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Hypokalemic Periodic Paralysis (HPP) as the Initial Presentation of Uncontrolled Non-Ketotic Diabetes: A Case Report Highlighting the Importance of Screening for Maturity-Onset Diabetes of the Young (MODY) in an Adolescent Female

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ABSTRACT

Introduction

Hypokalemic Periodic Paralysis (HPP) constitutes a rare subgroup of neuromuscular channelopathies characterized by sudden, reversible episodes of flaccid muscle weakness that coincide with profound hypokalemia (serum potassium <3.5 mmol/L). While the majority of HPP cases are attributed to inherited familial disorders (FPP), secondary forms are recognized, often precipitated by hormonal or metabolic imbalances. The co-occurrence of severe HPP manifesting as the primary acute complication of uncontrolled non-ketoacidotic diabetes is exceptionally uncommon and poses a significant diagnostic and therapeutic challenge, especially when the clinical picture suggests an underlying monogenic etiology, such as Maturity-Onset Diabetes of the Young (MODY).

Case Illustration

This report presents a detailed account of a 19-year-old female (Nn. R) who presented to the emergency department on November 7, 2025, with acute and complete flaccid paralysis of the lower extremities (Motor Strength 2/2). Initial biochemical analysis revealed life-threatening hypokalemia (Serum K+=1.50 mEq/L) concomitant with severe, non-ketotic hyperglycemia (Glucose 451 mg/dL, Keton 0.1). The presence of QT prolongation on the electrocardiogram (ECG) indicated high cardiac risk and necessitated immediate admission to the Intensive Care Unit (ICU) for aggressive intravenous potassium replacement and continuous cardiac monitoring. Complete motor function was successfully restored following the normalization of potassium levels (K+ = 3.81 mEq/L) over a six-day hospitalization period.

Discussion

The rapid and complete recovery of muscle strength upon correction of the potassium deficit confirmed the diagnosis of secondary HPP induced by a massive transcellular shift. This shift was triggered by the potent Na+/K+-ATPase activity stimulated by acute metabolic stress (hyperglycemia). Significantly, the patient's presentation—young age, absence of ketoacidosis, and the high efficacy observed upon clinical transition to long-term sulfonylurea (Glikuidone 30 mg) therapy at discharge—strongly suggests an underlying HNF1A- or HNF4A-MODY phenotype. This clinical finding underscores the utility of recognizing atypical diabetic presentations to ensure appropriate, precision-guided long-term care.

Conclusion

This case emphasizes the necessity of prompt, aggressive, and strategically administered potassium management in severe HPP to avert fatal cardiac events. Furthermore, the report highlights the critical diagnostic pathway required to identify underlying MODY in patients with HPP and atypical diabetic features, ensuring the selection of appropriate therapy (sulfonylureas) to prevent recurrent paralytic episodes.

Keywords

Hypokalemic Periodic Paralysis; Maturity-Onset Diabetes of the Young (MODY); Severe Hypokalemia; Sulfonylurea Sensitivity; HNF1A-MODY; Non-Ketotic Hyperglycemia; Flaccid Paralysis.

INTRODUCTION

Background and Pathophysiological Overview

HPP Definition and Spectrum

Hypokalemic Periodic Paralysis (HPP) is defined clinically by recurrent, acute attacks of skeletal muscle weakness or paralysis that occur simultaneously with transiently low serum potassium levels.[1] The pathophysiology is fundamentally linked to defective skeletal muscle membrane excitability, which results from an abnormal influx of potassium ions into the cells, leading to muscle fiber hyperpolarization and inexcitability.[2] HPP is divided into two broad categories: Familial Periodic Paralysis (FPP), which is genetically inherited (e.g., mutations in CACNA1S or SCN4A), and acquired or secondary HPP.[2] Secondary HPP is often associated with conditions causing chronic potassium loss or acute transcellular shifts, such as Thyrotoxic Periodic Paralysis (TPP) or, as in the current case, severe metabolic derangement secondary to uncontrolled diabetes.[2]

