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A Retrospective Study of Prevalence of Electrolyte Imbalance on Presentation and its Correlation with Disease Severity in COVID 19

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ABSTRACT

Hypokalemia and other electrolyte disorders may have clinically significant implications for patient management and potentially contribute to unraveling pathogenetic mechanisms underlying COVID-19. Present study aimed to assess the prevalence of electrolyte imbalance and correlate with disease severity of COVID-19. This retrospective study was conducted among the patients attended to AVMC hospital with RTPCR positive for Covid-19 and/ or HRCT findings suggestive of COVID pneumonia. Patients with CKD, cirrhosis, CAD, systemic hypertension, T2DM and patients on drugs like diuretics, ACEI and ARBs were excluded from the study. The diagnosed patients were analysed for electrolyte and correlated with the severity and outcome of the disease. Total of 250 patients fulfilling inclusion criteria are included in the present study. The mean age of patients was found to be 53.4±16.3yrs of age, with 53.9% were female patients. 55.8% of the patients had the electrolyte abnormality at baseline. Hyponatremia (35.8%) was the most frequent baseline electrolyte abnormality documented. Hypokalemia and Hypochloremia were found in 6.8% of patients each. Patient's severity of disease, ICU admission was higher among the patients with hyponatremia compared to the patients with lower severity. Baseline electrolyte abnormalities, particularly hyponatraemia, are associated with a poor prognosis in COVID-19 and baseline electrolyte testing, even after hospitalisation, might be useful in determining the risk of severe COVID-19.

INTRODUCTION

Coronavirus disease 2019 (COVID-19) is a contagious disease caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The first known case was identified in Wuhan, China in December 2019^[1]. The disease has since spread worldwide, leading to an ongoing pandemic. The COVID-19 pandemic is an emerging threat to global public health. While our current understanding of COVID-19 pathogenesis is limited, a better understanding will help us develop efficacious treatment and prevention strategies for COVID-19. The virus efficiently binds to the angiotensin converting enzyme 2 (ACE2) receptor which is highly expressed in many organs including the bronchus and lung parenchyma, heart, kidney and gastrointestinal tract. The lungs are the organs most affected by COVID-19 because the virus accesses host cells via the receptor for the enzyme angiotensin-converting enzyme 2 (ACE2), which is most abundant on the surface of type II alveolar cells of the lungs. The virus uses a special surface glycoprotein called a "spike" to connect to the ACE2 receptor and enter the host cell^[2,3].

The virus also affects gastrointestinal organs as ACE2 is abundantly expressed in the glandular cells of gastric, duodenal and rectal epithelium as well as endothelial cells and enterocytes of the small intestine. The exact mechanism of kidney involvement is unclear and likely multifactorial. Kidney disease may be caused by SARS-CoV-2 binding to the ACE2 receptor on kidney cells that allows the virus to enter. Moreover, normal kidney and intestinal tract have higher ACE2 expression than lungs. Electrolyte disorders are not uncommon in patients with COVID-19 and severe COVID-19 has frequently shown hypokalemia, hyponatremia and hypocalcemia. Electrolyte imbalances are caused by alteration of Renin Aldosterone system (RAS), gastrointestinal loss, effects of proinflammatory cytokines and renal tubular dysfunction by the invasion of SARS-CoV-2^[4]. Angiotensin-converting enzyme 2 (ACE2) plays an important role in the non-classical RAS pathway and binds to a receptor binding domain of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). The RAS blockade is known to increase ACE2 levels, but controversy remains regarding the effect of RAS blockade therapy in the course of COVID-19. Some reports have indicated a protective effect of RAS blockade on COVID-19, whereas others have reported an association of RAS blockade therapy with the occurrence of severe complications such as acute kidney injury and admission to the intensive care unit. A study which pooled data from different studies on sodium, potassium, chloride and calcium confirms that COVID-19 severity is associated with lower serum concentrations of sodium, potassium and calcium^[4-6].

The present study aimed to assess the prevalence of electrolyte imbalance among the patients with COVID-19 and also to establish the correlation of electrolyte imbalance with disease severity.

MATERIAL AND METHOD

This retrospective study was conducted among the patients attended to AVMC hospital with RTPCR positive for Covid-19 and/ or HRCT findings suggestive of COVID pneumonia. Patients with CKD, cirrhosis, CAD, systemic hypertension, T2DM and patients on drugs like diuretics, ACEI and ARBs were excluded from the study. The diagnosed patients were analysed for electrolyte and correlated with the severity and outcome of the disease. All the data analysis was performed using SPSS v21 operating on windows 10, with $p < 0.05$ considered as statistically significant.

