Content available at: https://www.ipinnovative.com/open-access-journals

Panacea Journal of Medical Sciences

Journal homepage: http://www.pjms.in/

Original Research Article

Association of serum potassium levels with severity of COVID infection and requirement of invasive mechanical ventilation in COVID 19 patients

Arun Suresh[1],*, Vishnu Vijay[1], Ruchi Tandon[1]

[1]*Dept. of Anaesthesiology, Gandhi Medical College, Bhopal, Madhya Pradesh, India*

ARTICLE INFO

Article history:
Received 27-06-2021
Accepted 19-10-2021
Available online 17-08-2022

Keywords:
Hypokalemia
COVID 19
Invasive ventilation
Clinical outcome

ABSTRACT

Background: The study was conducted to assess the prevalence of hypokalemia in COVID 19 patients and its correlation with severity of infection and requirement of invasive mechanical ventilation.
Materials and Methods: This was a prospective study conducted on COVID-19 patients admitted at tertiary care centre during the study period of 6 months. Detailed history was obtained and patients were subjected to routine and special investigations. Based upon serum potassium levels, patients were categorized into three groups i.e. normokalemia (>3.5 mmol/L), mild hypokalemia (3 to 3.5 mmol/L) and severe hypokalemia (<3 mmol/L). The outcome of patient was noted i.e. requirement for ICU admission or invasive ventilation or mortality.
Results: The study was conducted on 100 patients with mean age of 66.6±12.5 years. Hypokalemia was present in 31% cases. Of them, 25% had mild and 6% cases had severe hypokalemia. Hypokalemia was significantly associated with shorter duration of symptoms; higher mean diastolic blood pressure; higher CURB 65 score; lymphocytopenia; raised serum ferritin, D-dimer and Trop T levels ($p<0.05$) Hypokalemia was significantly associated with long duration of hospital stay, higher rate as well as duration of ICU admission and need for mechanical ventilation ($p<0.05$). Hypokalemia was an independent predictor of invasive mechanical ventilation (OR- 6.74; 95% CI- 1.39-16.85; $p<0.01$).
Conclusions: Hypokalemia is common in COVID 19 patients among Indian population. Hypokalemia is associated with severe illness and significantly affect clinical outcome. Hypokalemia is an independent predictor of risk of mechanical ventilation after adjusting for confounding variables.

1. Introduction

Coronavirus, a dreadful virus, responsible for one of the greatest pandemic has affected the individuals all over the World. The disease emerged in 2019 in the city of Wuhan China and has been recognized as the etiological factor for severe pneumonia cases.[1] The International Committee of taxonomy of viruses named the virus as severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). Electrolyte abnormalities are commonly observed in COVID 19 patients. This has been attributed to effect of SARS CoV-2 on angiotensin I converting enzyme 2 (ACE2). ACE 2 is distributed widely in vital organs such as heart, liver, kidney, and lungs.[2] This enzyme regulate renin–angiotensin system (RAS) which plays an important role in regulation of blood pressure as well as electrolyte balance.[3] SARS CoV binds to ACE2 and enhance its degradation. Thus, its counter effect on renin angiotensin system is affected leading to increased reabsorption of sodium and water, ultimately contributing to elevated blood pressure and potassium (K^+) excretion.[4] Further, electrolyte imbalance is aggravated by co-existing Gastrointestinal symptoms such as vomiting and diarrhea in COVID 19 patients.[5]

* *Corresponding author*.
E-mail address: suresharun9@gmail.com (A. Suresh).

https://doi.org/10.18231/j.pjms.2022.067

Literature suggest that electrolyte imbalance is often observed in patients at the time of admission with tendency to severe disease. Electrolyte imbalance can affect patient care as well as provide an insights into the pathophysiology of COVID-19. Lippi et al in recent study documented significantly lower serum potassium levels in COVID-19 patients with severe disease.[6]

O'Monero et al in their study in Spain documented hypokalemia to be an independent predictor of severity of infection and invasive mechanical ventilation requirement.[7] However such data is lacking in developing country like India. The present study was therefore conducted at tertiary care centre to study the prevalence of hypokalemia in COVID 19 patients and its correlation with severity of infection and requirement of invasive mechanical ventilation.