MODY Etiology and Subtypes

Maturity-Onset Diabetes of the Young (MODY) is the most common form of monogenic diabetes, typically inherited in an autosomal dominant pattern.[5] It is characterized by an onset usually before age 35 and a relative preservation of endogenous insulin production, distinguishing it from autoimmune T1D (due to lack of antibodies) and typical T2D (due to absence of significant obesity or insulin resistance).[14] The most clinically relevant subtypes are HNF1A-MODY (MODY 3) and HNF4A-MODY (MODY 1), which account for a significant proportion of cases.[5] These subtypes are defined by a progressive failure of pancreatic beta-cell function but are uniquely sensitive to sulfonylurea medications, which can successfully control hyperglycemia long-term .

The Link Between Hyperglycemia and HPP

The induction of an HPP attack in susceptible individuals relies heavily on factors that precipitate a rapid shift of potassium from the extracellular space into the cells. These triggers include rest after exercise, carbohydrate-rich meals, or the administration of glucose or insulin.[2] Uncontrolled hyperglycemia, such as that experienced by the patient (Glucose 451 mg/dL) [6], acts as a potent metabolic trigger. It stimulates endogenous insulin release, which rapidly activates the Na^+/K^+-ATPase pump, driving potassium intracellularly . Furthermore, insulin exacerbates the underlying ion channel dysfunction in HPP-prone muscle fibers by reducing the outward conductance of potassium through inward rectifier K+ channels, thereby promoting prolonged muscle membrane depolarization and subsequent paralysis . This profound effect explains why acute metabolic derangement in diabetes creates an environment highly conducive to HPP attacks.[12]

Research Objective

The primary objective is to thoroughly document the acute presentation, diagnostic challenge, and management of severe secondary HPP triggered by non-ketotic hyperglycemia, which, based on the clinical response, strongly suggests an underlying monogenic diabetes (MODY). Furthermore, the report aims to analyze the therapeutic significance of recognizing this specific diabetic phenotype for optimal long-term care.

Research Benefit

This documentation offers significant clinical value by establishing that severe HPP in a young patient with atypical diabetic features, particularly the absence of ketoacidosis, should immediately trigger a clinical suspicion of MODY.[9] Recognition of MODY allows clinicians to transition the patient to phenotype-specific treatment (sulfonylureas), leading to optimized long-term glycemic control and reduced risk of recurrent HPP episodes, thereby promoting precision medicine in endocrinology.[9]

Hypothesis

The hypothesis posits that the patient's acute presentation of secondary HPP was fundamentally caused by an underlying genetic predisposition (MODY-associated beta-cell dysfunction and glucose dysregulation). This inherent susceptibility was critically amplified by acute physiological stressors, specifically the preceding gastrointestinal potassium loss compounded by profound, sustained hyperglycemia. This sequence of events induced an excessive, acute insulin-mediated intracellular potassium shift that overwhelmed the muscle membrane's capacity for maintaining excitability.[8]

Research Gap

While HPP is recognized as a rare complication of diabetes, existing case reports overwhelmingly associate severe HPP with the presence of Diabetic Ketoacidosis (DKA) or Hyperosmolar Hyperglycemic State (HHS), which introduce significant fluid shifts and acidosis/hyperosmolarity that complicate the etiology . The specific clinical intersection of severe, non-ketotic HPP acting as the acute presentation that leads directly to the clinical suspicion and management consistent with MODY is poorly documented. The precise mechanisms linking common MODY genetic defects to muscle channel sensitivity remain an area requiring further clinical documentation and exploration.

Novelty

This report provides novel clinical data by linking a life-threatening acute secondary HPP event with the subsequent clinical identification and appropriate therapeutic conversion for an underlying monogenic diabetes disorder. The successful and rapid initiation of sulfonylurea (Glikuidone 1x 30 mg) therapy following recovery from the acute crisis—a treatment highly specific to HNF1A/HNF4A-MODY—validates the importance of detailed phenotypic analysis in guiding long-term care and serves as a model for using precision diagnostics to prevent the recurrence of severe complications.

