Acta Neurologica Indonesia 2026; 04(01)

Acta Neurologica Indonesia
Department of Neurology, Faculty of Medicine Universitas Indonesia
ISSN 3032-6303

Case Report

HYPOKALEMIC PERIODIC PARALYSIS TRIGGERED BY INSULIN THERAPY IN PATIENT WITH WELL-CONTROLLED TYPE 2 DIABETES MELLITUS: A CASE REPORT

Prenal L.M Ronsumbre[1], Helmi Aryosyaputra[1], Lucas Julisar Selawa[1], Edwin Pranata Laban[2]*

[1] Faculty of Medicine, Duta Wacana Christian University -Yogyakarta, Indonesia; email: helmiaryosyaputra@gmail.com
[2] Neurology Department Bethesda Hospital, Yogyakarta

ABSTRACT

Introduction: Hypokalemic Periodic Paralysis (HPP) is a rare neuromuscular disorder characterized by acute, reversible muscle weakness associated with low serum potassium levels. It may occur due to genetic abnormalities or secondary causes, including insulin therapy, which remains an underrecognized trigger.

Case Summary: A 71-year-old man with well-controlled type 2 diabetes mellitus and hypertension presented with sudden bilateral lower limb weakness. Physical examination revealed flaccid paraparesis without sensory deficits. Laboratory findings showed hyperglycemia (320 mg/dL) and hypokalemia (K^+ 2.96 mmol/L). The patient was on regular subcutaneous insulin therapy with no prior similar episodes. Muscle strength improved within 24 hours after oral potassium supplementation and metabolic correction.

Discussion: Insulin promotes intracellular potassium shift through activation of the Na^+/K^+-ATPase, leading to acute hypokalemia and impaired skeletal muscle excitability. The absence of sensory deficits and rapid clinical improvement following potassium supplementation supported the diagnosis of secondary HPP. In elderly patients with multiple comorbidities, this presentation may mimic neurological disorders, making electrolyte evaluation crucial.

Conclusion: HPP should be considered in diabetic patients receiving insulin who present with acute muscle weakness. Early recognition and prompt correction of hypokalemia are essential for rapid recovery and prevention of serious complications.

Keywords: Diabetes mellitus; Hypokalemic; Insulin therapy; Paralysis

INTRODUCTION

Hypokalemic Periodic Paralysis (HPP) is a rare neuromuscular disorder characterized by transient episodes of acute muscle weakness associated with decreased serum potassium levels. It is classified as a channelopathy caused by dysfunction of ion channels, particularly calcium and sodium channels in skeletal muscle, and typically presents as symmetric flaccid weakness predominantly involving proximal muscles while consciousness and sensory function remain preserved.[5,9]

Most cases of HPP are reported as primary or familial forms with onset during adolescence or early adulthood. However, secondary HPP may occur in elderly individuals, especially those with metabolic, endocrine, or renal disorders and multiple comorbidities. Insulin

therapy represents a rare but underrecognized trigger, as it increases Na^+/K^+-ATPase activity and promotes intracellular potassium shift, resulting in acute hypokalemia and muscle weakness or paralysis.[4,10,12]

To date, reports of HPP in older patients remain limited, particularly in those with comorbid conditions such as diabetes mellitus, hypertension, ischemic stroke, and renal impairment. Current literature predominantly emphasizes genetic aspects and younger populations, highlighting a gap in knowledge regarding clinical variability, diagnostic strategies, and differential diagnosis of HPP in geriatric patients.[9]

This case report aims to describe the clinical manifestations, diagnostic findings, and management of Hypokalemic Periodic Paralysis in an elderly patient with metabolic and neurological comorbidities. The novelty of this report lies in emphasizing the importance of electrolyte evaluation as part of the initial assessment of acute muscle weakness in older adults, in order to prevent misdiagnosis and potentially life-threatening complications.

CASE REPORT

A 71-year-old man presented to the Emergency Department of Bethesda Hospital, Yogyakarta, on June 2, 2025, with a chief complaint of bilateral low back pain accompanied by sudden weakness of both lower limbs since early morning upon awakening. The symptoms caused difficulty in walking and required assistance for mobilization. The pain was severe, with a numerical rating scale score of 8/10, particularly during movement or passive extension of the legs.

The patient was hospitalized for three days in the Galilea ward. There was no history of similar episodes. His past medical history included ischemic stroke with left-sided hemiparesis in 2022, well-controlled hypertension, type 2 diabetes mellitus, cervical and lumbar disc herniation, and a history of urolithiasis in 2024. He had a known allergy to irbesartan. Family history was unremarkable for neuromuscular disorders. Social history revealed heavy smoking since a young age, no alcohol or illicit drug use, and regular light physical activity in the form of morning walks.

On physical examination, the patient was in good general condition and fully conscious (GCS 15). Vital signs were blood pressure 149/88

mmHg, heart rate 57 beats/min, respiratory rate 20 breaths/min, body temperature 36°C, and oxygen saturation 98% on room air. General examination was unremarkable, with no edema and normal peripheral perfusion.