2. Materials and Methods

The present study was conducted as a prospective study among patients with COVID-19 pneumonia admitted in Covid care centre, Gandhi Medical College and Hamidia Hospital Bhopal during the period of 6 months i.e. from 1^{st} September 2020 to 28^{th} February 2021. Criteria for hospital admission at present institution was advanced age, presence of comorbidities, severe symptoms or poor clinical status, hypoxemia on room air (oximetry 3.5 mmol/l).[8] A total of 100 confirmed cases of COVID 19 infection by positive RTPCR admitted during the study period belonging to age range of 18 to 80 years were included whereas patients whose test results were negative or unavailable were excluded from the study.

After obtaining ethical clearance from Institute's ethical committee, written consent was obtained from all the study participants fulfilling the inclusion criteria. Detailed information regarding sociodemographic variables was obtained and entered in questionnaire. Clinical history was obtained in detail including presence of comorbidities and drug history. All the patients were subjected to detailed clinical examination; routine and special investigations such as CBC, arterial blood gas analysis, D-Dimer, S. ferritin, Procalcitonin, Chest X-ray etc. ABG was done at the time of admission as well as at 72 hours of admission. Based upon serum potassium levels, patients were categorized into three groups i.e. normokalemia (>3.5 mmol/L), mild hypokalemia (3 to 3.5 mmol/L) and severe hypokalemia (<3 mmol/L). The outcome of patient was noted i.e. requirement for ICU admission or invasive ventilation or mortality.

2.1. Statistical analysis

Data was compiled using MsExcel and analysed using IBM SPSS software version 20. Categorical variables were expressed as frequency and proportion whereas continuous variables were expressed as median (interquartile range (IQR). The Mann-Whitney U-test and Chi-square test were used for group comparisons for continuous and categorical variables respectively. Multiple logistic regression was used to assess the association between hypokalemia and the clinical outcomes. Spearman correlation coefficient was calculated to correlate serum potassium levels at admission and at 72 hours of admission. P value less than 0.05 was considered statistically significant.

3. Results

A total of 100 patients were enrolled with mean age of 66.6±12.5 years [Median- 62 (IQR- 44-76). About 53% cases were males and 47% cases were females.

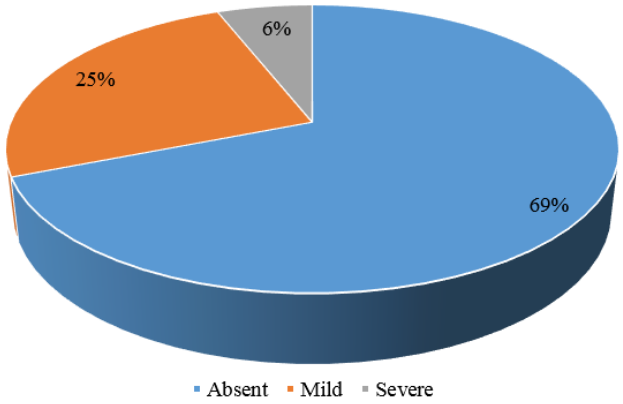

Fig. 1: Prevalence of Hypokalemia after 72 hours of admission

At the time of admission median potassium level was 4.4 mmol/l (IQR 3.6–4.7 mmol/l). Hypokaelmia was present in 12% cases and none of patient had severe hypokalemia. However, after 72 hours of admission, median potassium level was 3.6 mmol/L (IQR-2.8-4.2) whereas Hypokalemia was present in 31% cases. Of them, 25% had mild and 6% cases had severe hypokalemia (Figure 1). We documented a statistically significant positive correlation between potassium level at the time of admission and potassium levels after 72 hours of admission (Spearman's rho = 0.74, p<0.001).

Above table reveals association of serum potassium levels with demographic and clinical parameters. Hypokalemia was significantly associated with shorter duration of symptoms; higher mean diastolic blood pressure; higher CURB 65 score; lymphocytopenia; raised serum ferritin, D-dimer and Trop T levels (p<0.05) (Table 1).

Table 2 reveal that hypokalemia (both mild as well as severe) was significantly associated with long duration of hospital stay, higher rate as well as duration of ICU admission and need for mechanical ventilation (p<0.05). However, we documented no significant association of hypokalemia with mortality (p>0.05).