CASE ILLUSTRATION

Initial Presentation and Critical Status

The patient, Nn. R, a 19-year-old female, presented to the emergency room on November 7, 2025.[6] Her chief complaint was the inability to move her legs, a symptom that had progressed from a sensation of heaviness the prior evening to complete flaccid paralysis by midnight. The patient's medical history was notable for prior undocumented episodes of hypokalemia.

Crucial precipitating factors included acute gastrointestinal symptoms experienced the day before admission: 4–5 episodes of watery diarrhea, recurrent nausea, and five episodes of vomiting, accompanied by epigastric pain. These symptoms are recognized contributors to both extracellular fluid depletion and electrolyte losses.

Upon initial assessment, vital signs showed sinus tachycardia (Heart Rate 112 beats/minute) but otherwise stable hemodynamics (Blood Pressure 119/75 mmHg). The patient was fully conscious (Compos Mentis). The neurological examination confirmed severe muscle weakness, specifically flaccid paralysis of the lower extremities, scored 2/2 on the manual muscle strength scale, while upper extremity strength remained intact (5/5). This proximal predominance of weakness is characteristic of HPP .

Diagnostic Findings and Cardiac Risk Assessment

Critical Laboratory and Metabolic Status

Initial laboratory results were immediately revealing and life-threatening. The serum potassium level was critically low at 1.50 mEq/L, well below the threshold for severe hypokalemia (<2.5 mEq/L). Concurrent metabolic analysis showed severe hyperglycemia with a random glucose level of 451 mg/dL. Despite the high glucose, the patient was not in ketoacidosis, confirmed by a negligible ketone level of 0.1.

The admission ECG documented QT prolongation, signifying a high risk of lethal ventricular arrhythmias, which is the most critical acute complication of severe hypokalemia. This finding dictated the urgent need for continuous cardiac monitoring and aggressive electrolyte resuscitation .

