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Case Series

Unusual Endocrine Causes of Acute Hypokalemic Paralysis: Two Case Reports and a Focused Review of the Diagnostic Approach

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ABSTRACT

Acute hypokalemic paralysis is a neurological emergency characterized by sudden or progressive muscle weakness associated with marked hypokalemia. Although thyrotoxic periodic paralysis and familial hypokalemic periodic paralysis are well-recognized causes, endocrine disorders associated with renal potassium wasting may produce a similar clinical presentation and require a fundamentally different diagnostic and therapeutic approach. We report two men with acute hypokalemic paralysis caused by distinct endocrine disorders. A 39-year-old Egyptian man presented with progressive ascending weakness, severe hypokalemia (2.1 mEq/L), marked hypertension (210/120 mmHg), metabolic alkalosis, hyperglycemia, central obesity, purple abdominal striae, and hyperpigmentation. Endocrine evaluation demonstrated hypercortisolism with elevated adrenocorticotropic hormone (ACTH), establishing ACTH-dependent Cushing's syndrome. Imaging did not identify a definitive source of ACTH secretion, and inferior petrosal sinus sampling was unavailable. Neurological symptoms improved after potassium replacement; however, hypokalemia and metabolic alkalosis persisted despite potassium supplementation and spironolactone. Bilateral adrenalectomy was considered because of severe and persistent hypercortisolism, but the patient declined surgery and was discharged against medical advice. The second patient, a 27-year-old Filipino man, developed profound generalized weakness after a large carbohydrate load. Serum potassium was 2.1 mEq/L, with no reported acid–base abnormality. Thyroid testing demonstrated suppressed thyroid-stimulating hormone and elevated total thyroxine, while technetium-99m thyroid scintigraphy showed low uptake, supporting thyrotoxicosis due to painless thyroiditis. His weakness rapidly resolved after intravenous potassium, and he was discharged on propranolol. These cases underscore the importance of considering mineralocorticoid excess and painless thyroiditis in the differential diagnosis of acute hypokalemic paralysis. A high index of suspicion for atypical endocrine pathologies is warranted when routine causes are excluded or when specific clinical features, such as hypertension or metabolic alkalosis, point toward alternative diagnoses.

Key words: Hypokalemic paralysis, Cushing syndrome, ACTH-dependent Cushing syndrome, thyrotoxic periodic paralysis, painless thyroiditis, hypokalemia, metabolic alkalosis, mineralocorticoid excess

INTRODUCTION

Acute hypokalemic paralysis is an uncommon but potentially life-threatening manifestation of severe hypokalemia. It is characterized by acute flaccid weakness, usually affecting the proximal muscles and often involving the lower limbs more prominently than the upper limbs. Respiratory and bulbar muscles are generally spared, although severe cases may be associated with respiratory compromise or cardiac arrhythmias. The major diagnostic challenge is determining whether hypokalemia reflects a transcellular shift of potassium or a true deficit caused by potassium loss. This distinction has immediate therapeutic implications. [1,2]

Thyrotoxic periodic paralysis (TPP) is a recognized cause of hypokalemic paralysis, particularly among young Asian men. Attacks may be precipitated by carbohydrate-rich meals, strenuous exercise, or other factors that promote intracellular potassium uptake. The hypokalemia primarily reflects redistribution rather than depletion of total-body potassium. [3,4] Consequently, potassium replacement must be administered cautiously because excessive replacement can result in rebound hyperkalemia once the transcellular shift resolves. [1,5,6]

In contrast, endocrine disorders characterized by excessive mineralocorticoid activity can cause substantial renal potassium wasting. Cushing syndrome, particularly severe adrenocorticotropic hormone (ACTH)-dependent disease, may produce hypokalemia, metabolic alkalosis, hypertension, and hyperglycemia. At sufficiently severe levels of cortisol excess, cortisol may exert mineralocorticoid effects through activation of renal mineralocorticoid receptors, resulting in increased potassium and hydrogen ion secretion. [7,8]

Painless thyroiditis represents another unusual setting in which thyrotoxicosis may precipitate TPP. Unlike Graves' disease, thyroid hormone excess in painless thyroiditis results from release of preformed thyroid hormone following inflammatory destruction of thyroid tissue. Accordingly, radionuclide thyroid uptake is typically low rather than increased. [9]

These mechanisms can produce a similar emergency presentation—acute weakness with severe hypokalemia—but their diagnostic profiles differ substantially. We describe two patients with acute hypokalemic paralysis associated with ACTH-dependent Cushing syndrome and painless thyroiditis, respectively, and use these cases to emphasize a practical approach to differentiating potassium redistribution from potassium depletion.