Table 1: Association of hypokalemia withsociodemographic and clinical parameters

Variables		Normokalemia (n=69)	Mild hypokalemia (n=25)	Severe hypokalemia (n=6)	p [1]	p [2]	p [3]	p [4]
Demography	Age (median) years	61 (44-68)	64 (58-74)	71 (68-76)	0.13	0.59	0.26	0.08
	Gender (%) Male	36 (52.2)	13 (52)	4 (66.7)	0.80	0.99	0.48	0.53
	Gender (%) Female	33 (47.8)	12 (48)	2 (33.3)				
Comorbidities	Hypertension (%)	33 (47.8)	14 (56)	4 (66.7)	0.35	0.49	0.38	0.64
	Diabetes (%)	19 (27.5)	7 (28)	2 (33.3)	0.60	0.96	0.76	0.79
	CVD (%)	16 (23.2)	5 (20)	1 (16.7)	0.67	0.75	0.72	0.86
	Obesity (%)	26 (37.7)	7 (28)	2 (33.3)	0.40	0.39	0.83	0.79
Drug (%)	ACEI/ARB's	19 (27.5)	5 (20)	2 (33.3)	0.60	0.46	0.88	0.76
	Duration of symptoms (median) days	7 (5–11)	5.5 (4–7)	3 (1–5)	0.09	0.51	0.03	0.09
Clinical features	Fever (%)	52 (75.4)	19 (76)	5 (83.3)	0.83	0.94	0.66	0.70
	Cough (%)	52 (75.4)	16 (64)	1 (16.7)	0.04	0.28	0.002	0.04
	Dyspnea (%)	33 (47.8)	12 (48)	2 (33.3)	0.80	0.99	0.48	0.53
	Diarrhea (%)	16 (23.2)	5 (20)	1 (16.7)	0.67	0.75	0.72	0.86
	Fatigue (%)	8 (11.6)	4 (16)	1 (16.7)	0.53	0.57	0.72	0.97
	Myalgia/aches (%)	17 (24.6)	5 (20)	1 (16.7)	0.56	0.64	0.66	0.86
	Loss of taste/ smell (%)	10 (14.4)	3 (12)	0 (0)	0.51	0.76	0.31	0.38
	SpO2% at RA (median)	95(92–97)	94(91–97)	93 (90-96)	0.24	0.18	0.88	0.61
	Heart rate	94(79-105)	94(83–103)	96 (85-105)	0.75	0.66	0.81	0.60
	Respiratory rate	18 (16–24)	18 (16–26)	19 (16–29)	0.85	0.79	0.87	0.86
	SBP	130 (115.8–141.3)	130 (108.5–156.5)	134 (115.3–159)	0.89	1.0	0.58	0.58
	DBP	80 (68-90)	82 (70-94)	86 (70-94)	0.18	0.78	0.04	0.57
Investigations	PaO2:FiO2	332.9 (275.2–401)	324.6 (277.1-373.4)	324.1 (278–407)	0.47	0.65	0.48	0.69
	CURB65	1 (0–2)	1 (0–2)	2 (0.4-3.0)	0.04	0.07	0.26	0.92
	eGFR (ml/min/m^2)	84.6(59.2-92)	79.1(54.3-89)	75.5(36.7-88)	0.59	0.96	0.29	0.34
	eGFR <60 ml/min/m^2	18 (26.1)	7 (28)	3 (50)	0.52	0.81	0.21	0.31
	Lymphocytes,10^9 cells/l	1.1 (0.76–1.43)	0.87 (0.63–1.32)	0.83 (0.48–1.22)	0.02	0.03	0.03	0.76
	C-reactive protein mg/dl	5.4 (2.2–11.3)	7.3(2.8–14.1)	7.9(2.3–15.3)	0.08	0.06	0.18	0.67
	Procalcitonin, ng/mL	0.12 (0.05–0.2)	0.15 (0.06–0.22)	0.23 (0.08–0.37)	0.18	0.78	0.09	0.31
	Ferritin, mg/l	624 (290–1180)	966 (478–1450)	996.5 (553–2935)	0.002	0.003	0.001	0.76
	LDH, U/l	265 (210–350)	276 (238–373)	269 (240–415)	0.20	0.22	0.69	0.97
	D-Dimer, mg/mL	0.57 (0.36–1.25)	0.78 (0.49–1.60)	0.89 (0.66–2.89)	0.044	0.06	0.03	0.34
	Trop T, ng/L	9.3 (6.1–21)	12.1(8–23.3)	28(16–52)	0.02	0.19	0.01	0.29
	Opacities >50% of lung surface on X-ray, %	16 (23.2)	5 (20)	1 (16.7)	0.67	0.75	0.72	0.86

p^1- Normokalemia vs hypokalemia; p^2- Normokalemia vs mild hypokalemia; p^3- Normokalemia vs severe hypokalemia; p^4- mild hypokalemia vs severe hypokalemia

