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Hypokalemia Havoc: Unraveling the Mystery of Unexplained Potassium Depletion

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Abstract

Hypokalemia, defined as serum potassium below 3.5 mEq/L, can lead to severe complications such as arrhythmias and muscle paralysis, potentially resulting in rhabdomyolysis. The etiology of hypokalemia is often multifactorial, involving but not limited to gastrointestinal losses, renal losses, medication effects, and inadequate dietary intake. Chronic heavy alcohol use, obstructive sleep apnea (OSA), and the use of diuretics such as hydrochlorothiazide (HCTZ) are also significant contributing factors. Effective management requires thorough evaluation and investigation to effectively treat a patient. This case report aims to illustrate the diagnostic challenges and comprehensive treatment approach required in a patient with multiple comorbidities and severe hypokalemia, emphasizing the need for a multidisciplinary and comprehensive approach to address all underlying causes.

Keywords

Severe Hypokalemia, Electrolyte Derangements, Thiazide Diuretic Complications, Potassium Repletion

1. Introduction

Hypokalemia is a common and potentially life-threatening electrolyte disorder characterized by serum potassium levels below 3.5 mEq/L. Severe hypokalemia can lead to complications such as arrhythmias, muscle paralysis, and rhabdomyolysis [1] [2]. It affects approximately 20% of hospitalized patients and is associated with a two-fold higher mortality rate [3]. Severe hypokalemia, with serum levels below 2.5 mEq/L, significantly increases the risk of mortality [4]. The causes of hypokalemia are diverse, including contributors from gastrointestinal losses, renal losses, medication effects, and inadequate dietary intake. Chronic

heavy alcohol use can cause bone marrow suppression, leading to chronic anemia and reticulocytosis, which sequesters potassium in newly formed red blood cells. Obstructive sleep apnea (OSA) contributes to hypokalemia through intermittent hypoxia, which activates the renin-angiotensin-aldosterone system, increasing aldosterone production and promoting potassium loss. The use of diuretics, particularly hydrochlorothiazide (HCTZ), further exacerbates potassium depletion. Here, we present a case of a 40-year-old male with severe hypokalemia unresponsive to standard treatment, highlighting the intricate interplay of multiple contributing factors and the importance of a comprehensive and multidisciplinary approach to achieve normalization of potassium levels.

2. Case Report

We present a 40-year-old male with a past medical history of hypertension, obstructive sleep apnea, morbid obesity, and heavy alcohol use who presented to the emergency department with complaints of generalized weakness, muscle cramps, and palpitations for two weeks. Social history was significant for occasional marijuana use, and alcohol consumption, approximately 1.7 L of whiskey nearly daily. He lacked regular follow-up with a primary physician, but recently visited an Urgent Care and was prescribed hydrochlorothiazide for his elevated blood pressure. He continued the hydrochlorothiazide for two weeks but returned to Urgent Care due to complaints of dizziness and fatigue. Lab work demonstrated a potassium level of 2.3 mmol/L, and he was advised to stop the medication. In the weeks to follow, the patient developed episodes of vomiting, diarrhea and a decreased appetite. His symptoms progressively worsened leaving him nearly bed-bound from muscle weakness, which then prompted a visit to the Emergency Department. On presentation to the ED, his vital signs were notable for blood pressure 145/110, respiratory rate 20, heart rate 110 bpm, and SpO2 94% on room air; BMI was 37.8. He was alert and oriented but appeared lethargic and diaphoretic. Lab work on admission was notable for a serum potassium of 1.3 mmol/L, serum chloride of 78 mmol/L, magnesium of 1.4 mg/dL and a serum bicarbonate of > 45 mmol/L. Venous blood gas showed severe primary metabolic alkalosis with a pH of 7.6, pCO2 48 mmHg, and bicarbonate 34 mmol/L. EKG demonstrated sinus tachycardia with a QTc interval of 647 ms. The patient was subsequently admitted to the ICU for continuous cardiac monitoring and aggressive electrolyte replacement.