Neurological examination showed no meningeal signs. Cranial nerve function was largely normal, except for rightward tongue deviation without weakness or fasciculation. Motor strength was 5/5 bilaterally in both upper and lower extremities with normal muscle tone. Physiological reflexes were intact, with no pathological reflexes. Sensory, coordination, autonomic, pain provocation, and vertebral examinations were within normal limits.

Laboratory investigations on admission revealed hypokalemia with a serum potassium level of 2.96 mmol/L (reference range 3.5–5.1 mmol/L) and marked hyperglycemia with a random blood glucose level of 320 mg/dL. Serum sodium levels were within normal limits.

Based on clinical history, physical and neurological examinations, and laboratory findings, the patient was diagnosed with hypokalemic periodic paralysis presenting as lower motor neuron–type paraparetic weakness, likely related to ion channel dysfunction. Management included oral potassium chloride supplementation (600 mg), insulin therapy for glycemic control, oral mecobalamin 500 mg as a neuroprotective agent, and patient education regarding dietary modification and physical activity to prevent recurrence.

Table 1. Potassium and GDS results

Examination	Result	Unit	Reference range
Random Blood Glucose (POCT)	320	mg/dL	70-140
Potassium	2,9	mmol/L	3,5-5,1

DISCUSSION

Hypokalemia is defined as a plasma potassium concentration below 3.5 mmol/L and may occur through various mechanisms, including intracellular potassium shift induced by insulin therapy, particularly in patients with type 2 diabetes

mellitus. In patients receiving insulin, stimulation of the Na^+/K^+-ATPase pump leads to redistribution of potassium from the extracellular to the intracellular compartment, resulting in decreased serum potassium levels and an increased risk of severe muscle weakness or paralysis. Although severe hypokalemia is often acute and requires comprehensive evaluation of underlying causes, cases of hypokalemic periodic paralysis in diabetic patients receiving insulin illustrate how this electrolyte disturbance can lead to significant neuromuscular deficits. Therefore, understanding the relationship between insulin therapy, hypokalemia, and the risk of muscle weakness is essential in clinical management to prevent paralysis that may compromise motor function.[11]

Severe hypokalemia can cause flaccid muscle weakness and periodic paralysis in adult patients, particularly when it develops acutely. Insulin administration is one of the main precipitating factors, as it promotes intracellular potassium shift through activation of the Na^+/K^+-ATPase, leading to a marked reduction in serum potassium levels. In patients with type 2 diabetes mellitus, even when blood glucose levels are adequately controlled, therapeutic doses of insulin may precipitate hypokalemia and severe muscle weakness. Understanding this mechanism is crucial to prevent potentially life-threatening paralysis and to ensure timely and appropriate management.[10]

Hypokalemic periodic paralysis (HPP) is a rare neuromuscular disorder characterized by reversible episodes of flaccid muscle weakness associated with decreased serum potassium levels. The primary mechanism underlying HPP involves dysfunction of ion channels in the skeletal muscle cell membrane, particularly calcium channels (CACNA1S) and sodium channels (SCN4A), leading to impaired muscle membrane excitability during hypokalemia.[5,9] In this condition, a reduction in extracellular potassium alters the resting membrane potential, rendering muscle fibers inexcitable and resulting in muscle weakness.

This case demonstrates the manifestation of HPP in an elderly patient with multiple comorbidities, including type 2 diabetes mellitus, hypertension, a history of ischemic stroke, and renal impairment. These findings support the notion that HPP

is not limited to primary or familial forms but may also present as a secondary condition due to metabolic or renal disturbances. In older adults, this condition often poses diagnostic challenges, as its clinical presentation may mimic more common neurological disorders, such as recurrent stroke or metabolic myopathy.[12] The absence of focal neurological deficits, pathological reflexes, or sensory disturbances in this patient supports a diagnosis of HPP rather than a central nervous system etiology.

A history of renal impairment in this patient may also contribute as a cause of secondary hypokalemia, which subsequently triggers muscle weakness or flaccid paralysis, although the mechanism is not always directly neuromuscular in nature. In a case report of infantile nephrotic syndrome, a child with nephrotic syndrome developed severe hypokalemia due to a combination of electrolyte loss, malnutrition, and iatrogenic factors (use of diuretics and corticosteroids), ultimately presenting with symptoms such as head drop and flaccid muscle weakness that rapidly resolved following potassium and magnesium supplementation.[2] This case highlights that severe hypokalemia resulting from renal processes and nutritional status can manifest as acute, reversible neuromuscular symptoms.

The mechanisms underlying this association are diverse. The kidneys play a central role in potassium homeostasis through regulated reabsorption and excretion in the distal tubules. Tubular disorders such as renal tubular acidosis (RTA), particularly distal RTA, can lead to excessive urinary potassium loss, thereby reducing serum potassium levels and precipitating acute muscle weakness.[3] In addition, nephrotic syndrome and diuretic use promote sodium and potassium excretion, while corticosteroid therapy may increase sodium retention and enhance renal potassium excretion via the renal tubules.[2]

Laboratory findings in this patient revealed hypokalemia (2.96 mmol/L) at the onset of symptoms, which served as a key element in establishing the diagnosis. Hypokalemia is known to cause muscle weakness of varying severity, ranging from mild weakness to paralysis, depending on serum potassium levels. At potassium concentrations of 2.5–3.0 mmol/L, proximal muscle weakness typically begins to appear, whereas more

severe complications, such as cardiac arrhythmias, may occur at lower levels. The presence of severe hyperglycemia in this patient may also have acted as a precipitating factor, as increased insulin levels can promote intracellular potassium shift and exacerbate hypokalemia.

