

# Evaluation of electrolyte status of sodium, potassium and magnesium, and fasting blood sugar at the initial admission of individuals with COVID-19 without underlying disease in Golestan Hospital, Kermanshah

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## Abstract

We examined electrolyte imbalance and blood sugar levels in patients with COVID-19 who had no underlying disease. This cross-sectional study in a clinical center was performed in Kermanshah, west of Iran. All patients who had a record of magnesium ( $Mg^{2+}$ ), potassium ( $K^+$ ), sodium ( $Na^+$ ), and fasting blood sugar (FBS) tests in their clinical files at the time of admission to the hospital from April 21 to July 12, and didn't have a history of an underlying disease, were included in the study. Patients were divided into outpatient (as less severe COVID-19) and intensive care units (ICU) (as severe COVID-19). For statistical analysis of collected data, the SPSS software (version 16) was used. Among a total of 134 patients, 58 cases (24 ICU and 34 outpatients) were included in the study. The mean and median age was 56 and 62 years, respectively. From all included, 33 men (57%), 25 women (43%), 52 urban (89.7%), 6 rural (10.3%), 41 alive (70.7%), and 17 died (29.3%) were recorded. From all included patients, 49.1% hyperglycemia, 38% hyponatremia, 7.3% hypokalemia, and 32% hypomagnesemia were observed. Unlike the mean of age and the level of  $K^+$ , there was a statistically significant difference between the outpatient and ICU groups in terms of  $Mg^{2+}$ ,  $Na^+$ , and FBS ( $p < 0.05$ ). Hyperglycemia and electrolyte imbalance in COVID-19 patients is feasible. Therefore, notice to measuring these cases and monitoring the patient can be effective in the treatment process and prevent the serious complications of the disease.

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**Keywords:** Blood glucose, COVID-19, electrolytes, magnesium, potassium, sodium

**Original Submission:** 18 September 2020; **Revised Submission:** 26 October 2020; **Accepted:** 2 November 2020

**Article published online:** 7 November 2020

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## Introduction

Since December 2019, when coronavirus disease 2019 (COVID-19), an infectious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), appeared in Wuhan China, various reports have been published on the clinical status of infected patients. A number of these reports indicated hyperglycaemia [1] and electrolyte imbalances in

patients [2]. It was suggested that uncontrolled hyperglycaemia causes an increase in length of hospitalization and mortality caused by SARS-CoV-2 infection [3]. Electrolyte imbalances lead to cardiovascular and renal involvement [4].

The renin-angiotensin-aldosterone system is involved in electrolyte balance and blood pressure stabilization [5]. Angiotensin-converting enzyme 2 (ACE2) is a known receptor for the SARS-CoV-2 virus spike complex [6]. This receptor is present in the respiratory, gastrointestinal and urinary tracts. The virus can induce various clinical manifestations or complications by binding to these receptors [7]. Electrolyte imbalance following the binding of the virus to the ACE2 receptor may contribute to metabolic disorders. Hence, knowledge of such metabolic disturbances caused by SARS-CoV-2 infection, which in turn will lead to immune disorders [8], can play a role in disease prognosis and initial clinical measures and monitoring of

disease. Understanding the potential pathophysiological mechanisms of COVID-19 may offer an opportunity to establish a novel therapeutic strategy. The heterogeneity of reports of metabolic and electrolyte disturbances caused by SARS-CoV-2 has placed restrictions on the detailed analysis of this condition. Access to more reports would greatly reduce these limitations.

In this study, we provide a report on the status of electrolytes of magnesium ( $Mg^{2+}$ ), potassium ( $K^+$ ) and sodium ( $Na^+$ ), and of fasting blood sugar (FBS) at the initial admission with COVID-19 of individuals with no underlying disease. We also compare these factors between individuals with severe and less severe COVID-19.

## Materials and methods

This cross-sectional study was performed at Golestan Hospital, the main referral centre for individuals with COVID-19 in Kermanshah, western Iran, from April to July 2020. Our population consisted of individuals who received a diagnosis of COVID-19 according to PCR and/or chest CT scans.