RESULTS

Total of 250 patients fulfilling inclusion criteria are included in the present study. The mean age of patients was found to be 53.4 ± 16.3 yrs of age, with 53.9% were female patients. 55.8% of the patients had the electrolyte abnormality at baseline. Hyponatremia (35.8%) was the most frequent baseline electrolyte abnormality documented. Hypokalemia (7.1%) and Hypochloremia were found in 62.4% of patients each. Patient's severity of disease, ICU admission was higher among the patients with hyponatremia compared to the patients with lower severity.

DISCUSSION

Hypokalemia may have clinically significant implications for patient management and potentially contribute to unraveling pathogenetic mechanisms underlying COVID-19. Hypokalemia is known to exacerbate acute respiratory distress syndrome (ARDS) and acute cardiac injury, which are common

Table 1: Demographics and disease related findings in patients

Parameters	Patients (250)
Age (Mean \pm SD)	54.3 \pm 16.3
Gender (M/F)	116/134
SPO ₂ \leq 92%	223(89.2)
Fever n(%)	62 (24.8)
Electrolyte abnormalities, n (%)	176 (70.4)
Co-morbidities, n (%)	
Diabetes mellitus	56 (22.4)
Hypertension	72 (28.8)
CAD	26 (10.4)
COPD	9 (3.6)
Asthma	16 (6.4)

Table 2: Showing the baseline electrolyte abnormality with disease outcome

Mortality,	n(%)	OR	p-value
Hyponatremia	19/146 (13%)	5.4	0.01*
Hypokalemia	2/28 (7.1%)	1.15	0.05*
Hyperkalemia	-	-	-
Hypochloreaemia	6/28 (21.4%)	5.08	0.01*
Hypocalcemia	8/39 (20.5)	5.14	0.05*

complications in COVID-19, especially in patients with underlying lung or heart disease. Hypokalemia also provides a pathophysiologic clue; SARSCoV-2 binds to its host receptor, angiotensin-converting enzyme 2 (ACE2) and likely reduces ACE2 expression, thus leading to increased angiotensin II, which can cause increased potassium excretion by the kidneys, ultimately leading to hypokalemia. A second potential contributor to hypokalemia and other electrolyte imbalance in some COVID-19 patients may be gastrointestinal losses, with diarrhea and vomiting.

In COVID-19 adults, hyponatremia may be linked to the increased release of antidiuretic hormone (ADH) in response to a volume depletion following gastrointestinal fluid losses. In a Cohort study, 26.3% of the patients had diarrhea and 8.7% presented with vomiting, significantly more than non COVID-19 patients. Thus, these symptoms are common in patients with COVID-19 and can trigger the increased ADH release due to extracellular dehydration state. On the other hand, a syndrome of antidiuresis (SIAD) can occur in response to COVID-19 complications like pneumonia or acute respiratory distress syndrome.

In similar to present study Tezcan *et al.*, documented 55.8% of the patients with electrolyte abnormalities at the baseline. The presence of hyponatremia was most frequent abnormality (35.8%) followed with the hypocalcemia (9.5%), hypokalemia and hypochloraemia (6.8%)^[7].

Furthermore, sodium, potassium and chloride levels were found to have a strong predictive potential for COVID-19 developing to severe illness^[8]. In study Tezcan *et al.*, discovered that low baseline salt, chloride and calcium levels were associated with an increased risk of death, increased ICU and MV requirements and longer hospital stays. Various electrolyte imbalances were discovered to be related to severe illness or a bad prognosis in the research listed above. However, hyponatraemia was the most prevalent electrolyte imbalance associated with a poor outcome in these investigations. As a result, examining baseline electrolyte values, particularly sodium status, would aid clinicians in doing a risk assessment for COVID-19 severity^[7].

In concordance study by Carvalho HD *et al.*, documented that Hyponatremia and hypokalemia were both linked with COVID-19 among case patients, with an adjusted odds ratio of 1.89 [95% CI 1.24-2.89] for hyponatremia and 1.76 [95% CI 1.20-2.60] for hypokalemia. Hyponatremia and hypokalemia are independently related with COVID-19 infection in people attending the emergency department and they might serve as surrogate biomarkers for the emergency physician in suspected COVID-19 patients^[9].

Moreno-P *et al.* discovered that hypokalemia (potassium 3.5 mmol/l) was linked with the need for invasive mechanical ventilation (odds ratio: 8.98, 95% CI 2.54-31.74). However, our findings are restricted by the small number of COVID-19 patients admitted in the critical care unit (n = 96) compared to the number of cases with intermediate to severe COVID-19 (n = 498) and controls (n = 594)^[10].