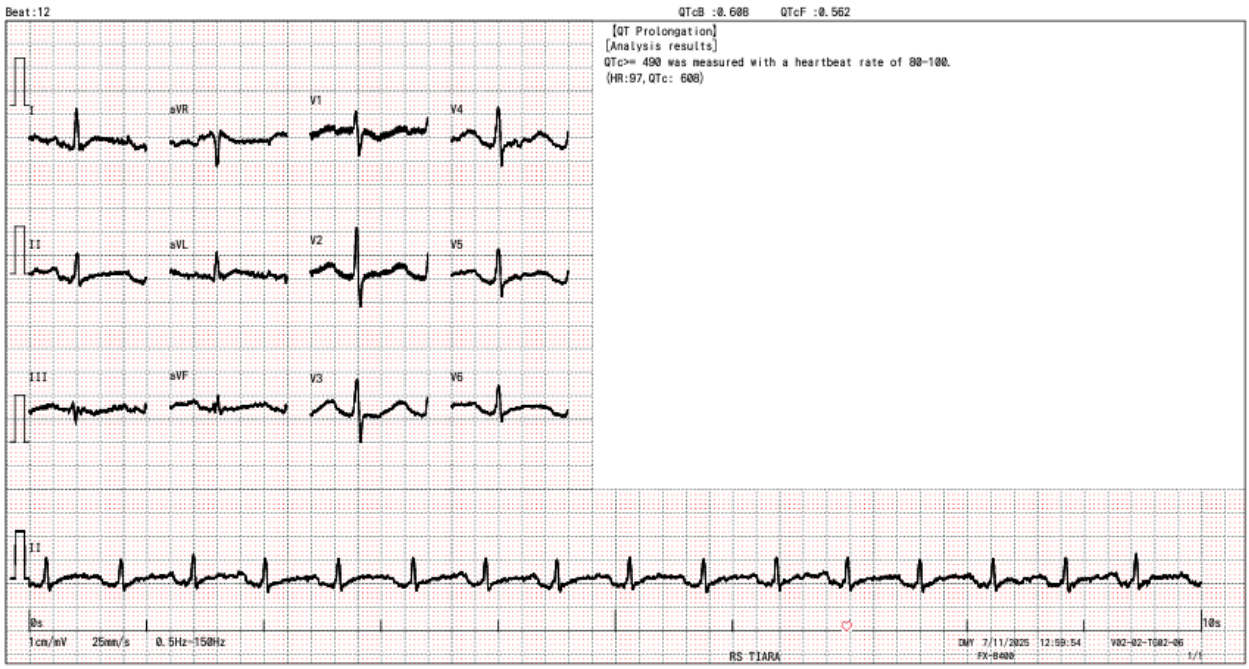

Figure 1. ECG Presentation

Table 1: Key Laboratory Parameters and Sequential Electrolyte Correction

Parameter	Initial Value (7/11/2025)	Day 4 (10/11/2025)	Day 5 (11/11/2025)	Day 6 (12/11/2025)

Hematology				
Hemoglobin (Hb)	14.6	-	-	-
Leukocyte Count (Leuko)	13.96 x 10^3/muL	-	-	-
Platelet Count	503,000/muL	-	-	-
Segmented Neutrophils	80%	-	-	-
Metabolic/Renal				
Random Glucose (GDS)	451 mg/dL	-	-	-
Serum Ketones	0.1	-	-	-
Urea (Ur)	39	-	-	-
Creatinine (Cr)	0.84	-	-	-

Electrolytes (mEq/L)				
Sodium (Na)	136	136	139	138
Potassium (K) (Critical)	**1.50**	**2.12**	**2.78**	**3.81**
Chloride (Cl)	104	108	108	107

Radiographic Evaluation

A chest radiograph (Thorax AP Supine Projection) was performed. The findings indicated that the heart size was not enlarged, and the mediastinum was not widened. The lung parenchyma was clear, with well-defined costophrenic angles, showing no evidence of infiltrates or nodules. This confirmed that the patient was not suffering from acute cardiac failure or severe respiratory compromise.