CASE SERIES

Case 1: Hypokalemic Paralysis Associated With ACTH-Dependent Cushing Syndrome

A 39-year-old Egyptian man employed as a laborer presented to the emergency department with acute weakness involving all four limbs. Weakness initially affected the lower extremities and subsequently progressed to the upper extremities. He denied vomiting, diarrhea, diuretic use, or other apparent causes of gastrointestinal or medication-related potassium loss. His medical and family histories were otherwise unremarkable.

On examination, blood pressure was 210/120 mmHg and pulse rate was 115 beats/min. Temperature was 36.5°C. General examination demonstrated central obesity, purple abdominal striae (**Figure 1**), and hyperpigmentation, particularly in sun-exposed areas. Cranial nerve examination was normal. Muscle strength was 4/5 in the upper extremities, and tendon reflexes were reduced (1+/2+) throughout. Plantar responses were flexor bilaterally.

Initial laboratory testing demonstrated severe hypokalemia with a serum potassium concentration of 2.1 mEq/L. Serum phosphorus was also reduced at 1.1 mg/dL, while serum bicarbonate was markedly elevated at 44 mmol/L. Random blood glucose was 21.3 mmol/L. Arterial blood gas analysis demonstrated alkalemia (pH 7.52) with a $PaCO_2$ of 48.4 mmHg and HCO_3^- of 39.7 mmol/L, consistent with a prominent metabolic alkalosis. Electrocardiography demonstrated sinus rhythm with prominent U waves.

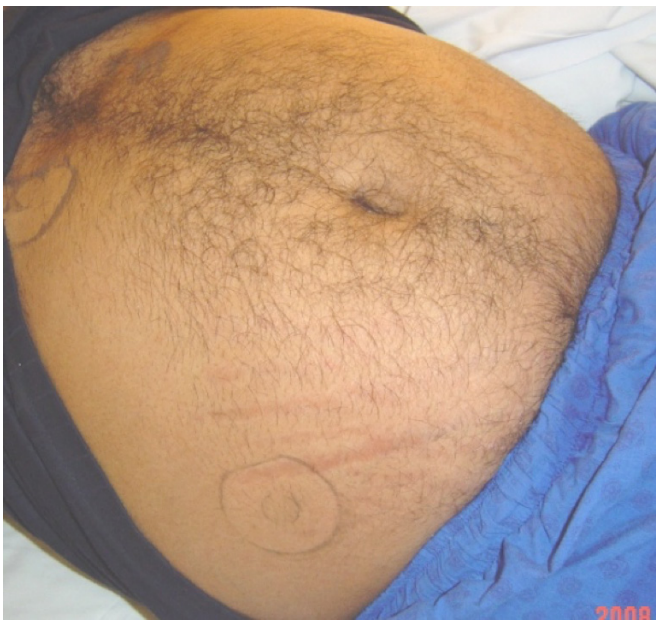

Figure 1: Abdominal examination demonstrating wide purple striae and central obesity in a patient with ACTH-dependent Cushing's syndrome presenting with hypokalemic paralysis.

The combination of severe hypokalemia, hypertension, metabolic alkalosis, hyperglycemia, and physical features suggestive of hypercortisolism prompted an endocrine evaluation. Serum cortisol was elevated, urinary free cortisol was elevated, and cortisol failed to suppress following dexamethasone administration. Serum ACTH was elevated, establishing an ACTH-dependent pattern of hypercortisolism.

Cross-sectional imaging, including abdominal imaging and magnetic resonance imaging of the head, neck, chest, and abdomen, did not identify a definitive ACTH-secreting lesion. Inferior petrosal sinus sampling was unavailable at the treating institution; therefore, a pituitary source could not be reliably distinguished from an ectopic ACTH source.

Intravenous potassium chloride replacement was initiated together with intravenous fluid therapy. The patient's neurological symptoms improved substantially within 24 hours. However, serum potassium remained low, increasing only to 2.6 mEq/L despite replacement. This biochemical response, together with persistent metabolic alkalosis, suggested ongoing potassium loss rather than an isolated transient intracellular shift.

The patient was subsequently treated with spironolactone 50 mg daily and oral potassium chloride 30 mEq every 8 hours. Amlodipine 10 mg daily and perindopril 4 mg daily were prescribed for blood-pressure control, and gliclazide MR 60 mg daily was initiated for hyperglycemia.