All patients who had a record of FBS,  $Mg^{2+}$ ,  $K^+$  and  $Na^+$  plasma tests in their clinical files at the time of admission to the hospital from 21 April to 12 July 2020 (the test period) were listed. Participants were divided into outpatient (as less severe COVID-19) and intensive care unit (ICU) (severe COVID-19). All demographic, epidemiological and laboratory data were recorded in a form provided for this purpose. After reviewing the clinical records, those with a history of cancer, diabetes, hypertension, or cardiovascular and renal disorders, were excluded from the study and those who did not have any history of these diseases were included in the investigation. Fluctuations in FBS,  $Mg^{2+}$ ,  $K^+$  and  $Na^+$  plasma levels due to SARS-CoV-2 infection were examined and compared between the severe and less severe COVID-19 groups.

Individuals in each group were divided according to the study variables as follows: four levels of  $Na^+$ ,  $K^+$  and  $Mg^{2+}$  in plasma (normal, hypo-, severe hypo- and hyper-) and two levels of FBS (normal and hyperglycemia).

The SPSS software v.16 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. The quantitative variables were shown by mean  $\pm$  standard deviation, and median. Depending on the type of variables, t-test and  $\chi^2$  tests or other non-parametric equivalents were used for comparison of the two groups.

## Results

A total of 134 individuals were included in the study: 65 (48%) in the ICU and 69 outpatient cases (51.5%). After reviewing the clinical records, 76 (56.7%) had an underlying disease, of which 41 were ICU patients and 35 were outpatients.

Finally, 58 patients were included in the research (24 ICU cases and 34 outpatient cases) to evaluate the state of FBS and electrolytes.

Mean age of patients was 56 years (14–94 years), and the median was 62 years. Patients comprised 33 men (57%), 25 women (43%), and 52 (89.7%) were from urban areas and 6 (10.3%) were from rural areas. Forty-one patients (70.7%) survived (33% of ICU patients and 97% of outpatients) and 17 (29.3%) died from COVID-19 (66% of ICU patients and 3% of outpatients). Those who died included 11 men and 6 women and the living patients were 22 men and 19 women.

There was no significant difference between the mean age of individuals in the ICU and outpatient groups ( $p$  0.489). The  $\chi^2$  test also showed that there was no significant difference between urban and rural living ( $\chi^2_{(1, n = 134)} = 0.12$ ,  $p = 0.20$ ,  $\phi = 0.125$ ).

In regards to blood sugar levels, 47.3% of patients had normal blood sugar levels (29.6% of ICU patients and 70% of outpatients), 3.5% had hypoglycaemia and 49.1% were hyperglycaemic (Table 1). In terms of blood sodium levels, 55% of patients had normal levels, 38% had hyponatraemia and 7% had hypernatremia (Table 2). For the blood potassium levels, 85% of patients were in the normal range, 1.8% were hypokalaemic, 7.3% were severely hypokalaemic and 5.5% were hyperkalaemic (Table 3). Blood magnesium levels were normal in 48% of

**TABLE 1.** Frequency and percentage of blood glucose level in two groups of COVID-19 patients without underlying disease at the point of admission to hospital

Group	Frequency and percent	FBS level in plasma			Total
		Normal (70–110 mg/dL)	Hypoglycaemia (<65 mg/dL)	Hyperglycaemia (>115 mg/dL)	
ICU	Count	8	1	15	24
	% within FBS	29.6%	50.0%	53.57%	42.1%
Outpatient	Count	19	1	13	33
	% within FBS	70%	50.0%	46.43%	57.9%
Total	Count	27	2	28	57
	% within FBS	100.0%	100.0%	100.0%	100.0%

Abbreviations: FBS, fasting blood sugar; ICU, intensive care unit.

**TABLE 2.** Frequency and percentage of blood sodium level in two groups of COVID-19 patients without underlying disease at the point of admission to hospital

Group	Frequency and percent	Na <sup>+</sup> level in plasma			Total
		Normal (135–145 mEq/L)	Hyponatraemia (121–134 mEq/L)	Hypernatraemia (>146 mEq/L)	
ICU	Count	12	8	4	24
	% within Na <sup>+</sup>	37.5%	36.4%	100.0%	41.4%
Outpatient	Count	20	14	0	34
	% within Na <sup>+</sup>	62.5%	63.6%	.0%	58.6%
Total	Count	32	22	4	58
	% within Na <sup>+</sup>	100.0%	100.0%	100.0%	100.0%

Abbreviation: ICU, intensive care unit.

