

## Familial Hypokalemic Periodic Paralysis Attack Following SARS-Cov-2 Infection: A Case Report

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### ABSTRACT

Familial hypokalemic periodic paralysis is a rare disorder that manifests with the sudden onset of flaccid paralysis that is triggered by low levels of blood potassium, which can be caused by various factors such as, rest after intense exercise, or high-carbohydrate foods. This report presents cases of hypokalemic periodic paralysis attack triggered by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection. A 29-year-old male patient was admitted with quadripareisis, fever, shortness of breath, and severe hypokalemia. The patient had a history of three episodes of Familial hypokalemic periodic paralysis (HPP). Diagnostic tests, such as chest computed tomography scan and polymerase chain reaction test, confirmed SARS-CoV-2 infection. The patient was treated with potassium chloride infusion, spironolactone, and remdesivir, and was eventually discharged from the hospital. In conclusion, SARS-CoV-2 infection can potentially exacerbate HPP and should be considered a risk factor for its occurrence.

### Introduction

Familial hypokalemic periodic paralysis (Familial HPP) is a monogenic disorder with an autosomal-dominant mode of inheritance with a prevalence of 1 in 100,000 [1]. This disorder manifests with the sudden onset of flaccid paralysis associated with hypokalemia, usually following rest after intense exercise or consuming foods with high carbohydrate content [2]. In this report, we describe a case of a patient who experienced quadripareisis and severe hypokalemia and the simultaneous diagnosis of SARS-CoV-2 infection.

### Case Report

A 29-year-old male patient presented at our hospital's emergency department with complaints of weakness affecting his upper and lower extremities bilaterally. The patient also reported experiencing shortness of breath, fever, and cough which had persisted for one day.

The individual has reported experiencing a sense of bodily weakness since the previous night, but this sensation has become more pronounced this morning, to the extent that they are no longer able to move any of their limbs. It should be noted that the weakness initially manifested in the lower limbs before rapidly spreading to the upper limbs. The patient has been previously hospitalized on three separate occasions due to comparable symptoms, with the most recent hospitalization having taken place two years ago.

The authors declare no conflicts of interest.

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Additionally, the patient has noted that their sister has experienced similar symptoms in the past.

The patient did not report any previous instances of nausea, vomiting, diarrhea, chest pain, loss of consciousness, abnormal body movements, or bowel or bladder complications. Additionally, the patient had no other health conditions such as thyroid dysfunction. The patient also did not mention taking medication.

Upon arrival at the emergency room, the patient was found to be conscious, alert, and oriented to time, place, and person. The vital signs recorded were a pulse rate of 78 beats per minute, a blood pressure of 130/70 mmHg, a respiratory rate of 22 breaths per minute, and a SpO<sub>2</sub> of 98% while on room air. Following a physical examination, there were no notable findings except for flaccid paralysis of both upper and lower limbs, hypotonia, and grade 1 power. Notably, there was no sensory involvement in any of the patient's limbs, and deep tendon reflexes were absent. Furthermore, there were no cerebellar or meningeal signs, and the abdominal, respiratory, and cardiovascular examinations were unremarkable.

In response to the patient's condition, further laboratory tests were conducted, which revealed severe hypokalemia with a serum K<sup>+</sup> level of 1.61 mmol/L (Table 1). The initial laboratory investigations are displayed in (Table 2), which confirmed the diagnosis of hypokalemia. Electrocardiograph (ECG) records also showed features of hypokalemia.

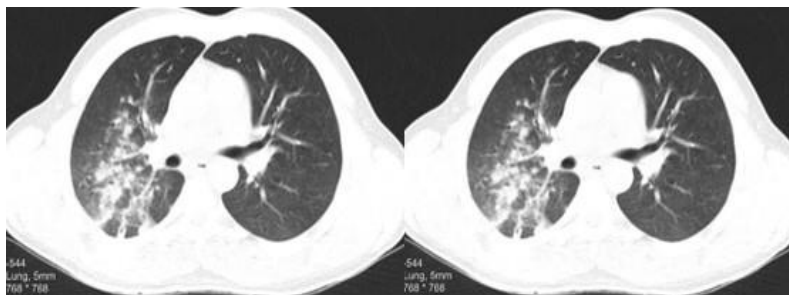
After hypokalemia was diagnosed, 15% potassium chloride was administered at a rate of 10 milliequivalents per hour (mEq/h) through an IV line. After four hours, a central venous line was inserted in the right femoral side and 15% potassium chloride was administered at a rate of 30 mEq/h. Moreover, the patient was prescribed spironolactone tablets at a dose of 25 mg every 12 hours. After receiving about 310 mEq of potassium, the serum level of potassium was within the normal range, and the patient's muscle paralysis was also resolved.

