

A Practical Approach to Refractory Hypokalaemia: A Rare Presentation of Bartter Syndrome

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Abstract

Hypokalaemia is a common finding in hospitalized patients. In most cases the cause will be obvious. However, in a subgroup of patients the cause is occasionally uncertain and establishing the diagnosis may present difficulties and can become a challenge. In such cases, measurement of urinary indices e.g. urine potassium/creatinine concentrations along with blood acid/base parameters and assessment of urinary excretion potassium, chloride, calcium and creatinine have been employed in the differential diagnosis. This article presents a case report of an adult male with severe resistant hypokalaemia which an initial potassium level of one mmol/L and associated with limb weakness, metabolic alkalosis and hypercalciuria. His potassium level was refractory to oral and intravenous replacements. This patient showed a marked increase of serum potassium after administration of indomethacin. This patient had the full biochemical features of Bartter syndrome (BS), there was a severe hypokalaemia, with inappropriate urine K⁺ loss, with severe urinary chloride excretion associated with mild metabolic alkalosis and normal blood pressure. He had also hypocalcaemic hypercalciuria, His urine calcium/creatinine ratio was 0.26 which was indicative of hypercalciuria. His age and late symptom presentation would have suggested Gitelman disease but due to hypercalciuria, the clinical diagnosis of Bartter Syndrome was made. We describe here a case of Bartter Syndrome type 3 whose diagnosis was made in adult life. This patient is an unusual, possibly extreme case of BS. Being 30 years old at the time of diagnosis, he is by far the oldest patient so far described with BS. Calculating the urinary potassium/creatinine ratio, urinary chloride excretion and urinary calcium/creatinine ratio in conjunction with plasma acid/base values provided simple and reliable tests to distinguish Bartter syndrome from other differential diagnosis of hypokalaemia and to provide a proper management in the intensive care unit until further advanced investigations to be done.

Keywords: Bartter syndrome; Gitelman syndrome; Refractory hypokalaemia; Trans-tubular potassium concentration gradient; Urinary potassium-creatinine ratio; Urinary calcium-creatinine ratio

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Introduction

Hypokalaemia is a frequent finding in hospitalized patients. In most cases the cause of hypokalaemia is usually evident in the clinical history. However, in some patients the cause is occasionally not clear and diagnosis can become a challenge. In such cases, measurement of urinary indices e.g. urinary excretion potassium, chloride, calcium and creatinine along with blood acid/base parameters have been employed in the pathophysiological

diagnosis. This article presents a case report of an adult male with severe persistent hypokalaemia which was refractory to oral and intravenous replacements. This patient showed a marked increase of serum potassium after administration of indomethacin.

Case Report

A 30-year-old male; no previous medical history, presented to the emergency room complaining of bilateral lower limb weakness

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with fasciculations in hands and legs and inability to walk in the past one day. No history of trauma, fever, incontinence of stool or bowel, sensory complaints, or history of alcohol or drug abuse. In the triage area, a venous blood gas was obtained and showed severe hypokalaemia (potassium of 1 mmol/L) with mild metabolic alkalosis. He was shifted to the resuscitation area, put on pads with 2 large bore IV lines inserted, ECG obtained (**Figure 1**), and was started on potassium replacement.

On examination, BP: 98/40, Pulse: 90, SPO₂: 99% on room air, Temperature: 37 degrees Celsius, and on neurologic exam his power in the right lower limb was 3/5 and the left lower limb was 2/5. Otherwise sensation, deep tendon reflexes, and cerebellar signs were normal, pupils were 2 mm reactive briskly and his GCS was 15/15. His other examination parameters were normal.

Apart from severe hypokalaemia, there was hypophosphatemia (1.6 mg/dL), mild hypocalcaemia (8.6 mg/dl), normomagnesemia (1.7 mg/dL), hypochloraemia (86 mmol/L), increased random urine sodium (41 mmol/L), increased random urine potassium (58.9 mmol/L) and increased urine chloride (150 mmol/L); the lab workup including a septic work up, renal function, thyroid function tests, and plasma and urine osmolality was normal. The patient was admitted under care of the medical team and referred to the intensive care unit (ICU) for persistent low potassium and risk of arrhythmias due to prolongation of QTc. Also, ECG showed prolonged PR interval and evidence of hypokalaemia (U waves in leads II, V₂, V₃, and V₄, and ST-segment depression in leads II, III, aVF).

A renal echography of our patient showed two adjacent small stones in left renal calyx with mild pelvicalyceal dilatation with absence of nephrocalcinosis or other renal abnormalities.

