

## Case Report

# Hypokalemic periodic paralysis: a case report

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## ABSTRACT

Hypokalemic periodic paralysis (HPP), a clinical syndrome characterized by low serum potassium, is a rare but treatable cause of acute muscular weakness. The etiology can be attributed to various factors and can be either idiopathic or due to secondary causes. The approach to a case of HPP involves identifying the underlying etiology and prompt replenishment and maintenance of the potassium levels. Further management depends on the cause, frequency of attacks, severity of symptoms and the duration of the illness.

**Keywords:** Familial, Hypokalaemia, Periodic paralysis Quadripareisis

## INTRODUCTION

Plasma potassium is normally kept at 3.5 to 5.0 mEq per liter by multiple mechanisms. Levels outside this range are associated with an increasing rate of death from multiple causes.<sup>1</sup> Hypokalemia is defined as a deficiency of potassium below 3.5 mEq/L<sup>2</sup> in the plasma and increased gastrointestinal and renal losses are the common culprits.<sup>3</sup> Homeostasis of this cation is tightly regulated and achieved mainly via alteration in renal excretion.<sup>4,5</sup>

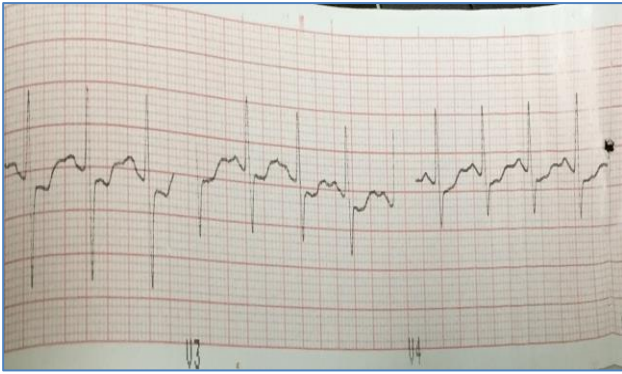
This can be impaired due several factors which can lead to symptoms ranging from muscle cramps to cardiac arrest.<sup>2</sup> Hypokalemic periodic paralysis is a relatively uncommon yet potentially life-threatening condition that, if correctly diagnosed and treated, can be completely reversed.

In an acute setting, it presents as muscular weakness and low serum potassium. Here we report a case of hypokalemic paralysis in a young male predominantly presenting as acute quadripareisis.

## CASE REPORT

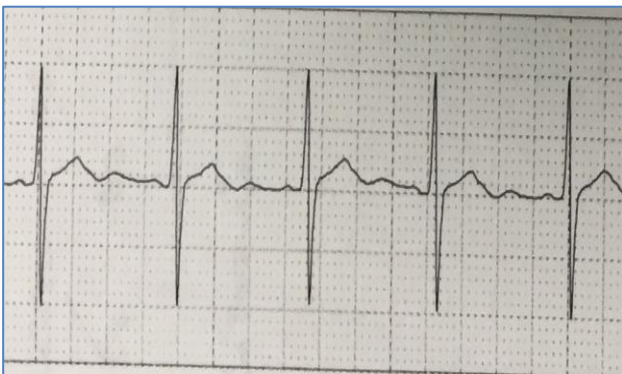
A 34-year-old male presented to the clinic with bilateral, cramp-like leg pain for 5 days. He was given analgesics and sent home. The patient went to bed at around 10 pm, with no complaints and woke up an hour later unable to move both upper and lower limbs. Neck and head movements were possible, swallowing and speech were intact. On neurological examination, power in all four limbs was 2/5, cranial nerves were intact, reflexes were normal and there was no sensory loss.

There was no preceding history of fever, diarrhea, vomiting, trauma, heavy carbohydrate meal, prior strenuous exercise or thyroid disease. Lab investigations showed a potassium level of 2 mEq/L and creatinine of 1.3 mg/dL. The ECG done at the time showed ST segment changes and T wave flattening (Figure 1). Blood pH was 7.64, pCO<sub>2</sub> was 16 mmHg. Oral potassium chloride was started and he regained power within 12 hours. He was discharged on oral potassium supplementation and acetazolamide.