Because of severe hypercortisolism with persistent hypokalemia, metabolic alkalosis, and hypertension despite medical therapy, bilateral adrenalectomy was considered

as a means of controlling cortisol excess while the source of ACTH remained unresolved. The patient declined surgical intervention and was discharged against medical advice. Long-term biochemical and clinical follow-up was therefore unavailable.

Case 2: TPP Associated With Painless Thyroiditis

A 27-year-old Filipino man presented to the emergency department in the early morning with sudden severe weakness involving both lower and upper extremities. The evening before presentation, he had consumed a large quantity of sweets. On awakening, he was unable to get out of bed because of profound weakness.

He reported fatigue and intermittent palpitations during the preceding three months but denied neck pain. His previous medical and family histories were unremarkable.

On examination, he appeared anxious and diaphoretic. Heart rate was 104 beats/min and blood pressure was 157/60 mmHg. Neurological examination demonstrated marked symmetrical weakness, with muscle strength of 3/5 in the upper limbs and 1/5 in the lower limbs. Deep tendon reflexes were reduced in the upper limbs and absent in the lower limbs. No sensory deficit was identified.

Serum potassium was 2.1 mEq/L, and electrocardiography demonstrated sinus tachycardia with prominent U waves. The clinical presentation, severe hypokalemia, male sex, Asian ethnicity, and preceding carbohydrate load raised strong suspicion for TPP.

Intravenous potassium replacement was administered. Within 12 hours, the patient's weakness improved dramatically, and the serum potassium increased to 3.4 mEq/L. Other reported blood chemistry parameters, arterial blood gas analysis, and complete blood count were within normal limits, with no reported metabolic alkalosis or acidosis.

Because TPP may occur in association with various causes of thyrotoxicosis, thyroid function testing was performed. Serum TSH was suppressed at 0.04 U/mL, while total T4 was elevated at 21.0 µg/dL. Technetium-99m thyroid scintigraphy demonstrated a normal-sized thyroid with low radionuclide uptake (**Figure 2**). In the context of biochemical thyrotoxicosis, the low uptake pattern supported a diagnosis of painless thyroiditis rather than Graves' disease. The patient was discharged on propranolol. Antithyroid therapy was not initiated because the thyrotoxicosis was attributed to destructive thyroiditis rather than increased thyroid hormone synthesis. Long-term follow-up was unavailable.