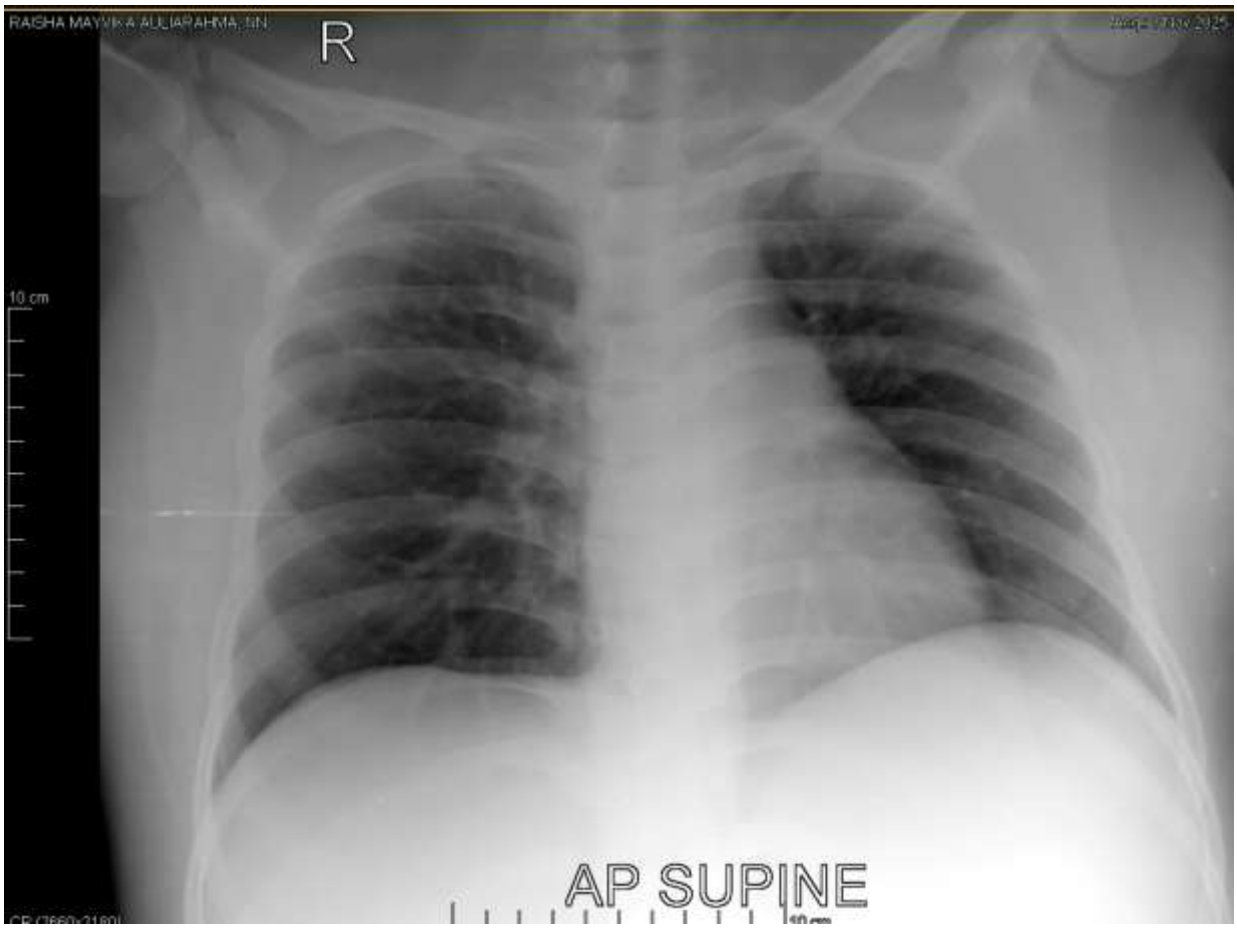

Figure 2. Chest Radiograph Presentation

Acute Management and Sequential Recovery (Day 1 - Day 6)

Due to the severity of the hypokalemia and cardiac instability, the patient was promptly admitted to the ICU.

Resuscitation and Correction Strategy

Initial management involved aggressive intravenous potassium repletion, a necessity for serum K+ < 2.5 mEq/L . The specialist prescribed intravenous infusion of KCl 25 mEq diluted in 500 cc of Normal Saline (0.9% NaCl), administered over 8 hours, and repeated six times initially.

The strict avoidance of glucose-containing solutions was maintained to prevent exacerbation of the hypokalemia via insulin stimulation. The patient also received supportive care, including antiemetics (Ondansetron 4 mg IV), a proton pump inhibitor (Omeprazole 40 mg IV), and empirical broad-spectrum antibiotics (Ceftriaxone 1x 2 gr).

Initial glycemic control was achieved using a sliding scale insulin regimen.

Clinical Progression and Therapeutic Adjustments

Serum potassium levels were monitored closely (Table 1). By Day 4 (10/11), potassium had only reached 2.12 mEq/L. To enhance potassium retention and accelerate correction, Spironolactone (25 mg once daily) was added to the regimen, alongside ongoing IV KCl infusion. The insulin regimen was simultaneously transitioned to a basal-bolus regimen (Sansulin rapid 3x10 units, Sansulin log G 1x10 units at night).

By Day 5 (11/11), potassium levels had reached 2.78 mEq/L, coinciding with the patient reporting significant clinical improvement, including the ability to move her legs slightly. Complete resolution occurred by Day 6 (12/11), when the serum potassium normalized to 3.81 mEq/L, and the patient regained full strength and ambulation ability. The correlation between potassium level correction and recovery of motor function confirms the HPP diagnosis .