Management of hypokalemic periodic paralysis (HPP) focuses on correction of hypokalemia, control of precipitating factors, and prevention of recurrence. In this case, oral potassium supplementation resulted in clinical improvement, further supporting the diagnosis of HPP. Patient education regarding lifestyle modification, dietary regulation with reduced simple carbohydrate intake, and limitation of strenuous physical activity constitutes an essential component of long-term management, particularly in patients with metabolic comorbidities.[7] Potassium supplementation remains the primary intervention, as acute hypokalemia is the direct cause of muscle weakness and paralysis. Potassium therapy, administered either orally or intravenously as indicated, rapidly increases serum potassium levels, thereby restoring skeletal muscle excitability and preventing serious complications such as cardiac arrhythmias or respiratory failure. In elderly patients, an individualized approach is crucial to balance therapeutic benefits against the risk of adverse events.[1] Early therapy with intravenous potassium chloride (KCl) within the first 24 hours has been shown to produce significant clinical improvement, emphasizing the importance of prompt correction of acute hypokalemia in the management of HPP to restore muscle function and prevent further complications.[8]

CONCLUSION

This case highlights hypokalemic periodic paralysis as an uncommon yet clinically significant complication of insulin therapy in older patients with type 2 diabetes mellitus. In this age group, acute muscle weakness can resemble other neurological disorders, particularly when multiple comorbid conditions are present. Thorough clinical evaluation supported by early electrolyte assessment, especially measurement of serum potassium, plays a key role in establishing the correct diagnosis. The prompt recovery observed after potassium replacement further demonstrates the

reversible nature of this condition with appropriate management. Therefore, clinicians should remain alert to the possibility of insulin-related hypokalemic paralysis in diabetic patients, as timely identification and correction are essential to prevent diagnostic errors, unnecessary diagnostic procedures, and potentially severe complications.

ACKNOWLEDGEMENT

The authors stated no conflict of interest.

REFERENCES

1. Aprilia SE, Tambun O. Potassium therapy in patients with hypokalemic periodic paralysis in RSUD Kabupaten Empat Lawang: a case report. Indones J Gen Med. 2024;3(1):1–10. doi:10.70070/z3w0tq50

2. Dhooria GS, et al. Head drop and flaccid weakness in infantile nephrotic syndrome. Indian J Nephrol. 2018;28(5):382–384. doi:10.4103/ijn.IJN_198_16

3. Husna AN, Mahestari AN, Puspitasari G, et al. Paralisis periodik hipokalemia dengan renal tubular asidosis tipe distal: sebuah laporan kasus. Damianus J Med. 2025;24(2):167–176. doi:10.25170/djm.v24i2.4886

4. Mahardieni K, Nazma D, Soesilo C, et al. Laporan kasus: hipokalemia periodik paralisis. J Akta Trimedika. 2024;1(1):115–123. doi:10.25105/aktatrimedika.v1i1.19214

5. Matthews E, Labrum R, Sweeney MG, et al. Voltage sensor charge loss accounts for most cases of hypokalemic periodic paralysis. Neurology. 2019;72(18):1544–1547.

6. National Organization for Rare Disorders (NORD). Hypokalemic periodic paralysis [Internet]. 2020 [cited 2025 Jun 20]. Available from: https://rarediseases.org/rare-diseases/hypokalemic-periodic-paralysis/

7. Phuyal P, Bhutta BS, Nagalli S. Hypokalemic periodic paralysis. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025 [cited 2025 Jun 20]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK559178/

8. Safitri NE, Yasa IMM. Case report: periodic hypokalemic paralysis. Nurs Health Sci J. 2024;3(2):1–7. doi:10.53713/nhsj.v3i2.256

9. Statland JM, Fontaine B, Hanna MG, et al. Review of the diagnosis and treatment of periodic paralysis. Muscle Nerve. 2018;57(4):522–530. doi:10.1002/mus.26009

10. Wang YL, Li J. Insulin-induced severe thyrotoxic periodic paralysis: A case report. World J Clin Cases. 2025;13(7):101214. doi:10.12998/wjcc.v13.i7.101214

11. Wibawa IDGAP, Dewi AAISK. Clinical approach: hypokalemia periodic paralysis with type 2 diabetes mellitus and insulin. Int J Res Med Sci. 2023;11(4):1450–1454.

12. Wijaya J, Nuranjumi N. Periodik paralisis hipokalemia pada pria 29 tahun. J Penelit Perawat Prof. 2022;4(1):281–288. doi:10.37287/jppp.v4i1.761