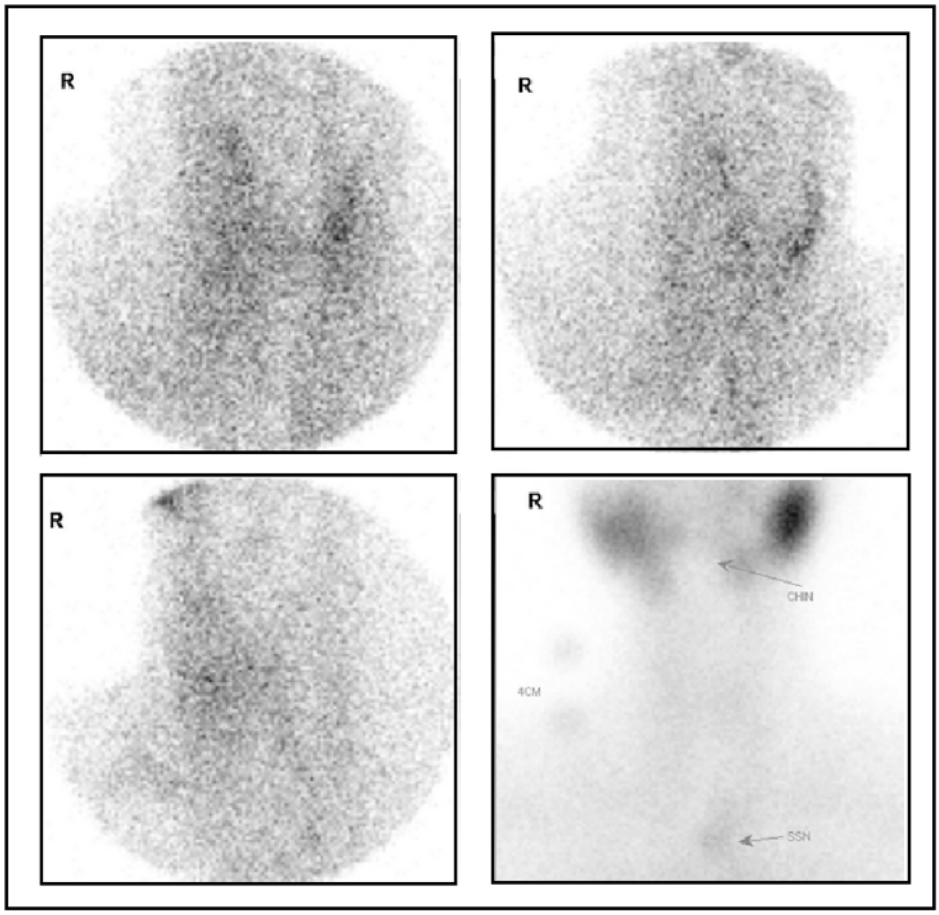

Figure 2: Thyroid scintigraphy performed 20 minutes after intravenous administration of 11 mCi Tc-99m pertechnetate demonstrating absent radiotracer uptake in the thyroid/central neck region, consistent with destructive thyroiditis. Normal salivary gland uptake is visible, confirming adequate tracer administration.

DISCUSSION

These two cases demonstrate that severe hypokalemia with acute paralysis can arise through fundamentally different mechanisms. In the first patient, the clinical phenotype strongly suggested renal potassium wasting associated with severe cortisol excess, whereas the second patient exhibited the typical pattern of transcellular potassium redistribution associated with TPP.

The most useful initial discriminator in a patient with hypokalemic paralysis is not the absolute potassium concentration alone but the combination of blood pressure, acid–base status, and the clinical circumstances surrounding the attack. [10] While TPP is predominantly observed in males and often precipitated by carbohydrate-heavy meals, [11,12] renal potassium wasting syndromes frequently present with hypertension and persistent metabolic derangements. Furthermore, clinicians should remain cognizant of the risk of rebound hyperkalemia during the recovery phase of TPP, [13,14] a complication that necessitates vigilant monitoring as serum potassium levels normalize following the reversal of intracellular shifts.

Case 1: Cushing Syndrome and Potassium-Wasting Hypokalemic Paralysis

The first patient presented with a striking combination of severe hypertension, hypokalemia, metabolic alkalosis, hyperglycemia, central obesity, and purple abdominal striae. This phenotype strongly suggested an endocrine disorder associated with mineralocorticoid activity. Subsequent demonstration of elevated cortisol and ACTH established ACTH-dependent Cushing syndrome.

Cortisol excess can produce hypokalemia and metabolic alkalosis through several mechanisms. In severe hypercortisolism, the capacity of renal 11β-hydroxysteroid dehydrogenase type 2 to inactivate cortisol may be overwhelmed, allowing cortisol to activate mineralocorticoid receptors. Increased distal sodium reabsorption is accompanied by enhanced renal potassium and hydrogen ion secretion, resulting in hypokalemia and metabolic alkalosis. [7]

This mechanism is clinically important because it distinguishes the first case from TPP. In TPP, hypokalemia is predominantly caused by a rapid intracellular shift of potassium. In cortisol-mediated mineralocorticoid excess, ongoing renal potassium wasting may continue until the hormonal abnormality is controlled.

The patient's biochemical response supports this distinction. Although neurological weakness improved after potassium replacement, the serum potassium remained markedly depressed despite continued supplementation. The persistence of metabolic alkalosis further supported ongoing potassium loss rather than a transient redistribution phenomenon.

The elevated ACTH established an ACTH-dependent form of Cushing syndrome; however, the source could not be localized. Importantly, the high-dose dexamethasone suppression test should not be interpreted as definitively distinguishing a pituitary from an ectopic source. Contemporary diagnostic approaches rely on integrated biochemical and imaging assessment, with inferior petrosal sinus sampling used when the source remains uncertain. [8,15]

In this patient, inferior petrosal sinus sampling was unavailable, and imaging did not demonstrate a clear source. Therefore, the most appropriate description is ACTH-dependent Cushing syndrome of unresolved source, rather than pituitary or ectopic Cushing syndrome.

Bilateral adrenalectomy was considered because of severe and persistent cortisol excess. However, the patient declined surgery and was lost to follow-up. Consequently, the definitive outcome of his hypercortisolism remains unknown.