While in the ICU, the patient received large doses of oral and IV potassium chloride (KCL) through a central line daily and it was noted that his weakness improves with the potassium replacement; however, his potassium blood levels were always low despite regular replacement (average 280 mmol daily) (**Figure 2**) and despite replacement of both magnesium and phosphate. The patient was persistently polyuria.

A differential diagnosis of refractory hypokalaemia was investigated. In such case, measurement of urinary indices such as excretory rate of potassium and random urine potassium/creatinine concentrations along with blood acid/base parameters have been employed in the differential diagnosis [1].

Urinary potassium/creatinine ratio is used as a simple appropriate index of potassium excretion and to establish a proper diagnosis and management in this medical emergency. His main biochemical parameters, as shown in **Table 1**, indicate that he had a severe hypokalaemia, with inappropriate urine K loss. This patient had the full biochemical features of Bartter syndrome: There was severe urinary chloride excretion (>150 mmol/l) associated with mild metabolic alkalosis and normal blood pressure, also he had hypocalcemic hypercalciuria. His urine calcium (4.1 mg/dL): Urine creatinine (16 mg/dL) ratio was 0.26 which was indicative of hypercalciuria. The main differential diagnosis is with Gitelman syndrome, which also known as the hypocalciuric variant of

Height: Unknown
PR: 0
QRS Dur: 110
QT: 428
QTc: 500
QT Disp: 54
Requested By: resus
Procedure Code: 3004000
Accession Number: 581691889
DX:
RX:

Weight: Unknown
P Axis: -127
QRS Axis: 112
T Axis: 52
HR: 82
BP:
Taken By:
Procedure Name: ELECTRO
CARDIOGRAM (ECG)
Undefined:

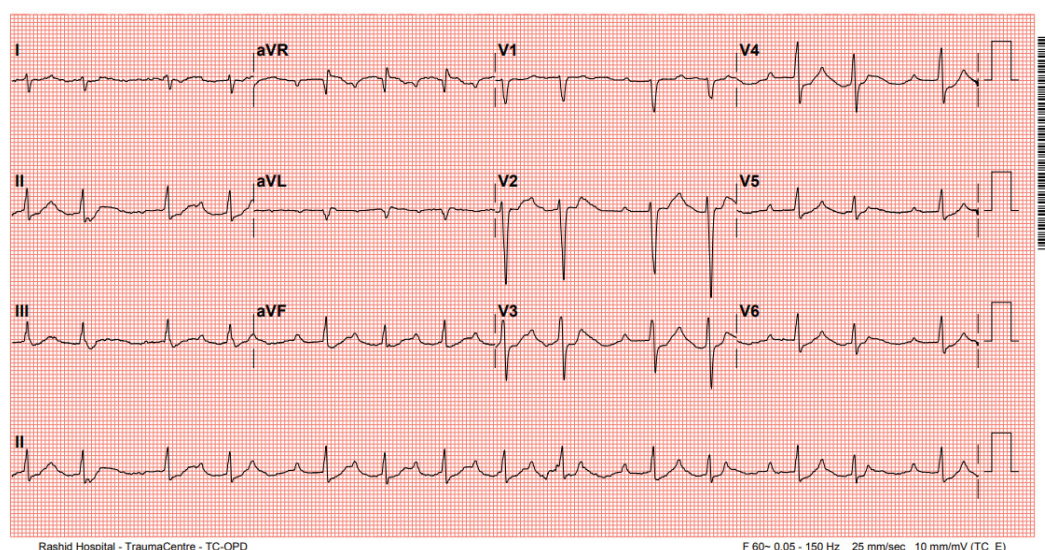


Figure 1 ECG showed prolonged PR interval and evidence of hypokalemia (U waves in leads II, V₂, V₃ and V₄, and ST-segment depression in leads II, III, aVF).