**TABLE 3.** Frequency and percentage of blood potassium level in two groups of COVID-19 patients without underlying disease at the point of admission to hospital

Group	Frequency and percent	K <sup>+</sup> level in plasma				Total
		Normal (3.5–5 mEq/L)	Hypokalaemia (3–3.4 mEq/L)	Severe hypokalaemia (<3 mEq/L)	Hyperkalaemia (>5.5 mEq/L)	
ICU	Count	19	0	2	2	23
	% within K <sup>+</sup>	40.4%	.0%	50.0%	66.7%	41.8%
Outpatient	Count	28	1	2	1	32
	% within K <sup>+</sup>	59.6%	100.0%	50.0%	33.3%	58.2%
Total	Count	47	1	4	3	55
	% within K <sup>+</sup>	100%	100%	100%	100%	100%

Abbreviation: ICU, intensive care unit.

**TABLE 4.** Frequency and percentage of blood magnesium level in two groups of COVID-19 patients without underlying disease at the point of admission to hospital

Group	Frequency and percent	Mg <sup>2+</sup> level in plasma				Total
		Normal (1.8–2.2 mg/dL)	Hypo-magnesaemia (1.26–1.7 mg/dL)	Severe hypo-magnesaemia (<1.25 mg/dL)	Hyper-magnesaemia (>2.6 mg/dL)	
ICU	Count	5	8	2	5	20
	% within Mg <sup>2+</sup>	20.8%	50.0%	66.7%	71.4%	40%
Outpatient	Count	19	8	1	2	30
	% within Mg <sup>2+</sup>	79.2%	50.0%	33.3%	28.6%	60%
Total	Count	24	16	3	7	50
	% within Mg <sup>2+</sup>	100.0%	100.0%	100.0%	100.0%	100%

Abbreviation: ICU, intensive care unit.

patients, 32% were hypomagnesaemic, 6% were severely hypomagnesaemic and 14% were hypermagnesaemic (Table 4).

The sample t-test showed that there was a significant difference between the increase in blood glucose levels due to disease between the ICU and outpatient groups ( $p = 0.042$ ) and the mean  $\pm$  SD of glucose level in ICU patients ( $143 \pm 54.5$  mg/dL) was more than that in outpatients ( $116 \pm 42.2$  mg/dL). The mean glucose level of those who died due to disease was  $156 \pm 56.7$  mg/dL and that of those who survived was  $115 \pm 40.6$  mg/dL. There was a statistically significant difference between the glucose levels of the two groups ( $p = 0.003$ ).

There was a significant difference between the outpatient and ICU groups in terms of blood sodium levels (both hyponatraemia and hypernatraemia), ( $\chi^2_{(2, n = 58)} = 0.32, p = 0.04, \phi = 0.32$ ).

There was no significant difference in blood potassium levels between the two groups.

The frequency of people with normal blood magnesium levels was higher in the outpatient group than in the ICU group. Also, hypermagnesaemia in the ICU group was higher than in the outpatient group and there was a significant difference between the two groups ( $\chi^2_{(3, n = 50)} = 0.4, p = 0.04, \phi = 0.4$ ).

## Discussion

Our aim was to study electrolytes ( $Mg^{2+}$ ,  $K^+$  and  $Na^+$ ) and glucose concentration in serum of patients with severe (ICU) and less severe (outpatient) COVID-19 after diagnosis of disease and before any intervention therapy. Previous studies suggested some imbalances in serum electrolytes and glucose levels in individuals with COVID-19 [1,2,9].