Case 2: TPP Associated With Painless Thyroiditis

The second patient displayed a different diagnostic pattern. He was a young Asian man who developed sudden paralysis after consuming a large carbohydrate load. Severe hypokalemia occurred without a reported acid–base abnormality, and neurological recovery occurred rapidly following potassium administration.

This pattern is characteristic of TPP. Thyrotoxicosis increases Na^+/K^+-ATPase activity, particularly in skeletal muscle, promoting intracellular potassium uptake. A carbohydrate-rich meal can further stimulate insulin secretion and enhance this potassium shift. [1,5]

The patient's suppressed TSH and elevated T4 confirmed thyrotoxicosis. However, determining the cause of thyrotoxicosis was essential because TPP is classically associated with Graves' disease but can occur with other forms of thyrotoxicosis.

The low uptake on technetium-99m thyroid scintigraphy favored a destructive thyroid process. In painless thyroiditis, preformed thyroid hormone is released because of follicular destruction, resulting in transient thyrotoxicosis accompanied by low radionuclide uptake. [9]

This distinction has direct therapeutic implications. Antithyroid drugs inhibit new thyroid hormone synthesis and therefore do not treat the underlying mechanism of destructive thyroiditis. Management is generally supportive, with beta-blockade used to control adrenergic symptoms and prevent recurrent attacks during the thyrotoxic phase. [6,9]

The patient's rapid recovery following potassium replacement also supports a redistribution mechanism. Nevertheless, potassium replacement in TPP should be done cautiously because the total-body potassium deficit is generally limited, and potassium may rapidly shift back into the extracellular compartment as the attack resolves. [16] Subsequent episodes in such cases are typically prevented by stabilizing the thyrotoxic state, often through the judicious use of beta-adrenergic blockade to modulate thyroid-mediated enzyme activity. [17,18]

Although both cases exhibited hypokalemic paralysis, the underlying causes, mechanisms, and responses to potassium differ. It is clinically significant to distinguish between potassium loss and redistribution. In individuals experiencing hypokalemic paralysis, assessing urinary potassium can clarify whether the kidneys are effectively conserving potassium. While urinary potassium indices were not accessible in the current cases, they should be included in the evaluation

whenever possible. The key differences between the two cases are outlined in **Table 1**.

Practical Diagnostic Approach to Differential Diagnosis

A patient presenting with acute weakness and hypokalemia should first undergo immediate cardiac monitoring and appropriate potassium replacement while simultaneously undergoing etiological evaluation.

The next step should be to determine whether the clinical picture is more consistent with transcellular potassium redistribution or true potassium depletion.

A normal acid–base profile in a young patient with sudden paralysis, particularly after carbohydrate intake or exercise, should raise suspicion for TPP or familial hypokalemic periodic paralysis. Thyroid function testing is therefore appropriate even when classic clinical manifestations of hyperthyroidism are absent. [19,20]

Conversely, hypokalemia accompanied by hypertension and metabolic alkalosis should prompt evaluation for renal potassium wasting and mineralocorticoid activity. Relevant causes include primary hyperaldosteronism, severe Cushing syndrome, ectopic ACTH secretion, and selected renal tubular disorders. [21,22]

The diagnostic evaluation should be guided by the clinical phenotype and may include:

1. Serum electrolytes, including potassium, bicarbonate, magnesium, and phosphorus.
2. Electrocardiography and continuous cardiac monitoring when hypokalemia is severe.
3. Arterial or venous blood gas analysis when the acid–base status is uncertain.
4. Urinary potassium assessment to determine whether renal potassium wasting is present.
5. Thyroid function testing when periodic paralysis is suspected.
6. Cortisol and ACTH evaluation when clinical features suggest hypercortisolism.
7. Renin and aldosterone assessment when hypertension and metabolic alkalosis suggest mineralocorticoid excess.
8. Appropriate imaging and specialized testing to localize an endocrine source once biochemical diagnosis has been established.

This approach avoids prematurely labeling every episode of hypokalemic paralysis as TPP and ensures that patients with endocrine potassium wasting receive treatment directed at the underlying disorder. A stepwise clinical algorithm for Acute Hypokalemic Paralysis is illustrated in **Figure 3**. This stepwise clinical algorithm illustrates the concurrent emergency management and etiological investigation of patients presenting with acute weakness and hypokalemia. Initial assessment comprises confirmation of hypokalemia, electrocardiographic evaluation, cardiac monitoring (when indicated), and determination of serum electrolytes and acid-base status. The pathway subsequently stratifies patients based on acid-base status, blood pressure, and urinary potassium excretion to differentiate transcellular redistribution from true potassium depletion. Normotensive patients with a normal acid-base profile are directed toward evaluation for thyrotoxicosis or familial periodic paralysis. Patients with metabolic alkalosis undergo further assessment for mineralocorticoid excess, Cushing syndrome, renal tubular disorders, or extrarenal losses. Targeted endocrine and renal investigations are then tailored to the clinical phenotype to elucidate the underlying cause and prevent recurrent episodes of hypokalemia and paralysis.