Table 2: Association of serum potassium with clinicaloutomes

	Normokalemia (n=69)	Mild hypokalemia (n=25)	Severe hypokalemia (n=6)	p [1]	p [2]	p [3]	p [4]
Length of hospital stay (days)	8 (7-14)	14 (9-18)	17 (12-24)	0.001	0.001	0.001	0.42
ICU admission	8 (11.6)	9 (36)	3 (50)	0.002	0.007	0.01	0.54
Length of ICU stay	9 (8-14)	14 (12-18)	19 (15-24)	0.02	0.04	0.02	0.49
Invasive mechanical ventilation	5 (7.2)	8 (32)	3 (50)	0.001	0.002	0.001	0.42
Mortality	8 (11.6)	4 (16)	2 (33.3)	0.30	0.57	0.14	0.34

Table 3: Multivariate logistic regression for risk of mechanical ventilation

Risk factor		Number of patients	Patients on IMV	Odds Ratio	95% CI	P value
Age	<65 years	68	9 (13.2)	3.23	1.23-5.39	0.08
	>65 years	32	7 (21.8)			
Gender	Male	53	8 (15.1)	0.76	0.08-1.67	0.78
	Female	47	8 (17.0)			
Hypertension		51	7 (13.2)	0.97	0.36-1.78	0.37
Diabetes		28	3 (10.7)	1.33	0.56-2.34	0.16
CVD		22	2 (9.1)	2.3	0.6-9.9	0.25
Obesity		35	3 (8.6)	0.91	0.16-1.87	0.35
ACEI/ARB's		26	2 (7.7)	0.87	0.07-3.39	0.45
Duration of symptom	<3 days	23	3 (13.0)	2.79	0.78-6.67	0.06
	>3 days	77	13 (18.6)			
Fever		76	12 (15.8)	1.19	(1.0-2.16)	0.51
Cough		69	13 (18.8)	2.13	(1.3-3.9)	0.07
Dyspnea		47	8 (17.0)	1.76	1.08-3.67	0.78
Diarrhea		22	2 (9.1)	1.21	1-3.8	0.66
Fatigue		13	1 (0.9)	0.35	0.04-1.5	0.910
Myalgia		23	2 (8.7)	1.19	(0.0-1.16)	0.51
Loss of taste and smell		13	1 (0.9)	0.35	0.04-1.5	0.910
SpO2% at RA <95%		5	1 (20)	3.56	0.09-5.44	0.07
Heart rate <60 or >100		12	1 (8.3)	0.22	(0.0-1.36)	0.50
Respiratory rate >20		12	1 (8.3)	0.22	(0.0-1.3)	0.50
SBP >140 mmHg		15	2 (13.3)	2.79	0.78-6.67	0.06
DBP >90 mmHg		11	1 (9.1)	1.21	1-3.8	0.66
Hypokalemia (<3.5 mmol/L)		31	11 (35.5)	6.74	1.39-16.85	0.001
PaO2:FiO2 <300		6	1 (16.7)	1.79	0.89-2.90	0.45
CURB65 >1		7	1 (14.3)	1.35	0.66-2.34	0.56
eGFR <60 ml/min/m^2		28	2 (7.1)	1.01	0.34-1.89	0.67
Lymphocytes <0.79x10^9 cells/l		21	2 (9.5)	1.33	0.8-3.93	0.36
C-reactive protein >10mg/dl		28	2 (7.1)	1.01	0.34-1.89	0.67
Procalcitonin >0.2 ng/mL		17	1 (5.9)	0.89	0.09-1.59	0.76
Ferritin >300mg/l		89	11 (12.4)	1.21	0.56-1.88	0.54
LDH >367U/l		27	2 (7.4)	1.11	0.3-1.85	0.66
D-Dimer >0.5 mg/mL		63	9 (14.3)	1.25	(0.9-2.16)	0.58
Trop T >10ng/L		18	2 (11.1)	1.17	0.77-3.37	0.45
Opacities >50% of lung on X-ray		22	4 (18.2)	2.01	1.01-3.01	0.32

In present study, hypokalemia was observed to be an independent predictor of invasive mechanical ventilation (OR- 6.74; 95% CI- 1.39-16.85; p<0.01). However, all other variables (as shown in Table 3) were not associated with requirement of invasive mechanical ventilation (p>0.05).