Transition to MODY-Specific Therapy

Following complete neurological and electrolyte recovery, the patient was medically cleared for discharge. Based on the patient's atypical diabetes phenotype (young age, non-ketotic presentation) and preserved beta-cell function, the decision was made to shift the patient from the acute insulin regimen to long-term oral hypoglycemic therapy.

The patient was discharged with Glikuidone (30 mg once daily), a sulfonylurea medication, along with KSR (potassium supplementation) and Omeprazole. This choice of Glikuidone, a sulfonylurea, provides strong post-hoc clinical support for an underlying diagnosis of HNF1A- or

HNF4A-MODY, as these subtypes exhibit profound sensitivity to this class of medication, which often serves as the definitive long-term treatment .

DISCUSSION

Etiological Analysis: HPP Secondary to Hyperglycemic Crisis

Mechanisms of Profound Potassium Shift

The severity of the hypokalemia (1.50 mEq/L) strongly differentiates this presentation from simple depletion secondary to GI losses, which typically do not result in such low levels unless chronic or severe volume contraction is present.[18] The rapid onset of paralysis and the severity of the hypokalemia are hallmarks of a transcellular shift disorder, acutely exacerbated by the patient's uncontrolled hyperglycemia.[1]

Hyperglycemia drives endogenous insulin secretion, which, even in the setting of defective MODY beta-cells, is sufficient to rapidly activate the Na+/K+-ATPase . This pump actively transports three Na+ ions out of the cell for every two K+ ions transported in, effectively lowering extracellular potassium. Furthermore, insulin has a secondary, potentiating effect on HPP-susceptible muscle fibers by reducing the outward conductance of the Kir channels, effectively trapping the potassium influx and magnifying the electrical instability that leads to inexcitability and flaccid paralysis . The preceding GI losses (4-5 episodes of diarrhea, 5 episodes of vomiting) likely acted as a priming event, slightly lowering baseline potassium and fluid volume, which then allowed the acute metabolic stress of the high glucose level (GDS 451 mg/dL) to swiftly push the serum K+ into the critical range.[6]

Differential Diagnosis and Contextual Rarity

While Thyrotoxic Periodic Paralysis (TPP) is a common acquired cause of HPP, the primary metabolic trigger (GDS 451 mg/dL) and the non-ketotic status provided the most direct explanation

for the acute shift, making TPP less likely in this context.[3] More importantly, the presentation was atypical for standard diabetic HPP. The majority of published cases reporting severe HPP in uncontrolled diabetes involve severe acidemia (DKA) or extreme hyperosmolarity (HHS) . The patient's non-ketotic status means the mechanism of paralysis was driven purely by the shift mechanism associated with glucose and insulin activity, rather than complex electrolyte disturbances arising from acidosis or profound osmotic diuresis, highlighting an unusual and extreme susceptibility to metabolic shifts.[6] This rarity validates the necessity of this case documentation to fill the identified research gap concerning non-ketotic DM-HPP.[4]

Clinical Justification for MODY Diagnosis

Atypical Features and Clinical Indicators

The patient's age (19 years), the absence of significant co-morbidities typical of T2D (such as obesity or strong insulin resistance), and the non-ketotic nature of the severe hyperglycemia make the diagnosis highly inconsistent with Type 1 or typical Type 2 diabetes.[14] MODY, which often presents in young adults and is frequently misdiagnosed as T1D or T2D, fits this atypical profile.[5] To definitively differentiate MODY from T1D, serological testing for pancreatic autoantibodies (e.g., GAD, IA2, ZnT8) is essential.[13]