The differential diagnosis should be organized according to the underlying pathophysiological mechanism: whether hypokalemia results from transcellular redistribution or from a true deficit caused by potassium loss. This distinction is clinically essential because redistribution disorders, such as TPP, generally require cautious potassium replacement and treatment of the precipitating endocrine abnormality, whereas potassium-depletion states require identification and correction of ongoing renal or extrarenal losses. [23] Accordingly, the principal causes and distinguishing clinical features of acute hypokalemic paralysis are summarized in **Table 2**.

Table 1: Comparison of clinical and diagnostic features of the two cases.

Feature	Case 1	Case 2
Mechanism	Predominantly potassium wasting	Predominantly intracellular potassium shift
Underlying disorder	ACTH-dependent Cushing syndrome	Thyrotoxicosis due to painless thyroiditis
Onset	Progressive	Sudden
Blood pressure	Markedly elevated	Mildly elevated systolic pressure
Acid–base status	Metabolic alkalosis	No reported abnormality
Hyperglycemia	Present	Not reported
Clinical endocrine clues	Central obesity, purple striae, hyperpigmentation	Palpitations, thyrotoxic biochemical profile
Potassium response	Persistent biochemical hypokalemia	Rapid correction
Trigger	Hormonal potassium wasting	High-carbohydrate meal
Definitive etiological evaluation	ACTH-dependent; source unresolved	Low thyroid uptake supporting painless thyroiditis

ACTH, adrenocorticotropic hormone.