In the part of CT examinations, the patients had pulmonary ground-glass opacities, that was shown in (Figure 1). A SARS-CoV-2 PCR test was also performed, and due to the positive test, the patient was treated with remdesivir. After 5 days of hospitalization in the ICU, the patient was transferred to the internal ward with a good general condition.

Time after admission	Potassium level (mEq/L)
0	1.61
3 <sup>th</sup> hour	1.85
5 <sup>th</sup> hour	2.16
9 <sup>th</sup> hour	2.09
13 <sup>th</sup> hour	4.79
15 <sup>th</sup> hour	5.07
1 <sup>th</sup> day	3.93
3 <sup>th</sup> day	4.05
5 <sup>th</sup> day	4.16

**Table 2- Laboratory testing upon admission**

Test	Report	Reference range
Hemoglobin (gr/dl)	16.2	13.5-18
WBC ( $\times 10^3$ / $\mu$ l)	11.8	4-10.5
Platelet ( $\times 10^3$ / $\mu$ l)	170	150-400
Neutrophil %	95.5%	54-62
Lymphocyte %	3.2%	25-33
CRP (mg/L)	5.3	0-8
ESR (mm/h)	10	0-15
Lactate (mg/dl)	20	5.5-14.5
CPK (U/L)	1344	Up to 195
BS (mg/dl)	103	<200
TSH (Miu/L)	1	0.35-4.94
T3 (ng/ml)	1.1	0.52-1.85
T4 ( $\mu$ g/dl)	4.9	4.87-11.72
iPTH (pg/ml)	12.3	8.5-72.5
Aldolase (Iu/l)	9.8	Less than 7.6
AST(U/L)	24	Up to 38
ALT (U/L)	46	Up to 41
ALP (U/L)	183	100-320
Phosphor (mg/dl)	3.5	2.6-4.5
Creatinine (mg/dl)	0.9	0.6-1.4
Urea (mg/dl)	39	19-44
Calcium (mg/dl)	9.3	8.6-10.3
Magnesium (mg/dl)	2.6	1.8-2.6
Na (mEq/L)	142	135-150
HCO <sub>3</sub> (mmol/l)	20.2	22-26
PCO <sub>2</sub> (mmHg)	41.1	35-45
ABG PO <sub>2</sub> (mmHg)	69	80-100
SaO <sub>2</sub> %	91.6	-
pH	7.3	7.35-7.45



**Table 1- Serum potassium level (mEq/L) in different times after hospital admission**

**Figure 1- Lung computed tomography showing opacity****Discussion**

Is a rare condition that causes muscle weakness, which can be either localized or widespread? The disorder is triggered by low levels of blood potassium, which can be caused by various factors such as surgery, alcohol, steroids, insulin, pregnancy, rest after intense exercise, or high-carbohydrate foods [2-3]. In HPP, hypokalemia causes depolarization of muscle cells, which causes inactivation of sodium channels and decrease in excitability of fibers and finally muscle paralysis [4].

To our knowledge, there has only been one reported case of hypokalemic periodic paralysis occurring after a SARS-COV-2 infection. In 2021, V Rajesh et al. documented a 34-year-old man who experienced an attack of hypokalemic periodic paralysis after contracting SARS-COV-2 [5]. The patient in the study had hypokalemia and paralysis that were believed to be a result of a combination of factors, including SARS-COV-2 infection, inhaled salbutamol, and a high-carbohydrate diet. However, our case reveals that SARS-COV-2 infection alone was responsible for the hypokalemia as no other contributing factors were found.

It has been observed that 24.3% of SARS-COV-2 patients experience hypokalemia, which is associated with a poor prognosis and outcome. The exact cause of hypokalemia in SARS-COV-2 infection is not yet clear.

**Conclusion**

This report is the second case of a familial hypokalemic periodic paralysis attack after SARS-CoV-2 infection. Considering the mechanism of hypokalemia caused by SARS-CoV-2 infection, it is reasonable to use an aldosterone receptor inhibitor, including spironolactone, in regard to the prevention and treatment of hypokalemic periodic paralysis attacks following SARS-CoV-2 infection.

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