Intravenous calcium gluconate was administered immediately to stabilize the cardiac membrane to prevent arrhythmias. Potassium repletion was also initiated via a central line. Despite aggressive replenishment measures, his potassium levels only marginally increased from 1.3 to 2 mmol/L within the first 48 hours after receiving nearly 1000 mEq of intravenous and oral potassium. This potassium deficit posed a perplexing query to the medical team as this was quite unusual as repletion at this amount should have normalized his potassium. Further workup included evaluation of potassium wasting syndromes, however, urine

chemistries were low (urine potassium: 7 mmol/L; urine chloride: 39 mmol/L) suggesting that the kidneys were appropriately retaining electrolytes. Next, we conducted a secondary hypertension workup as the patient was noted to have persistent hypertension despite HCTZ use. We obtained cortisol levels, 24-hour catecholamine, renin and aldosterone levels which were normal. A CT of the abdomen with adrenal protocol to assess for masses was completed but also unremarkable. We then took a closer look at the patient's other medical conditions and noted that his obstructive sleep apnea was most likely perpetuating periods of intermittent RAAS activation and increased aldosterone, contributing to his secondary hypertension and worsening hypokalemia. The patient was also noted to have normocytic anemia with an elevated reticulocyte count of 6.8%. This significant elevation of immature RBCs led us to consider an additional source of potassium depletion by the mechanism of sequestration of potassium. This mechanism is known to utilize intracellular potassium for various metabolic functions, including building the structure and integrity of new RBCs.

The patient continued with potassium repletion and ultimately received about 1650 mEq which helped bring his serum potassium to 4.1 mmol/L over a five-day time span. As his potassium levels had finally stabilized to a normal range, the remaining electrolytes and metabolic alkalosis had resolved as well. The patient gradually regained his strength and returned to his normal state of health.

3. Discussion

This case underscores the critical need for prompt recognition and treatment of severe hypokalemia to prevent life-threatening complications such as arrhythmia, muscle paralysis, and rhabdomyolysis, all while uncovering the causes of such severe electrolyte derangements. The original culprit in this case was the hydrochlorothiazide, a thiazide diuretic significantly associated with hypokalemia. However, to have a potassium deficit of approximately 1650 mEq is unusual unless there was consideration of underlying renal potassium wasting disorders such as Gittelman or Bartter Syndrome. With additional urine chemistries indicating low urine potassium and chloride, these pathologies had been ruled out as these levels are expected to be elevated [5]. Our patient had multiple morbidity factors further contributing to potassium depletion including obesity contributing to OSA, poor oral intake as noted by his generalized weakness and continued GI losses from HCTZ, and his chronic alcohol use leading to bone marrow suppression. All of these factors most likely contributed to a small role individually, but when considered together could explain the patient's severe potassium depletion.

Our patient was a heavy alcohol user who indulged in 1.7 L bottle of whiskey daily. Heavy alcohol consumption can lead to hypokalemia through various mechanisms, including diuresis, poor nutritional intake and chronic anemia from bone marrow suppression. Assuming intact bone marrow, anemia can trigger subsequent reticulocytosis to proliferate new red blood cells to replace

those lost by alcohol's toxic effects [6] [7]. A reticulocyte count of 6.8% in this patient was suggestive of immature red blood cells continuously being produced. Notably, reticulocyte maturation is also associated with higher K-Cl cotransport proteins which can cause increased uptake of intracellular potassium, further depleting serum potassium levels. The new RBCs utilized the constant influx of IV potassium to build structure, necessitating the need to aggressively replete potassium [6] [8]. After repleting the potassium levels in this patient, the reticulocyte count nearly normalized. Another contributor to our patient's hypokalemia was his morbid obesity and subsequent obstructive sleep apnea. OSA is associated with intermittent hypoxia which can intermittently activate the renin-angiotensin-aldosterone system, thereby leading to increased aldosterone production, causing hypertension and potassium excretion [9] [10]. When combined with the recent use of HCTZ, it would be reasonable to hypothesize that potassium was further lost through this mechanism. The patient's potassium deficit was profound, requiring almost 1650 mEq to achieve a serum potassium level of 4.1 mmol/L from 1.3 mmol/L. When considering poor oral intake, chronic alcohol use leading to anemia and reticulocytosis, underlying OSA, and recent HCTZ use, it is reasonable to conclude that our patient's intracellular potassium stores were nearly depleted on admission, necessitating an aggressive potassium repletion regimen to restore him to baseline levels.

4. Conclusion

This case demonstrates a rare and severe presentation of hypokalemia in a middle-aged male and provides valuable insight into the diagnosis, management, and potential differentials for the cause of such a severe form of electrolyte abnormality. Clinicians should maintain a high index of suspicion for other causes of hypokalemia in patients with relevant symptoms and history, especially if typical repletion protocols are insufficient to return the patient to their baseline potassium levels. Future research should not only aim to assess the effectiveness of different electrolyte repletion strategies, but also to develop preventive guidelines for high-risk populations, especially those with characteristics similar to our patient, including obesity, chronic alcohol use and diuretic therapy.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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