Hyperglycaemia can be a result of related conditions such as severe sepsis, systemic inflammatory response syndrome and traumatic brain injury. The initial response to these conditions is an increase in the levels of some cytokines, which is accompanied by high levels of blood glucose (hyperglycaemia). It has been shown that there is a correlation between glucose blood levels and morbidity/mortality of patients [10,11]. The characteristic of the pro-inflammatory phase in critical cases of these conditions is metabolic stress, which leads to the breakdown of glycogen, the synthesis of adrenocorticotrophic and glucagon hormones, and insulin resistance, which all cause an increase of blood glucose [12]. In our study, glucose levels were higher in those individuals who died, and there was a significant difference in glucose levels between the two groups. Studies of risk factors for the mortality and morbidity of community-acquired pneumonia, SARS and Middle East respiratory syndrome, have shown that hyperglycaemia and/or diabetes are involved [13,14]. Wang *et al.* indicated that the level of FBS at the time of admission is a significant prognostic factor for COVID-19 [1]. In our study, there was a significant difference between the increase in the level of FBS at the time of admission between the severe and less severe COVID-19 groups and, consistent with the results of Wang *et al.*, the level of FBS was higher in the ICU group than in the outpatients. The mean level of blood glucose in outpatients was not as high as in ICU patients. According to the previous study, a mild increase in blood glucose may relate to patient stress [15]. However, it was suggested that acute insulin resistance, characterized by hyperglycaemia and hyperinsulinaemia, causes a higher level of glucose, which has been shown in ICU patients [12].

During the 2003 SARS epidemic, patients with no history of diabetes revealed hyperglycaemia. In immunohistochemical staining of the pancreatic tissue of these patients, ACE2 receptors were shown, similar to the myocardium and alveolar epithelium of the lung. All reported symptoms of COVID-19 (dyspnoea, diarrhoea, acute heart damage and kidney failure) could be correlated with organ expression of ACE2. The expression of this receptor in the endocrine part of the pancreas indicates that the coronavirus that causes SARS, SARS-CoV, enters the islet cells using ACE2 as a receptor and causes acute diabetes by damaging these cells [16].

According to the pathophysiology of SARS-CoV-2, the virus upon binding to its receptor, ACE2, is likely to reduce ACE2 protein expression and cause an increase of angiotensin II, which can increase renal potassium excretion, eventually leading to hypokalaemia [17]. Chen *et al.* reported a high prevalence of hypokalaemia in individuals with COVID-19 [9]. In our study, only 10% of participants with both severe and less severe forms of COVID-19 developed hypokalaemia. One of the reasons for this discrepancy in the results of patients' blood potassium concentrations was the inclusion of patients with underlying cardiovascular disease and type 2 diabetes by Chen *et al.*, whereas in our study, all those with the underlying disease were excluded [9]. Also, we only examined electrolytes at the time of admission and did not follow the results for the following days, so our findings are based on the initial results.

There is less information about the status of magnesium in individuals with COVID-19, but it cannot be ruled out that there is an inverse relationship between hypomagnesaemia and increased inflammatory markers, including interleukin-6, tumour necrosis factor- $\alpha$ , soluble intracellular adhesion molecule 1, soluble vascular cell adhesion molecule 1 and C-reactive protein, which also increase in this disease [18]. Magnesium co-supplementation has been shown to down-regulate the genes controlling the expression of interleukin-1 and tumour necrosis factor- $\alpha$  [8]. Magnesium is also known as a calcium-channel blocker and inhibits the influx of calcium to immunocompetent cells, thereby preventing the activation of nuclear factor- $\kappa B$ , the production of cytokines, especially interleukin-6, and also systemic inflammation [19]. Therefore, it seems that the decrease in magnesium due to COVID-19 will exacerbate the inflammation caused by the virus and cause a cytokine storm. According to the hypotheses of Lotti *et al.*, individuals with COVID-19 manifest with hypomagnesaemia; one of the reasons for this is the stress caused by this pandemic, which, under the effect of stress hormones catecholamines and corticosteroids, magnesium shifts to the extracellular space and urinary excretion of  $Mg^{2+}$  increases. This also starts a defective cycle, releasing more stress hormones and exacerbating the hypomagnesaemia [20].

As hypomagnesaemia affects the performance of the sodium-potassium ATPase pump, the intracellular potassium concentration decreases following hypomagnesaemia. As a result of hypomagnesaemia, the renal outer medullary potassium channel causes a decrease in potassium by increasing the distal secretion of potassium. Increased distal sodium delivery and aldosterone may also contribute to potassium loss along with magnesium depletion [21]. In our study, 38% of patients developed hypomagnesaemia, which did not differ in frequency between the groups with severe and less severe disease. The prevalence of hypomagnesaemia was higher than that of

hypokalaemia; this may be the reason why hypomagnesaemia precedes hypokalaemia.

