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Hypokalemia and Clinical Implications in Patients with Coronavirus Disease 2019 (COVID-19)

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Abstract

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Abstract

BACKGROUND: SARS-CoV-2 has caused a series of COVID-19 globally. SARS-CoV-2 binds angiotensin I converting enzyme 2 (ACE2) of renin-angiotensin system (RAS) and causes prevalent hypokalemia

METHODS: The patients with COVID-19 were classified into severe hypokalemia, hypokalemia, and normokalemia group. The study aimed to determine the relationship between hypokalemia and clinical features, the underlying causes and clinical implications of hypokalemia.

RESULTS: By Feb 15, 2020, 175 patients with COVID-19 (92 women and 83 men; median age, 46 [IQR, 34-54] years) were admitted to hospital in Wenzhou, China, consisting 39 severe hypokalemia-, 69 hypokalemia-, and 67 normokalemia patients. Gastrointestinal symptoms were not associated with hypokalemia among 108 hypokalemia patients ($P>0.05$). Body temperature, CK, CK-MB, LDH, and CRP were significantly associated with the severity of hypokalemia ($P<0.01$). 93% of severe and critically ill patients had hypokalemia which was most common among elevated CK, CK-MB, LDH, and CRP. Urine K⁺ loss was the primary cause of hypokalemia. severe hypokalemia patients was given 3 g/day, adding up to an average of 34 (SD=4) g potassium during hospital stay. The exciting finding was that patients responded well to K⁺ supplements when they were inclined to recovery.

CONCLUSIONS: Hypokalemia is prevailing in patients with COVID-19. The correction of hypokalemia is challenging because of continuous renal K⁺ loss resulting from the degradation of ACE2. The end of urine K⁺ loss indicates a good prognosis and may be a reliable, in-time, and

COVID-19 SARS-CoV-2 preprints from medRxiv and bioRxiv

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sensitive biomarker directly reflecting the end of adverse effect on RAS system.

Competing Interest Statement

The authors have declared no competing interest.

Funding Statement

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Author Declarations

All relevant ethical guidelines have been followed; any necessary IRB and/or ethics committee approvals have been obtained and details of the IRB/oversight body are included in the manuscript.

Yes

All necessary patient/participant consent has been obtained and the appropriate institutional forms have been archived.

Yes

I understand that all clinical trials and any other prospective interventional studies must be registered with an ICMJE-approved registry, such as ClinicalTrials.gov. I confirm that any such study reported in the manuscript has been registered and the trial registration ID is provided (note: if posting a prospective study registered retrospectively, please provide a statement in the trial ID field explaining why the study was not registered in advance).

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I have followed all appropriate research reporting guidelines and uploaded the relevant EQUATOR Network research reporting checklist(s) and other pertinent material as supplementary files, if applicable.

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Hypokalemia is caused by SARS-CoV-2 virus due to its affinity for the Angiotensin Converting Enzyme (ACE) receptor that is present in the lungs, heart, blood vessels and the gastrointestinal tract of humans. It has been suggested from animal experiments that medications inhibiting this receptor (called ACEI or ARBs) could be a potential management strategy(1-2). Because ACEI and ARBs are medications mainly use for high blood pressure and would lower the BP, it is recommended that these medications should at least be used in patients with COVID-19 who are already suffering from hypertension or whose BP is not lower than 100 mm Hg systolic.

It would also be interesting to know the recovery and death rate of COVID-19 patients with hypertension or heart failure who were already using an ACEI or ARB medications compared with those who were not on suchmedications.

Abbreviations: ACEI= Angiotensin Converting Enzyme Inhibitors, ARBs= Angiotensin Receptor Inhibitors, BP= Blood pressure

References

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2. Dimitrov, D. S. The secret life of ACE2 as a receptor for the SARS virus. Cell, 2003; 115(6), 652–653.

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