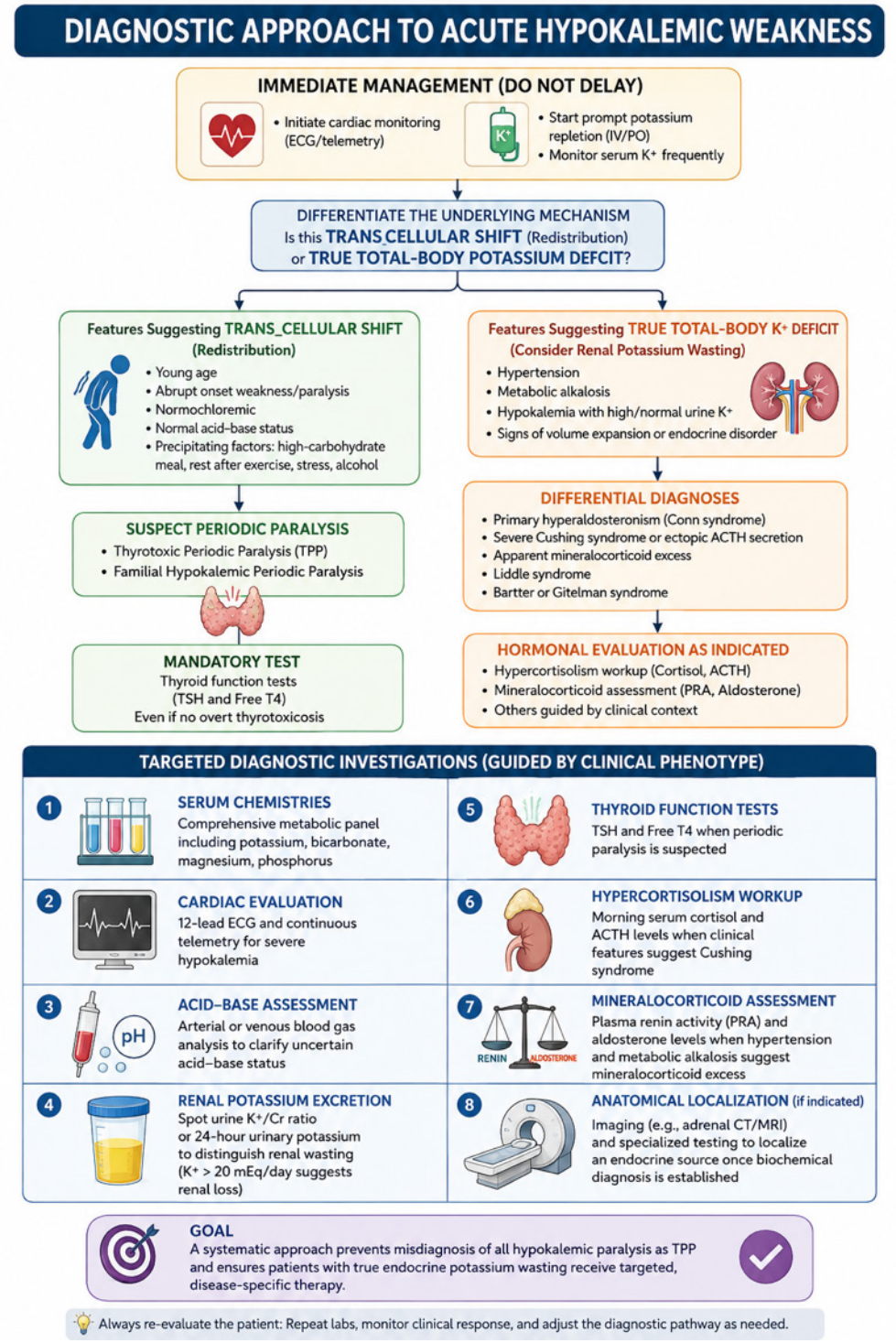

Figure 3: Diagnostic algorithm for acute hypokalemic paralysis.

Therapeutic Considerations

Treatment of hypokalemic paralysis should address both the immediate neurological/electrolyte emergency and the underlying cause.

In severe hypokalemia, potassium replacement should be accompanied by cardiac monitoring and serial electrolyte measurements. The rate and route of potassium administration should be determined according to the severity of hypokalemia, electrocardiographic findings, symptoms, and institutional protocols. [24,25]

In TPP, potassium replacement should be conservative because the underlying problem is predominantly redistribution rather than a major total-body potassium deficit. Excessive replacement can produce rebound hyperkalemia when potassium returns to the extracellular compartment. [19,26]

Table 2: Clinical approach to the differential diagnosis of acute hypokalemic paralysis.

Etiology	Typical clues	Acid–base pattern	Mechanism
Thyrotoxic periodic paralysis	Young man, thyrotoxicosis, carbohydrate/exercise trigger	Usually normal	Intracellular shift
Familial hypokalemic periodic paralysis	Family history, recurrent attacks, young age	Usually normal	Intracellular shift
Cushing syndrome/ectopic ACTH	Hypertension, hyperglycemia, Cushingoid features	Metabolic alkalosis	Renal potassium wasting
Primary hyperaldosteronism	Hypertension, suppressed renin, elevated aldosterone	Metabolic alkalosis	Renal potassium wasting
Diuretic-associated hypokalemia	Medication history	Usually metabolic alkalosis	Renal potassium loss
Gastrointestinal potassium loss	Vomiting or diarrhea	Variable	Extrarenal potassium loss
Gitelman syndrome	Chronic/recurrent hypokalemia, hypomagnesemia	Metabolic alkalosis	Renal potassium wasting
Bartter syndrome	Chronic hypokalemia, metabolic alkalosis	Metabolic alkalosis	Renal salt wasting
Liddle syndrome	Hypertension, low renin and aldosterone	Metabolic alkalosis	ENaC overactivity
Insulin/beta-adrenergic shift	Insulin administration, catecholamine exposure	Usually normal	Intracellular shift
Hypomagnesemia	Refractory hypokalemia	Variable	Impaired potassium retention

ENaC, epithelial sodium channel.

In contrast, persistent hypokalemia in the setting of mineralocorticoid activity requires treatment of the ongoing renal potassium loss. Mineralocorticoid receptor antagonists, such as spironolactone, may be useful for cortisol-mediated mineralocorticoid effects, while definitive management depends on the underlying cause of hypercortisolism. [27] Prompt identification and treatment of the primary etiology are also essential to prevent long-term complications, including cardiac arrhythmias and recurrent paralytic episodes. [28,29]

In painless thyroiditis, treatment is generally directed toward controlling symptoms during the thyrotoxic phase. Beta-blockers can reduce adrenergic symptoms and may help reduce the risk of recurrent TPP attacks. Antithyroid drugs are generally not useful when thyrotoxicosis results from release of preformed hormone rather than increased hormone synthesis. [30,31]

Limitations

This report has several limitations. First, the ACTH source in Case 1 could not be definitively localized because inferior petrosal sinus sampling was unavailable, and the available imaging did not identify a causative lesion. Accordingly, the case should be described as ACTH-dependent Cushing syndrome of unresolved source rather than as definitively pituitary or ectopic disease.

