International Journal of Science and Research (IJSR)
ISSN: 2319-7064
Impact Factor 2024: 7.101

Diagnostic Approach and Management of Severe Hypokalemia in a Suspected Case of Bartter Syndrome: A Clinical Case Study

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Abstract: ***Severe and life-threatening hypokalemia is defined as a plasma potassium level of <2.5 mEq/L. Hypokalemia can result from various factors, one of which is excessive potassium loss via the gastrointestinal tract, kidneys, or sweat. Bartter syndrome is a rare genetic disorder marked by impaired potassium reabsorption in the thick ascending limb of the loop of Henle, with an estimated prevalence of 1 in 1,000,000. This case study discusses the clinical presentation, diagnostic workup, and management of a 53-year-old male with severe hypokalemia (1.4 mEq/L) and suspected Bartter syndrome. The patient exhibited acute limb weakness without gastrointestinal or renal history and was evaluated with ECG, serum electrolytes, and transtubular potassium gradient, revealing renal potassium loss. Treatment involved IV and oral potassium supplementation, spironolactone, and dietary support. Diagnosis was based on clinical patterns and lab markers consistent with Bartter syndrome, although genetic testing was unavailable. The case underscores the importance of prompt identification and management of underlying renal causes of hypokalemia in emergency settings. This case report is intended to raise awareness and enhance clinical knowledge among physicians, enabling timely and appropriate management to reduce complications and improve the quality of life of patients with Bartter syndrome.***

Keywords: Hypokalemia, Bartter Syndrome, potassium reabsorption, renal tubular disorder

1. Introduction

Hypokalemia is a condition defined by a plasma potassium concentration of less than 3.5 mEq/L. [1] Severe and life-threatening hypokalemia is defined as a potassium level <2.5 mEq/L. [2] Hypokalemia is a common electrolyte disorder, particularly among hospitalised patients. [2] More than 20% of hospitalised patients have been found to develop hypokalemia; however, only 4–5% exhibit clinical symptoms. Severe hypokalemia is relatively uncommon. [2] Approximately 80% of patients receiving diuretic therapy develop hypokalemia. [2] Hypokalemia may result from several conditions, including inadequate potassium intake, excessive potassium loss through the gastrointestinal tract, kidneys, or sweat, or an increased intracellular shift of potassium (translocation). [1] Muscle weakness, fatigue, muscle pain, and restless legs syndrome are neuromuscular symptoms that may occur in patients with potassium levels below 3.0 mEq/L. More profound reductions may lead to paralysis or rhabdomyolysis. The cardiac effects of hypokalemia include arrhythmias, such as atrial fibrillation and ventricular tachycardia, which occur due to delayed ventricular repolarisation resulting in increased re-entry currents. Hypokalemia may also cause impaired glucose tolerance and disturbances in protein metabolism. [1]

Hypokalemia may be caused by various factors, including genetic disorders that impair renal tubular reabsorption, resulting in excessive urinary potassium excretion. One such condition is Bartter syndrome, which leads to defects in potassium reabsorption in the thick ascending limb of the loop of Henle and is estimated to affect approximately 1 in 1,000,000 individuals. A thorough diagnostic evaluation-including detailed history taking, physical examination, and comprehensive ancillary investigations—is required to establish the diagnosis of Bartter syndrome, as patients typically have no significant prior complaints. The purpose of this case report is to highlight the diagnostic process and management of severe hypokalemia due to suspected Bartter syndrome and to enhance clinical awareness of this rare but serious condition.

2. Case Report

A 53-year-old man presented to the emergency department (ED) with sudden generalised weakness of all four limbs, which prevented him from getting out of bed without assistance. The weakness and fatigue involved the entire body except for the neck and head. Before this episode, he had been able to carry out his daily activities as usual. He denied any history of falls or back pain. He also denied a history of hypertension, asthma, diabetes mellitus, or tuberculosis. There was no associated abdominal pain, loss of appetite, nausea, vomiting, or diarrhoea. He reported a similar episode several years earlier, for which he was treated at a clinic with medication for one week, after which he was able to walk and resume normal activities. He denied taking any regular medications.

On arrival at the emergency department, the patient was fully conscious (compos mentis). His vital signs showed a blood pressure of 103/63 mmHg, heart rate of 60 beats/min, respiratory rate of 20 breaths/min, body temperature of 37.3 °C, and oxygen saturation of 99% on room air. General physical examination revealed non-pale conjunctivae and non-icteric sclerae. Motor strength was decreased in all four extremities, graded 2/5 in both upper and lower limbs, consistent with quadriparesis. Cardiovascular, respiratory, and abdominal examinations were within normal limits.