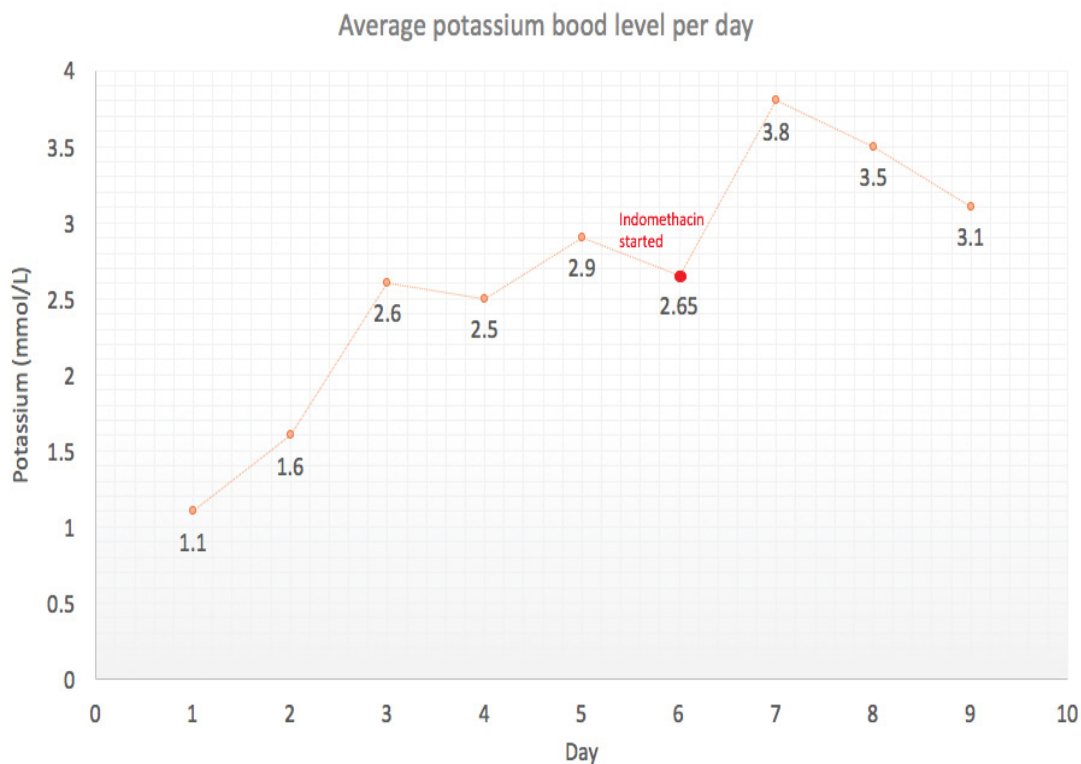


Figure 2 The average potassium blood level per day.

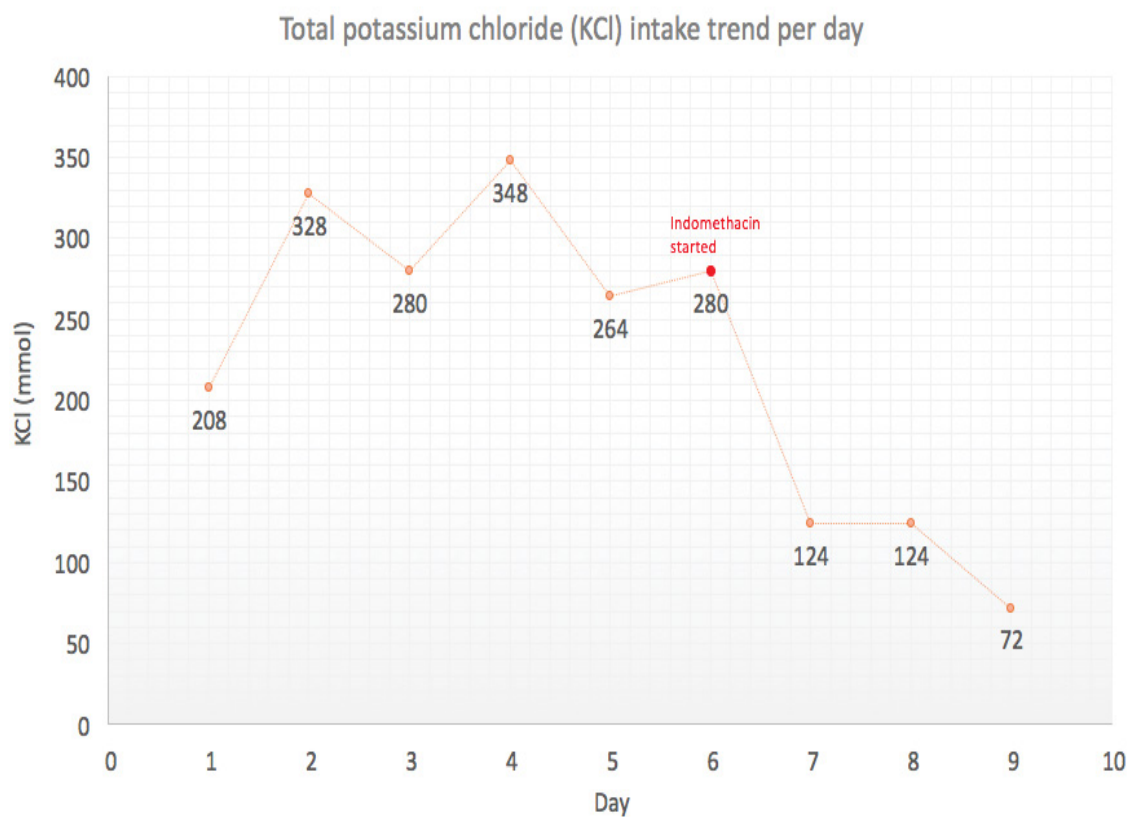


Figure 3 The total potassium chloride intake trend per day.