**Figure 1: ST changes and T wave flattening.**

He had heaviness and pain in both legs for almost a year despite being on potassium supplements. Serum potassium was maintained at 3.6 mEq/L and other biochemical tests were normal. Blood sugar levels, Thyroid profile and nerve conduction studies were normal. ECG at this time is showed U waves (Figure.2). Acetazolamide and oral potassium citrate were continued and he was advised a high potassium and low carbohydrate diet. His symptoms gradually resolved thereafter.



**Figure 2: Lead V2 showing U waves.**

## DISCUSSION

Weakness is a common, yet nonspecific presentation of various neurological and non-neurological conditions. While hypokalemic paralysis is an important cause of acute flaccid paralysis, there are many clinical differentials like Guillain Barré syndrome, acute transverse myelitis, polymyositis, poliomyelitis, and porphyria; that should be considered. Of the differentials, the immediate life-threatening causes like stroke and space occupying lesions(SOLs) should be ruled out at the earliest. Most cases of periodic paralysis are familial or primary hypokalemic periodic paralysis. We believe this was the cause in our patient after having ruled out other possible etiologies for the low serum potassium (secondary periodic paralysis). Sporadic cases are associated with numerous other conditions including barium poisoning, hyperthyroidism, renal disorders, certain endocrinopathies and gastrointestinal potassium

losses. Hypokalemic paralysis presents as acute flaccid weakness with hypokalemia (serum potassium <3.5 mmol/l), without sensory signs, facial, bulbar, autonomic, bladder and bowel involvement, normal creatine kinase, and NCV.

Hypokalemic familial periodic paralysis is believed to be due to an increase in muscle membrane sodium permeability.<sup>6,7</sup> There are a multitude of factors that can trigger weakness or paralysis including acute stress, pain, anesthesia, surgery, alcohol, strenuous exercise, steroids etc.<sup>8,9</sup> Attacks are typically precipitated by rest or sleep as was in our patient. Patients remain alert during the attacks. Patients often report additional symptoms either before, during, or after attacks. These include paresthesias, sweating, myalgia, extreme fatigue, thirst, shortness of breath (either due to anxiety or to the episode itself), palpitations, clumsiness, irritability, and mental dullness.<sup>10</sup> Weakness is a common, yet nonspecific presentation of various neurological and non-neurological conditions.

While hypokalemic paralysis is an important cause of acute flaccid paralysis, there are many clinical differentials like Guillain Barré syndrome, acute transverse myelitis, polymyositis, poliomyelitis, and porphyria; that should be considered. Of the differentials, the immediate life-threatening causes like stroke and space occupying lesions(SOLs) should be ruled out at the earliest. Most cases of periodic paralysis are familial or primary hypokalemic periodic paralysis. We believe this was the cause in our patient after having ruled out other possible etiologies for the low serum potassium (secondary periodic paralysis). Sporadic cases are associated with numerous other conditions including barium poisoning, hyperthyroidism, renal disorders, certain endocrinopathies and gastrointestinal potassium losses. Hypokalemic paralysis presents as acute flaccid weakness with hypokalemia (serum potassium <3.5 mmol/l), without sensory signs, facial, bulbar, autonomic, bladder and bowel involvement, normal creatine kinase, and NCV.

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ECG, TSH, free T3 and T4 are the minimum indicated laboratory investigations, with renal and adrenal function also recommended. It is important to note that this

disorder is autosomal dominant in two-thirds of cases; with male preponderance when providing genetic counselling.<sup>11</sup> The basic guidelines to follow when caring for the patient include control of plasma potassium, avoidance of large glucose and salt loads (which promote intracellular shift), maintenance of body temperature, acid–base balance, and cautious use of neuromuscular blocking agents.<sup>12,13</sup> The specific treatment of hypokalemic FPP is oral potassium supplementation, repeated at 15-30 minute intervals depending on the response of the ECG, serum potassium level, and muscle strength. Replenishment may be done intravenously if the patient is vomiting or unable to swallow. Prophylaxis against recurrent periodic attacks has been successful with a wide variety of treatment modalities including spironolactone and acetazolamide.

## CONCLUSION

Periodic paralysis is important to consider when seeing a patient with sudden onset weakness or paralysis, especially those with no history or evidence of other diseases and no significant risk factors for stroke. Failure to properly diagnose and treat Periodic Paralysis can be fatal, and rapidly correcting the potassium can resolve symptoms completely. When possible, the underlying cause must be adequately addressed to prevent the persistence or recurrence of paralysis.

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