Hyponatraemia has been reported in COVID-19 [22–24]. In a retrospective study conducted by Zhang et al., the correlation between hyponatraemia and the severity of COVID-19 was considered [25]. In a review study of electrolyte imbalances in patients with COVID-19, five studies were identified with a total of 1415 participants, indicating a relationship between decreased blood sodium and disease severity [2]. As one of the expression sites of the ACE2 receptor is in the proximal tubule [26], hyponatraemia can occur due to increased expression of the ACE2 receptor in the proximal tubule. In an individual with severe hyponatraemia, it was shown that SARS-CoV-2 causes a syndrome of inappropriate secretion of antidiuretic hormone and manifestations of hyponatremia [27]. The results of the present study show that hyponatraemia was more common in outpatients than in severe patients, but all cases of hypernatraemia were observed in patients with severe disease.

According to the results of this study, hyperglycaemia and electrolyte imbalance occur in individuals with COVID-19. Measurement of these factors and monitoring the patient can be effective in the treatment process and may help to prevent the serious complications of this disease.

## Funding

Imam Reza Hospital Clinical Research Development Center was supportive in collecting data and teaching the correct writing of the article, but there was no financial support in writing this article.

## Authors' contributions

All authors contributed to the design and execution of the study, participated in article drafting and critical revision, and read and approved the final version of the manuscript.

## Conflict of interest

The authors have stated that there are no conflicts of interest.

## Acknowledgements

We are grateful to the Clinical Research Development Centre of Imam Reza Hospital for their kind assistance and advice.

## References

- [1] Wang S, Ma P, Zhang S, Song S, Wang Z, Ma Y, et al. Fasting blood glucose at admission is an independent predictor for 28-day mortality in patients with COVID-19 without a previous diagnosis of diabetes: a multi-center retrospective study. *Diabetologia* 2020. epub ahead of print.
- [2] Lippi G, South AM, Henry BM. Electrolyte imbalances in patients with severe coronavirus disease in 2019 (COVID-19). *Ann Clin Biochem* 2020;57:262–5. <https://journals.sagepub.com/doi/full/10.1177/0004563220922255>.
- [3] Bode B, Garrett V, Messler J, McFarland R, Crowe J, Booth R, et al. Glycemic characteristics and clinical outcomes of COVID-19 patients hospitalized in the United States. *J Diabetes Sci Technol* 2020;14:813–21.
- [4] Post A, Dullaart RPF, Bakker SJL. Is low sodium intake a risk factor for severe and fatal COVID-19 infection? *Eur J Intern Med* 2020;75:109.
- [5] Muñoz-Durango N, Fuentes CA, Castillo AE, González-Gómez LM, Vecchiola A, Fardella CE, et al. Role of the renin-angiotensin-aldosterone system beyond blood pressure regulation: molecular and cellular mechanisms involved in end-organ damage during arterial hypertension. *Int J Mol Sci* 2016;17(7):797. <https://doi.org/10.3390/ijms17070797>.
- [6] Cao Y, Li L, Feng Z, Wan S, Huang P, Sun X, et al. Comparative genetic analysis of the novel coronavirus (2019-nCoV/SARS-CoV-2) receptor ACE2 in different populations. *Cell Discov* 2020;6:11.
- [7] Xu H, Zhong L, Deng J, Peng J, Dan H, Zeng X, et al. High expression of ACE2 receptor of 2019-nCoV on the epithelial cells of the oral mucosa. *Int J Oral Sci* 2020;12:8.
- [8] Afshar Ebrahimi F, Foroozanfar F, Aghadavod E, Bahmani F, Asemi Z. The effects of magnesium and zinc co-supplementation on biomarkers of inflammation and oxidative stress, and gene expression related to inflammation in polycystic ovary syndrome: a randomized controlled clinical trial. *Biol Trace Elem Res* 2018;184:300–7.
- [9] Chen D, Li X, Song Q, Hu C, Su F, Dai J, et al. Assessment of hypokalemia and clinical characteristics in patients with coronavirus disease 2019 in Wenzhou, China. *JAMA Netw Open* 2020;3(6). e2011122-e.
- [10] Osterbur K, Mann FA, Kuroki K, DeClue A. Multiple organ dysfunction syndrome in humans and animals. *J Vet Intern Med* 2014;28(4):1141–51.
- [11] Whitcomb BW, Pradhan EK, Pittas AG, Roghmann M-C, Perencevich EN. Impact of admission hyperglycemia on hospital mortality in various intensive care unit populations\*. *Critic Care Med* 2005;33:2772–7. <https://doi.org/10.1097/01.ccm.0000189741.44071.25>.
- [12] Bar-Or D, Rael LT, Madayag RM, Banton KL, Tanner A, Acuna DL, et al. Stress hyperglycemia in critically ill patients: insight into possible molecular pathways. *Front Med* 2019;6:54.
- [13] Alanazi K, Abedi G, Midgley C, Alkhamis A, Alsaqer T, Almoaddi A, et al. Diabetes mellitus, hypertension, and death among 32 patients with MERS-CoV infection, Saudi Arabia. *Emerg Infect Dis J* 2020;26:166.
- [14] Lepper PM, Ott S, Nüesch E, von Eynatten M, Schumann C, Pletz MW, et al. Serum glucose levels for predicting death in patients admitted to hospital for community-acquired pneumonia: a prospective cohort study. *BMJ (Clin Res ed)* 2012;344:e3397.
- [15] McAlister FA, Majumdar SR, Blitz S, Rowe BH, Romney J, Marrie TJ. The relation between hyperglycemia and outcomes in 2,471 patients admitted to the hospital with community-acquired pneumonia. *Diabet Care* 2005;28:810.
- [16] Yang J-K, Lin S-S, Ji X-J, Guo L-M. The binding of SARS coronavirus to its receptor damages islets and causes acute diabetes. *Acta Diabetol* 2010;47:193–9.
- [17] Kuba K, Imai Y, Rao S, Gao H, Guo F, Guan B, et al. A crucial role of angiotensin-converting enzyme 2 (ACE2) in SARS coronavirus-induced lung injury. *Nat Med* 2005;11:875–9.