Electrolyte abnormalities in COVID-19 might be caused by a number of factors. The primary aetiology for electrolyte imbalance would be kidney involvement and incorrect anti-diuretic hormone syndrome. Endotheliitis, proximal tubule damage, angiotensin-converting enzyme 2 upregulation in kidney tissue, renal hypoxia and aberrant coagulation might all explain electrolyte imbalances associated with kidney injury^[11-13].

CONCLUSION

Baseline electrolyte abnormalities, particularly hyponatraemia, are associated with a poor prognosis in COVID-19 and baseline electrolyte testing, even after hospitalisation, might be useful in determining the risk of severe COVID-19.

REFERENCES

1. Sharma, A., S. Tiwari, M.K. Deb and J.L. Marty, 2020. Severe acute respiratory syndrome coronavirus-2 (SARS-CoV-2): A global pandemic and treatment strategies. *Int. J. Antimicrob. Agents*, Vol. 56. 10.1016/j.ijantimicag.2020.106054
2. Ni, W., X. Yang, D. Yang, J. Bao and R. Li *et al.*, 2020. Role of angiotensin-converting enzyme 2 (ACE2) in COVID-19. *Crit. Care*, Vol. 24, No. 1. 10.1186/s13054-020-03120-0
3. Beyerstedt, S., E.B. Casaro and É.B. Rangel, 2021. Covid-19: Angiotensin-converting enzyme 2 (ACE2) expression and tissue susceptibility to SARS-CoV-2 infection. *Eur. J. Clin. Microbiol. & Infect. Dis.*, 40: 905-919.
4. Ghazanfar, H., S. Kandhi, D. Shin, A. Muthumanickam and H. Gurjar *et al.*, 2022. Impact of COVID-19 on the gastrointestinal tract: A clinical review. *Cureus*, Vol. 14, No. 3. 10.7759/cureus.23333
5. Hikmet, F., L. Méar, Å. Edvinsson, P. Micke, M. Uhlén and C. Lindskog, 2020. The protein expression profile of ACE2 in human tissues. *Mol. Syst. Biol.*, Vol. 16, No. 7. 10.15252/msb.20209610
6. Salamanna, F., M. Maglio, M.P. Landini and M. Fini, 2020. Body localization of ACE-2: On the trail of the keyhole of SARS-CoV-2. *Front. Med.*, Vol. 7. 10.3389/fmed.2020.594495

7. Tezcan, M.E., G.D. Gokce, N. Sen, N.Z. Kaymak and R.S. Ozer, 2020. Baseline electrolyte abnormalities would be related to poor prognosis in hospitalized coronavirus disease 2019 patients. *New Microbes New Infec.*, Vol. 37 .10.1016/j.nmni.2020.100753
8. Duan, J., X. Wang, J. Chi, H. Chen and L. Bai *et al.*, 2020. Correlation between the variables collected at admission and progression to severe cases during hospitalization among patients with COVID 19 in Chongqing. *J. Med. Virol.*, 92: 2616-2622.
9. Carvalho, H.D., M.C. Richard, T. Chouihed, N. Goffinet and Q.L. Bastard *et al.*, 2021. Electrolyte imbalance in COVID-19 patients admitted to the emergency department: A case-control study. *Internal Emergency Med.*, 16: 1945-1950.
10. Moreno-Pérez, O., J.M. Leon-Ramirez, L. Fuertes-Kenneally, M. Perdiguero and M. Andres *et al.*, 2020. Hypokalemia as a sensitive biomarker of disease severity and the requirement for invasive mechanical ventilation requirement in COVID-19 pneumonia: A case series of 306 mediterranean patients. *Int. J. Infect. Dis.*, 100: 449-454.
11. Su, H., M. Yang, C. Wan, L.X. Yi and F. Tang *et al.*, 2020. Renal histopathological analysis of 26 postmortem findings of patients with COVID-19 in China. *Kidney Int.*, 98: 219-227.
12. Yousaf, Z., S.D. Al-Shokri, H. Al-soub and M.F.H. Mohamed, 2020. Covid-19-associated SIADH: A clue in the times of pandemic!. *Am. J. Physiol.-Endocrinol. Metab.*, 318:.
13. Varga, Z., A.J. Flammer, P. Steiger, M. Haberecker and R. Andermatt *et al.*, 2020. Endothelial cell infection and endotheliitis in COVID-19. *The Lancet*, 395: 1417-1418.