Ancillary testing showed a haemoglobin level of 13 g/dL, haematocrit of 37.2%, white blood cell count of 12.9 ×10³/μL, and platelet count of 326 ×10³/μL. Differential count (basophils/eosinophils/neutrophils/lymphocytes/monocytes) was 0.3/4.0/82.8/9.4/3.5%. Random plasma glucose was 125 mg/dL. Clinical chemistry revealed sodium of 140 mEq/L, potassium of 1.4 mEq/L, and chloride of 102 mEq/L, with a plasma osmolality of 287 mOsm/kg H_2O and a transtubular potassium gradient (TTKG) of 6. Blood urea nitrogen (BUN) was 37 mg/dL, serum creatinine was 1.26 mg/dL, and the estimated glomerular filtration rate (eGFR) was 68 mL/min/1.73 m². Arterial blood gas analysis showed a pH of 7.50, $PaCO_2$ of 30.5 mmHg, PaO_2 of 121.3 mmHg, bicarbonate (HCO_3^-) of 23.7 mmol/L, and oxygen saturation of 98%. Urinalysis over 24 hours showed a urine volume of 4.14 L, urine osmolality of 272.69 mOsm/kg H_2O, urine sodium of 90 mEq/L, urine potassium of 8 mEq/L, and urine chloride of 85 mEq/L. Chest radiography showed no evidence of active pulmonary tuberculosis or cardiomegaly. Electrocardiography demonstrated sinus rhythm at 60 beats/min, flattened T waves (particularly in leads I, III, and aVR), ST-segment depression, and prominent U waves, most evident in leads V2–V3. Abdominal ultrasonography showed no abnormalities of the intra-abdominal organs.

The patient was hospitalised for five days. During admission, he received intravenous fluid therapy with Ringer's acetate 500 mL with potassium chloride (KCl) 50 mEq infused over 12 hours, oral potassium chloride sustained-release 1,200 mg three times daily (local brand: KSR), and other symptomatic treatments according to his complaints. Throughout hospitalisation, the patient was provided a regular diet of 1,700 kcal/day with high potassium content, supplemented with additional bananas.

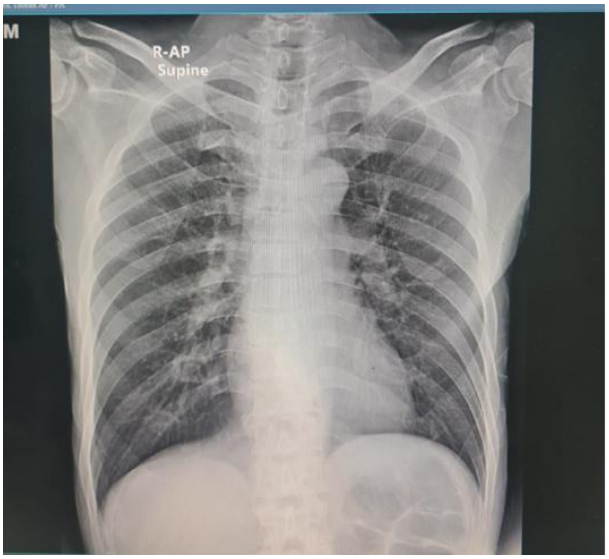

Figure 1: Chest radiograph of the patient

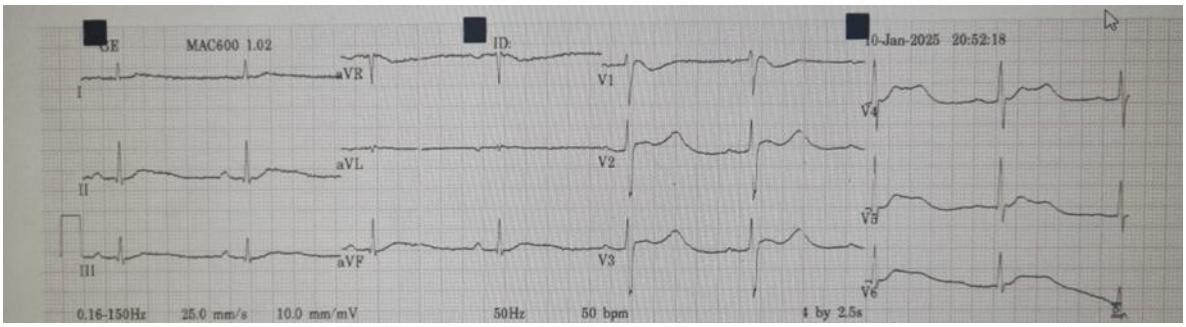

Figure 2: Electrocardiogram of the patient.

