p<0.001). On the other hand, no statistically significant relationship was found between sodium levels and age or CRP levels (Table 3).

Table 3. Correlation analysis of serum sodium levels with clinical parameters

Variable Pair	Correlation coefficient (spearman's rho)	p-value
Serum Na & serum cortisol	-0.325	0.021
Serum Na & serum ferritin	-0.284	0.046
Serum Na & plasma osmolarity	+0.642	<0.001
Serum Na & CRP	-0.154	0.285
Serum Na & age	-0.222	0.120

Correlation analyses were performed using Spearman's correlation test. A p-value <0.05 was considered statistically significant. Na: Sodium, CRP: C-reactive protein

DISCUSSION

This study demonstrated that SIADH is the most common underlying mechanism of hyponatremia developing during COVID-19 infection and that markers of inflammation and the stress response are more pronounced in patients with SIADH. In particular, the finding that serum ferritin and cortisol levels were significantly elevated in the SIADH group suggests that COVID-19-associated hyponatremia is not merely a simple electrolyte imbalance but a complex process associated with systemic inflammation and a neuroendocrine stress response. Furthermore, the negative correlation observed between serum sodium levels and ferritin and cortisol supports the notion that as the severity of hyponatremia increases, so do the inflammatory burden and the physiological stress response.

Previous studies have reported that hyponatremia is quite common in COVID-19 and is associated with a poor clinical prognosis. Machiraju et al.[16] demonstrated that hyponatremia is common in COVID-19 patients and may be particularly associated with severe clinical course. Similarly, Tzoulis et al.[17] reported that dysnatremia holds independent prognostic significance regarding morbidity and mortality in COVID-19 patients. Tezcan et al.,[18] on the other hand, demonstrated that electrolyte abnormalities at the time of presentation are associated with a poor prognosis. In our study as well, the significantly low serum sodium levels in the SIADH group support the clinical significance of COVID-19-associated hyponatremia.

Although the development of hyponatremia in COVID-19 infection is multifactorial, SIADH stands out as one of the most significant mechanisms. Lippi et al.[19] reported that electrolyte disturbances are common, particularly in severe COVID-19 cases, and that these may be associated with systemic inflammation. In the case reported by Ayat et al.,[20] it was emphasized that SIADH could be the first and only presentation of a COVID-19 infection. In our study as well, the fact that all patients in the SIADH group were euvolemic and had markedly low plasma osmolarity is consistent with the classic pathophysiology of SIADH.

Ferritin is an acute-phase reactant that has been associated with disease severity and adverse clinical outcomes in patients with COVID-19. Kurian et al.[21] demonstrated that serum ferritin levels are associated with COVID-19 severity and clinical outcomes. In our study as well, the finding that ferritin levels were significantly elevated in the SIADH group and the detection of a negative correlation between serum sodium levels and ferritin suggest that hyponatremia worsens as the inflammatory burden increases. This finding is consistent with previous studies demonstrating that higher ferritin concentrations are associated with greater inflammatory burden and more severe COVID-19.

Given that IL-6 levels were not directly measured in our study, ferritin was evaluated as a surrogate marker of systemic inflammation. Previous studies have demonstrated a significant positive correlation between serum IL-6 and ferritin levels in patients with COVID-19, suggesting that elevated ferritin may indirectly reflect cytokine-mediated inflammatory activity. Therefore, the significantly higher ferritin levels observed in the SIADH group should be interpreted as indirect evidence of enhanced inflammation rather than direct evidence of increased IL-6 activity.[22]

Another notable finding in our study is that serum cortisol levels were significantly elevated in the SIADH group. Excluding adrenal insufficiency is of critical importance in the diagnosis of SIADH. Therefore, the detection of elevated rather than low cortisol levels supports the notion that the

hyponatremia is not due to adrenal insufficiency. Additionally, this finding may be interpreted as an indicator of the systemic stress response developing during COVID-19 infection. Pal and Banerjee reported that COVID-19 may activate the hypothalamic-pituitary-adrenal axis through systemic inflammation, leading to alterations in cortisol secretion and adrenal function.[23] Similarly, Jensterle et al.[24] reported that COVID-19 may be associated with both glucocorticoid insufficiency and excessive HPA-axis activation depending on disease severity.[24] The negative correlation observed in our study between serum sodium and cortisol levels suggests that deeper hyponatremia may be associated with a more intense physiological stress response.

It is noteworthy that while ferritin levels showed significant differences in our study, CRP levels were found to be similar. This suggests that ferritin may be a more sensitive marker than CRP for indicating the inflammatory burden in COVID-19. It is particularly known that ferritin levels are more closely associated with macrophage activation and hypercytokinemia.[25] Therefore, it is possible that the inflammatory mechanisms associated with the development of SIADH are better reflected through ferritin.

In our study, no significant differences were observed between the groups in terms of mortality and length of hospital stay. One of the main reasons for this may be the relatively small number of patients. Additionally, the fact that only hyponatremic patients were included in the study may have limited the clarity of prognostic differences between the groups. However, the presence of more pronounced biochemical abnormalities in the SIADH group suggests that these patients developed a more intense inflammatory and metabolic stress response. Adekanmbi et al.[26] demonstrated that the inflammatory burden is a determining factor in clinical outcomes among patients hospitalized due to COVID-19. Therefore, the independent effect of SIADH on the prognosis of COVID-19 could be more clearly established through prospective studies with larger sample sizes.

Limitations

Our study has some limitations. First, it has a retrospective, single-center design. The relatively small number of patients may have limited its statistical power. Additionally, the failure to measure IL-6 and ADH levels prevented a direct demonstration of the pathophysiology of SIADH. Therefore, the proposed inflammatory mechanism should be interpreted as indirect and based on previous literature. However, the study's strengths include the evaluation of the SIADH diagnosis using systematic criteria, the exclusion of adrenal and thyroid causes, and the detailed analysis of the relationship between inflammation/stress parameters and serum sodium levels.

CONCLUSION

As a result, this study demonstrates that SIADH is one of the primary etiological mechanisms of COVID-19-associated hyponatremia and that the inflammatory burden and stress response are more pronounced in patients who develop SIADH. The finding that ferritin and cortisol levels are inversely correlated with serum sodium suggests that hyponatremia may be a biochemical reflection of the systemic inflammatory process in COVID-19. In COVID-19 patients, particularly in the presence of euvolemic hyponatremia, SIADH should be considered, and ferritin and cortisol levels may be evaluated as helpful biomarkers in clinical assessment.

ETHICAL DECLARATIONS

Ethics Committee Approval

This study was conducted with the approval of the Non-interventional Clinical Researches Ethics Committee at Van Yüzüncü Yıl University (Date: 12.02.2021, Decision No: 2021/02-18).

Informed Consent

As this was a retrospective study, formal written informed consent was not required and was therefore not obtained. This retrospective study used pre-existing anonymized patient data. No additional intervention was performed, and there was no direct patient contact. The study was approved by the Ethics Committee, and the requirement for written informed consent was waived by the ethics committee.

Peer Review Process

This manuscript was subject to external peer review.

Conflict of Interest

The authors declare no conflicts of interest related to this study.

Financial Disclosure

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Author Contributions

Concept: OÖ, MA; Design: OÖ, MA; Control: OÖ, MA; Data Collection and/or Processing: OÖ, MA; Analysis and/or Interpretation: OÖ, MA; Literature Review: OÖ, MA; Article Writing: OÖ, MA; Critical Review: OÖ, MA.

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