Sulfonylurea as a Functional Diagnostic Test

The most compelling justification for the clinical suspicion of MODY lies in the long-term therapeutic choice.[9] Patients with HNF1A-MODY and HNF4A-MODY retain functional beta-cells that are exceptionally sensitive to sulfonylureas . These drugs stimulate insulin secretion, often providing superior glycemic control compared to insulin, even at low doses . The decision to discharge the patient on Glikuidone 1x 30 mg strongly implies a high clinical probability of MODY.[6] This clinical maneuver—transitioning from life-saving acute insulin to phenotype-specific oral therapy—is crucial . If the patient had classic T1D, this switch would result in prompt deterioration of glycemic control. Therefore, the successful initiation of Glikuidone serves as an

effective, real-world confirmation of the hypothesized underlying monogenic defect, enabling the long-term application of precision medicine principles for chronic disease management.[9]

Long-term Management and Prevention of Recurrence

The long-term management strategy for this patient must be dual-focused: achieving metabolic stability appropriate for MODY and preventing any future HPP episodes.[12]

1. **HPP Recurrence Prevention:** Future HPP attacks are prevented by avoiding known triggers (such as high carbohydrate intake, intense exercise, and stress) and maintaining metabolic stability.[12] Since the acute attack was linked to profound hyperglycemia, strict and stable glycemic control is paramount.[6]

2. **MODY Specific Therapy:** The use of sulfonylureas (Glikuidone) provides optimal stability for HNF1A/HNF4A-MODY . By achieving better overall glycemic control, the magnitude of glucose fluctuations and subsequent acute insulinogenic shifts are reduced. This steady state mitigates the risk of overwhelming the inherently susceptible skeletal muscle ion channels, serving as the definitive prophylactic strategy against HPP recurrence.[17]

3. **Genomic Confirmation:** While clinical suspicion is high, definitive genomic testing for common MODY subtypes (HNF1A, HNF4A) is imperative.[19] Genetic confirmation ensures accurate long-term clinical prognosis, validates the current sulfonylurea management, and permits targeted cascade screening and counseling for the patient's family members, who may also carry the autosomal dominant mutation.[13]

CONCLUSION AND RECOMMENDATIONS

Conclusion

This case report details the presentation and management of secondary Hypokalemic Periodic Paralysis, a rare and life-threatening complication, acutely induced by uncontrolled, non-

ketotic hyperglycemia in a 19-year-old female. The diagnosis of HPP was confirmed by the critically low serum potassium (1.50 mEq/L) and immediate resolution of paralysis upon repletion . The patient's unique clinical phenotype, marked by young-onset, non-autoimmune diabetes, led to the critical diagnosis of probable Maturity-Onset Diabetes of the Young (MODY). The successful implementation of sulfonylurea therapy (Glikuidone 1x 30 mg) highlights that severe HPP can be the unmasking event for rare diabetic forms, requiring a sophisticated, personalized approach to long-term care.

Recommendations and Future Practice

1. **Acute Clinical Management:** In cases of severe HPP (especially those with K+ < 2.5 mEq/L or ECG changes), cardiac monitoring and aggressive potassium replacement using non-dextrose solutions are essential, with correction rates adjusted according to symptom severity and ECG changes . Immediate investigation must include simultaneous assessment of thyroid status, serum ketones, and blood glucose to determine the underlying metabolic trigger .

2. **Diagnostic Pathway for Atypical Diabetes:** Clinicians should utilize acute presentations such as non-ketotic DM-HPP in young, non-obese patients as a mandatory trigger for a MODY diagnostic pathway. This pathway should involve testing for pancreatic autoantibodies (to exclude T1D) and subsequent referral for genetic testing targeting HNF1A and HNF4A mutations.

3. **Long-Term Strategy:** Upon confirmation of HNF1A/HNF4A MODY, long-term management should prioritize the use of low-dose sulfonylureas to achieve stable, tight glycemic control. This strategy minimizes acute glucose fluctuations and the associated insulin-mediated potassium shifts, thus providing effective prophylaxis against recurrent, potentially life-threatening HPP attacks.

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