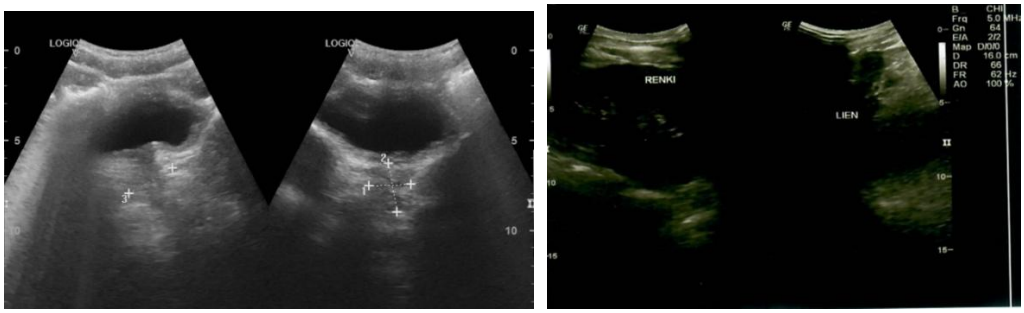

Figure 3: Abdominal ultrasonography of the patient.

Table 1: Trends in the patient's laboratory results.

Laboratory Results	Day 1	Day 3	Day 5
Sodium (mEq/L)	140	141	139
Potassium (mEq/L)	1.4	2.2	3.7
Chloride (mEq/L)	102	103	102
Magnesium (mg/dL)	NA	2.1	NA
Aspartate aminotransferase (AST, U/L)	45	NA	NA
Alanine aminotransferase (ALT, U/L)	13	NA	NA

3. Discussion

Hypokalemia is defined as a plasma potassium concentration below 3.5 mEq/L [1]. Hypokalemia is classified according to plasma potassium levels [1,2]. Although precise data on the prevalence of hypokalemia in the general population are lacking, it is estimated that fewer than 1% of individuals not using medications have serum potassium levels below 3.5 mEq/L. This low prevalence is influenced by several factors, including interindividual differences in potassium intake and variability in sensitivity to conditions that may precipitate

hypokalemia. In hospitalised patients, however, up to 21% are estimated to have serum potassium levels below 3.5 mEq/L, and approximately 5% of these patients have potassium levels below 3.0 mEq/L. In the older population, nearly 25% are estimated to have serum potassium levels below 3.0 mEq/L. Among outpatients receiving thiazide diuretic therapy, approximately 10–40% are estimated to have serum potassium levels below 3.5 mEq/L; however, this degree of hypokalemia is generally well tolerated in individuals who were previously healthy [3].

In a recent study involving 21,616 adult patients presenting to the emergency department, the prevalence of hypokalemia was reported to be 0.4%, with a mean age of 73 years, and a predominance of male patients [4]. The incidence of hypokalemia was higher among patients with a history of kidney disease and cardiovascular disease [5].

In this case report, a 53-year-old male patient presented with complaints of generalised weakness and an inability to move both the upper and lower limbs, which occurred suddenly upon awakening from sleep. Before symptom onset, the patient had been able to perform daily activities typically. Based on the clinical history, other potential causes of weakness were excluded. The patient denied any history of trauma preceding the onset of symptoms, as well as a history of cardiac disease, diabetes mellitus, hypertension, hypercholesterolemia, or kidney disease. Long-term medication use was also denied. Additional symptoms, such as vomiting, diarrhoea, or excessive sweating, were not reported. However, the patient reported being hospitalised repeatedly at another hospital for similar complaints. On physical examination, motor strength was 2/5 in all four extremities (Medical Research Council scale), consistent with quadriparesis. Subsequent electrolyte evaluation revealed a serum potassium level of 1.4 mEq/L, indicating severe hypokalemia. In this case, the patient was diagnosed with hypokalemic periodic paralysis secondary to severe hypokalemia. This condition is believed to result from sodium–calcium channel dysfunction, leading to impaired electrical conduction in muscle cells. Consequently, muscle fibres fail to receive adequate stimulation, leading to reduced muscle contraction and functional paralysis. Large proximal muscles, such as those of the upper and lower limbs, are most commonly affected, whereas respiratory muscles are rarely involved [6].