Second, urinary potassium measurements, renin, aldosterone, and magnesium values were not reported. These investigations would have provided additional evidence regarding the mechanism and severity of renal potassium wasting and would have strengthened the differential diagnosis.

Third, long-term follow-up was unavailable for both patients. In Case 1, the patient declined definitive surgical treatment and was discharged against medical advice, preventing assessment of subsequent cortisol control, potassium status, blood pressure, or recurrence of paralysis. In Case 2, the absence of long-term follow-up prevents confirmation of resolution of the thyrotoxic phase and documentation of recurrent paralysis.

Fourth, the report includes only two patients and therefore cannot establish the relative frequency of these etiologies or generalize treatment outcomes.

Despite these limitations, the contrasting biochemical and clinical phenotypes provide useful practical lessons for the evaluation of acute hypokalemic paralysis.

CONCLUSIONS

Acute hypokalemic paralysis represents a heterogeneous clinical syndrome rather than a single disease entity. The two cases presented here illustrate two contrasting endocrine mechanisms: potassium wasting associated with severe ACTH-dependent Cushing syndrome and intracellular potassium redistribution associated with TPP secondary to painless thyroiditis.

The combination of blood pressure, acid–base status, clinical phenotype, and response to potassium replacement provides an important initial framework for distinguishing these mechanisms. Severe hypokalemia accompanied by hypertension and metabolic alkalosis should prompt evaluation for mineralocorticoid activity and renal potassium wasting, whereas sudden paralysis following a carbohydrate load with normal acid–base status should raise suspicion for TPP.

A systematic diagnostic approach is therefore essential. Early recognition of the underlying endocrine disorder allows

treatment to move beyond temporary potassium correction toward definitive control of the hormonal abnormality, thereby reducing the risk of recurrent paralysis, cardiac complications, and other endocrine morbidity.

Learning Points

1. Acute hypokalemic paralysis is a medical emergency requiring prompt potassium assessment, electrocardiographic evaluation, cardiac monitoring, and appropriate potassium replacement.
2. Hypokalemia should not automatically be attributed to periodic paralysis. The underlying mechanism may be potassium redistribution or true potassium depletion.
3. Hypertension plus metabolic alkalosis and severe hypokalemia should raise suspicion for mineralocorticoid activity and renal potassium wasting.
4. Severe ACTH-dependent Cushing syndrome can rarely present with hypokalemic paralysis, particularly when cortisol excess produces clinically significant mineralocorticoid effects.
5. High-dose dexamethasone suppression should not be used alone to distinguish pituitary from ectopic ACTH secretion. When localization remains uncertain, additional biochemical, imaging, and, when appropriate, inferior petrosal sinus sampling are required.
6. Sudden paralysis after a carbohydrate-rich meal in a young man with hypokalemia and normal acid–base status should prompt consideration of TPP.
7. TPP can occur with painless thyroiditis, not only Graves' disease. Low radionuclide uptake in the setting of thyrotoxicosis supports a destructive thyroiditis mechanism.
8. Potassium replacement should be tailored to the underlying mechanism. Excessive replacement in potassium-shift disorders can cause rebound hyperkalemia.
9. Persistent hypokalemia despite potassium replacement suggests ongoing potassium loss and should prompt assessment of renal potassium handling and endocrine causes.
10. Treatment of the underlying endocrine disorder is essential to prevent recurrent hypokalemic paralysis and associated cardiovascular complications.

CONSENTS

Written informed consent was obtained from the patients for publication of this case report.

AUTHORS' CONTRIBUTION

All authors have significantly contributed to the work, whether by following the case at the bedside, conducting literature searches, drafting, revising, or critically reviewing the article. They have given their final approval of the version to be published, have agreed with the journal to which the article has been submitted, and agree to be accountable for all aspects of the work.

DISCLOSURE OF ARTIFICIAL INTELLIGENCE ASSISTANCE

The authors disclose the use of AI technologies in the preparation of this manuscript, specifically for linguistic refinement, content restructuring, and figure creation. Despite this assistance, the intellectual framework, analysis, and scholarly conclusions remain the exclusive original work of the authors.

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CONFLICT OF INTEREST

None.

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