4. Discussions

Electrolyte imbalance especially hypokalemia is often observed in COVID 19 patients.[2] Our study documented higher prevalence of hypokalemia i.e. 31%. Of them, 25% had mild and 6% had severe hypokalemia. Hypokalemia and electrolyte imbalance have been attributed to increased excretion potassium due to effect of SARS CoV virus on ACE 2 enzyme, thus affecting renin–angiotensin system (RAS).[2,3] The findings of present study were supported by findings of O'Monera et al in which about one third patients had hypokalemia.[7] Similarly, lower potassium levels was significantly associated with COVID 19 infection in a study by Lippi et al.[6] However, Chen et al documented higher prevalence of hypokalemia i.e. 54% in younger patients as compared to present study.[8] The observed difference in prevalence of hypokalemia between present study and reference study could be attributed to genetic factors and quantitative expression of ACE 2 in various tissues among individuals of different race. Thus susceptibility or response to SARS CoV-2 vary among different populations.[9,10]

Our study aimed to assess the association of serum potassium levels with clinical outcomes. We documented a statistically significant association of hypokalemia with shorter duration of onset of symptoms (p<0.05). Also, hypokalemia was associated with increased diastolic blood pressure i.e. lower the serum potassium levels, higher the diastolic blood pressure (p<0.05). The degree of hypokalemia was associated with raised serum ferritin, D-dimer and Trop T levels highlighting severe disease with increasing level of hypokalemia (p<0.05). These findings were similar to findings of O'Monera et al.[7] They also documented statistically significant association of hypokalemia with short duration of hospital stay, lower lymphocyte levels, and markers of inflammation (p<0.05).[7] However, we documented significantly lower prevalence of cough among patients with hypokalemia. This could be explained by activity of SARS CoV-2 on ACE receptor. Coronavirus, through its action on ACE2 promote ACE2 depletion leading to an imbalance between RAS and ACE2/angiotensin axis. These changes lead to elevation of angiotensin II levels, promoting vasoconstriction and proinflammatory–profibrotic activity contributing to increased reabsorption of sodium and water, and hence increasing potassium excretion.[11,12] Increased ACE 2 activity leads to synthesis of bradykinin which contribute to dry cough.[13]

Among various clinical outcomes, hypokalemia was significantly associated with longer duration of hospital stay, rate of ICU admission, longer duration of ICU admission, and higher requirement of mechanical ventilation (p<0.05). Similar findings were recorded by O'Monera et al.[7] In another study by Huang et al, contrasting findings were observed i.e. they documented higher potassium level with higher rate of ICU admission highlighting the association of hyperkalemia with severity of illness.[14] Chen et al, on the other hand observed statistically significant association of hyperkalemia with mortality (p<0.05).[8] Thongprayoon et al documented that both hypokalemia as well as hyperkalemia is associated with higher risk of mortality regardless of diagnosis when compared to normokalemia (p<0.05) and show a U shaped curve i.e. mortality risk increased in accordance with the severity of the hypokalemia or hyperkalemia.[15]

Bielecka-Dabrowa et al observed that hypokalemia may contribute to development of myocardial dysfunction, ventricular arrhythmia and respiratory muscle dysfunction.[16] Though, the impact of hypokalemia among COVID 19 patients on myocardial as well as respiratory function remains unclear, literature suggest that Coronavirus infect heart as evidenced from raised Trop I in present study. As hypokalemia in coronavirus infection may be associated with myocardial and respiratory dysfunction, higher risk of requirement of invasive mechanical ventilation in such patients may be explained.

Our study had certain limitation. This was observational study and impact of treatment and correction of hypokalemia was not studies in this study.

5. Conclusions

Hypokalemia is common in COVID 19 patients among Indian population. Hypokalemia is associated with severe illness and significantly affect clinical outcome. Hypokalemia is an independent predictor of risk of mechanical ventilation after adjusting for confounding variables.

6. Conflicts of Interest

No potential conflict of interest relevant to this article was reported.

7. Source of Funding

None.