Hypokalemia may result from several mechanisms, including a shift of potassium from the extracellular to the intracellular compartment and potassium loss through the gastrointestinal tract or urine, which is commonly associated with severe vomiting and diarrhoea. Other causes include genetic renal potassium-wasting disorders (e.g., Bartter syndrome and Gitelman syndrome), typically characterised by metabolic alkalosis, as well as renal tubular acidosis (RTA), which is associated with metabolic acidosis [7,8]. Based on the present case report, the patient did not experience severe nausea or vomiting and had no prior history of insulin use. In addition, the patient had been hospitalised previously with similar complaints. Following the diagnostic algorithm for hypokalemia, this case was determined to represent true hypokalemia, as supported by the clinical history, physical examination, and ancillary investigations.

Electrocardiographic examination revealed sinus rhythm with a heart rate of 60 beats per minute, flattened T waves predominantly in leads I, III, and aVR, ST-segment depression, and U waves, most prominent in leads V2 and V3. Urinary electrolyte analysis demonstrated increased urinary electrolyte excretion, with a transtubular potassium gradient of 6, indicating renal potassium loss. Renal ultrasonography showed no anatomical abnormalities. Arterial blood gas analysis revealed metabolic alkalosis, and the plasma magnesium level was 2.1 mg/dL. Taken together, these findings suggest that the patient most likely had Bartter syndrome.

Bartter syndrome is a rare congenital disorder characterised by abnormalities of the renal tubules. The prevalence of Bartter syndrome is estimated to affect approximately 1 in 1,000,000 individuals. This condition is caused by impaired potassium reabsorption due to dysfunction of the renal loop of Henle, particularly in the thick ascending limb, resulting in electrolyte wasting in the urine [9,10]. Bartter syndrome is associated with genetic mutations involving *SLC12A1*, *KCNJ1*, *CLCNKA*, and *CLCNKB*.

One differential diagnosis of Bartter syndrome is Gitelman syndrome. Gitelman syndrome is also a congenital renal disorder caused by genetic mutations that result in impaired electrolyte reabsorption in the distal convoluted tubule, affecting approximately 1 in 25,000 individuals [9,10]. The typical clinical features include hypokalemia, metabolic alkalosis, hypocalciuria, and hypomagnesemia. Historically, Bartter syndrome could be differentiated from Gitelman syndrome based on clinical findings such as age at onset, disease severity, the presence of hypercalciuria, a history of polyhydramnios, or growth retardation. However, advances in medical knowledge have shown that the onset of Bartter syndrome may occur later in life and that hypercalciuria may be absent in some cases [9,11]. In addition to clinical history, physical examination, and ancillary investigations, differentiation between Bartter syndrome and Gitelman syndrome can be supported by a thiazide test, which involves administration of hydrochlorothiazide (1 mg/kg body weight up to a maximum dose of 50 mg) for 1 week, followed by repeat urinary electrolyte analysis. In patients with Gitelman syndrome, urinary chloride excretion shows minimal change (< 2.3%) due to defects in the sodium–chloride cotransporter (NCCT). In contrast, patients with Bartter syndrome exhibit a measurable increase in chloride excretion relative to baseline. However, due to the increased risk of fluid loss, this test is not recommended. The definitive diagnosis of Bartter syndrome is established by molecular genetic testing to identify underlying genetic abnormalities. In this case, such testing was not performed due to the limited diagnostic resources at our institution [9].