- [18] Costello RB, Elin RJ, Rosanoff A, Wallace TC, Guerrero-Romero F, Hruby A, et al. Perspective: the case for an evidence-based reference interval for serum magnesium: the time has come. *Adv Nutr* 2016;7: 977–93.
- [19] Sugimoto J, Romani AM, Valentin-Torres AM, Luciano AA, Kitchen CMR, Funderburg N, et al. Magnesium decreases inflammatory cytokine production: a novel innate immunomodulatory mechanism. *J Immunol* 2012;188:6338–46.
- [20] Iotti S, Wolf F, Mazur A, Maier JA. The COVID-19 pandemic: is there a role for magnesium? Hypotheses and perspectives. *Magnes Res* 2020;33:21–7. <https://doi.org/10.1684/mrh.2020.0465>.
- [21] Huang C-L, Kuo E. Mechanism of hypokalemia in magnesium deficiency. *J Am Soc Nephrol* 2007;18:2649–52.
- [22] Guan W-j, Ni Z-y, Hu Y, Liang W-h, Ou C-q, He J-x, et al. Clinical characteristics of coronavirus disease 2019 in China. *N Engl J Med* 2020;382:1708–20.
- [23] Huang C, Wang Y, Li X, Ren L, Zhao J, Hu Y, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *Lancet* 2020;395(10223):497–506.
- [24] Sever MY, Uzun N, Ceritli S, Mutlu H, Bayramoğlu A. Hyponatremia in COVID-19 patient using angiotensin type I receptor (AT1R) blocker and diuretic: a case report. *J Res Clin Med* 2020;8:22.
- [25] Zhang W, Lu S, Zhang M, Zheng H, Huang Y, Chen S, et al. [Correlation between hyponatremia and the severity of coronavirus disease 2019]. *Zhonghua Wei Zhong bing Ji jiu Yi Xue* 2020;32:774–8.
- [26] Zhang Q-L, Duan T, Jin L-P. Single-cell RNA expression profiling of ACE2 and AXL in the human maternal–fetal interface. *Reprod Dev Med* 2020;4:7–10.
- [27] Habib MB, Sardar S, Sajid J. Acute symptomatic hyponatremia in the setting of SIADH as an isolated presentation of COVID-19. cases. *ID Cases* 2020;21. <https://doi.org/10.1016/j.idcr.2020.e00859>.