References

1. Lu H, Stratton CW, Tang YW. Outbreak of pneumonia of unknown etiology in Wuhan, China: the mystery and the miracle. *J Med Virol*. 2020;92(4):401–2. doi:10.1002/jmv.25678.
2. Hamming I, Timens W, Bulthuis ML, Lely AT, Navis G, Van Goor H, et al. Tissue distribution of ACE2 protein, the functional receptor for SARS coronavirus. A first step in understanding SARS pathogenesis. *J Pathol*. 2004;203(2):631–7. doi:10.1002/path.1570.

3. Santos RA, Ferreira AJ, Simoes E. Recent advances in the angiotensin-converting enzyme 2-angiotensin(1-7)-Mas axis. *Exp Physiol*. 2008;93(5):519–27. doi:10.1113/expphysiol.2008.042002.
4. Weir MR, Rolfe M. Potassium homeostasis and renin-angiotensin-aldosterone system inhibitors. *Clin J Am Soc Nephrol*. 2010;5(3):531–48. doi:10.2215/CJN.07821109.
5. Wang D, Hu B, Hu C, Zhu F, Liu X, Zhang J, et al. Clinical Characteristics of 138 Hospitalized Patients With 2019 Novel Coronavirus-Infected Pneumonia in Wuhan, China. *JAMA*. 2020;323(11):1061–9. doi:10.1001/jama.2020.1585.
6. Lippi G, South AM, Henry BM. Electrolyte imbalances in patients with severe coronavirus disease 2019 (COVID-19). *Ann Clin Biochem*. 2020;57(3):262–5. doi:10.1177/0004563220922255.
7. Moreno PO, Leon-Ramirez JM, Fuertes-Kenneally L, Perdiguero M, Andres M, Garcia-Navarro M, et al. 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*. 2020;100:449–54. doi:10.1016/j.ijid.2020.09.033.
8. Chen T, Wu DI, Chen H, Yan W, Yang D, Chen G, et al. Clinical characteristics of 113 deceased patients with coronavirus disease 2019: retrospective study. *BMJ*. 2020;368:m1091. doi:10.1136/bmj.m1091.
9. Cao Y, Xiao Y, Wang N, Wang G, Zhou X, Huang L, et al. CD19/CD22 Chimeric Antigen Receptor T Cell Cocktail Therapy following Autologous Transplantation in Patients with Relapsed/Refractory Aggressive B Cell Lymphomas. *Transplant Cell Ther*. 2020;136(1). doi:10.1182/blood-2020-140308.
10. Devaux CA, Rolain JM, Raoult D. ACE2 receptor polymorphism: Susceptibility to SARS-CoV-2, hypertension, multi-organ failure, and COVID-19 disease outcome. *J Microbiol Immunol Infect*. 2020;53(3):425–35. doi:10.1016/j.jmii.2020.04.015.
11. Weir MR, Rolfe M. Potassium homeostasis and renin-angiotensin-aldosterone system inhibitors. *Clin J Am Soc Nephrol*. 2010;5(3):531–48. doi:10.2215/CJN.07821109.
12. Hoffmann M, Kleine-Weber H, Schroeder S, Krüger N, Herrler T, Erichsen S, et al. SARS-CoV-2 Cell Entry Depends on ACE2 and TMPRSS2 and Is Blocked by a Clinically Proven Protease Inhibitor. *Cell*. 2020;181(2):271–80. doi:10.1016/j.cell.2020.02.052.
13. Nicolau LA, Magalhães PJ, Vale ML. What would Sérgio Ferreira say to your physician in this war against COVID-19: How about kallikrein/kinin system. *Med Hypotheses*. 2020;143:109886. doi:10.1016/j.mehy.2020.109886.
14. 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. doi:10.1016/S0140-6736(20)30183-5.
15. Thongprayoon C, Cheungpasitporn W, Hansrivijit P, Mao MA, Medaura J, Bathini T, et al. Admission serum potassium levels in hospitalized patients and one-year mortality. *Medicines*. 2020;7(1):2. doi:10.3390/medicines7010002.
16. Bielecka-Dabrowa A, Mikhailidis DP, Jones L, Rysz J, Aronow WS, Banach M, et al. The meaning of hypokalemia in heart failure. *Int J Cardiol*. 2012;158(1):12–7.

Author biography

Arun Suresh, Senior Resident

Vishnu Vijay, Junior Resident

Ruchi Tandon, Associate Professor

Cite this article: Suresh A, Vijay V, Tandon R. Association of serum potassium levels with severity of COVID infection and requirement of invasive mechanical ventilation in COVID 19 patients. *Panacea J Med Sci* 2022;12(2):355-360.