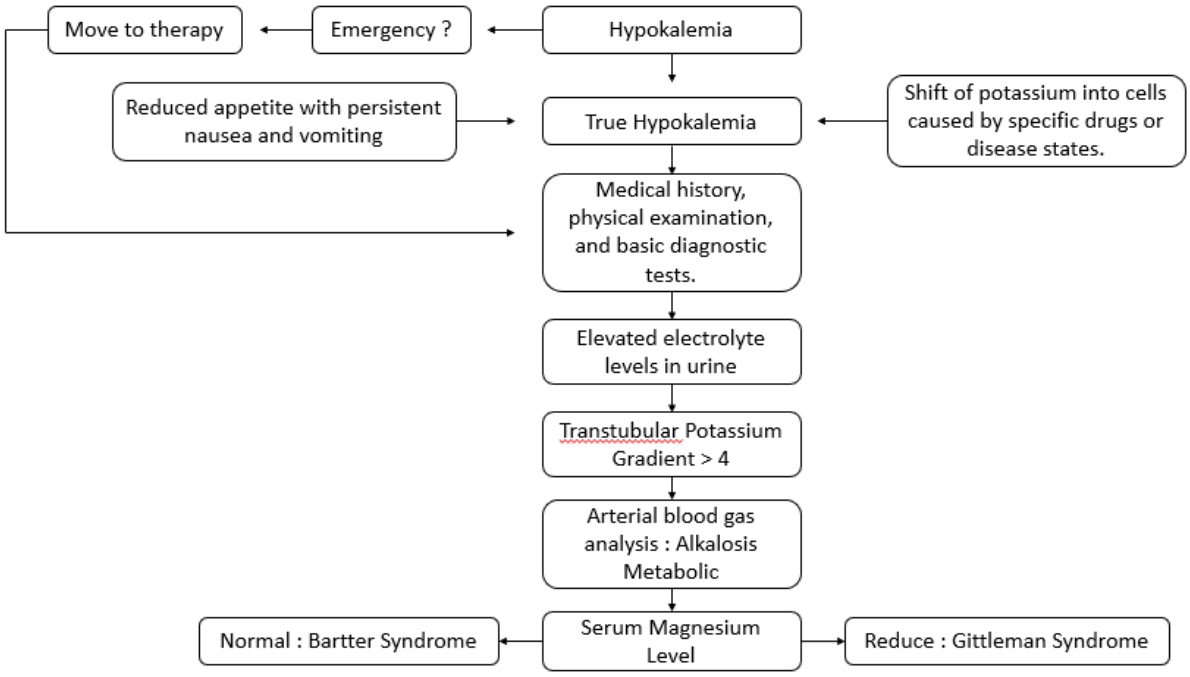

Figure 4: Diagnostic algorithm applied in this case [8]

In this case, the patient was admitted to the high care unit (HCU) for close observation. Treatment included intravenous fluid therapy with Ringer's acetate 500 mL combined with potassium chloride (KCl) 50 mEq administered over 12 hours, oral potassium slow-release (KSR) 1200 mg three times daily, spironolactone 25 mg once daily, and symptomatic therapy. During hospitalisation, serial electrolyte measurements were performed to monitor serum potassium levels. On the fifth day of treatment, the patient reported complete resolution of symptoms, and the serum potassium level increased to 3.7 mEq/L. The patient was subsequently discharged and scheduled for outpatient follow-up at the internal medicine clinic.

To date, there is no definitive therapy that can cure Bartter syndrome.[12] This is partly attributable to the limited available clinical evidence and the frequent difficulty in establishing a definitive diagnosis. Consequently, current treatment strategies vary considerably among clinicians and are primarily based on an understanding of renal physiology, clinical observations, and individual clinical experience. Potassium supplementation, administered either orally or intravenously, is used to correct low serum potassium levels. In addition, potassium-sparing diuretics, such as spironolactone or amiloride, may raise serum potassium levels and help correct metabolic alkalosis. [12]

This case contributes to clinical awareness of Bartter syndrome as a differential diagnosis for hypokalemia, especially in settings where genetic testing is unavailable. It demonstrates the value of comprehensive laboratory and clinical evaluation in managing rare tubular disorders.

4. Conclusion

Hypokalemia is defined as a condition in which the plasma potassium concentration is below 3.5 mEq/L. In a recent study involving 21,616 adult patients presenting to the emergency department, the prevalence of hypokalemia was reported to be 0.4%. The affected patients had a mean age of 73 years, were predominantly male, and most commonly presented with nonspecific or general symptoms.

Bartter syndrome is estimated to affect approximately 1 in 1,000,000 individuals in the general population. This condition results from impaired potassium reabsorption due to dysfunction of the renal loop of Henle, particularly the thick ascending limb, leading to excessive urinary electrolyte loss. Bartter syndrome generally has a favourable prognosis when appropriately managed and in the absence of complications involving other organs.

A thorough medical history, comprehensive physical examination, and appropriate diagnostic investigations are essential to establish an accurate diagnosis. In patients presenting with hypokalemia, potential life-threatening conditions should be assessed and managed first; emergency treatment must take priority over determining the underlying aetiology. In patients with a history of recurrent hypokalemia without apparent clinical symptoms—such as severe nausea and vomiting, loss of appetite, chronic diarrhoea, or the use of specific long-term medications—functional disorders of renal tubular reabsorption with a genetic basis, such as Bartter syndrome, should be considered. Bartter syndrome is associated with a favourable prognosis when patients adhere to regular medication use and undergo routine follow-up.

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