Table 1 The patient's main biochemical parameters.

Laboratory tests	Reference Range and Units	Result
Osmolality, Plasma (Measured)	275-295 mOsm/Kg.H ₂ O	281
Osmolality, Urine	50-1200 mOsm/Kg.H ₂ O	381
Phosphate Blood	2.7-4.5 mg/dL	1.6
Serum Magnesium	1.7-2.55 mg/dL	1.7
Blood Urea	12-40 mg/dL	20
Chloride, Random Urine	mmol/L	150
Chloride, Serum	98-108 mmol/L	86
Creatinine, Random Urine	20-320 mg/dL	16
Serum Creatinine	0.7-1.2 mg/dL	1
Serum Sodium	136-145 mmol/L	139
Sodium, Random Urine	mmol/L	41.0
Serum Potassium	3.3-4.8 mmol/L	1.6
Potassium, Random Urine	mmol/L	58.9
Free T4	11.0-22.0 pmol/L	25.1
Free T3	2.8-7.1 pmol/L	6
TSH	0.3-4.2 uIU/mL	2.9
Calcium	8.9-10.2 mg/dL	8.6
Calcium, Random Urine	mg/dL	4.1
CPK	0-190 u/L	2166

Bartter syndrome but due to hypercalciuria, the clinical diagnosis of Bartter syndrome was made.

The patient had a resistant severe hypokalaemia in spite of large potassium replacement. Hypokalaemia was marked improved after administration of indomethacin in dose of 50 mg twice daily. After indomethacin, his potassium levels were maintained at a normal level and his daily KCL requirement decreased (**Figure 3**). The patient was shifted to the medical ward and later discharged to home and for advanced further workup of Bartter Syndrome to be done in the outpatient clinic.

This patient is an unusual, possibly extreme case of Bartter syndrome, being 30 years old at the time of diagnosis, he is by far the oldest patient so far described with Bartter syndrome. His age and late symptom presentation would have suggested the more Gitelman disease, but hypercalciuria and clearance data suggested Bartter syndrome.

Discussion

Hypokalaemia is a common clinical problem which the etiological diagnosis can frequently be based on the patient history and the clinical situation [1]. The cause can usually be determined from the history such as hypokalaemia due to vomiting, diarrhoea, or diuretic use and no further investigation will be necessary. However, in a subgroup of patients the cause is occasionally uncertain and establishing the diagnosis may present difficulties [2].

In situations where the cause of hypokalaemia is not clear, measurement of urinary potassium excretion and assessment of blood pressure and acid-base values are often helpful [3].

The causes of hypokalaemia can be simply divided into two categories: The one condition includes diseases causing renal potassium wasting such as diabetic ketoacidosis, Cushing's syndrome, primary hyperaldosteronism, Bartter syndrome, Gitelman syndrome, hypomagnesemia, and renal tubule acidosis, as well as intake of loop and thiazide diuretics, which results in K loss by the kidneys; and the other consists of those leading to extra-renal excretion or transient shifting of K into cells, as in thyrotoxic periodic paralysis, exogenous insulin infusion, hyperinsulinemia, vomiting, and diarrhoea [4].

The traditional approach to differentiate between renal and extrarenal causes of hypokalaemia is based on urinary potassium excretion measured in 24 hours urine samples or random urinary potassium concentration values. A urinary potassium concentration that is more than 15 to 20 mmol/day or more than 15 to 20 mmol/L suggests that there are renal causes for potassium wasting. Hypokalaemia in which these values are less than 15 mmol/day or 15 mmol/L respectively indicates that there are extrarenal causes for potassium depletion. Obtaining a 24 hours potassium excretion rate is not practical in a medical emergency because potassium replacement must be given promptly which can compromise the accuracy of 24 hours urinary potassium [5].

A spot urine collection before potassium replacement should be used to help for establishing the diagnosis [5]. In our patient there was high urinary potassium concentration (58.9 mmol/L) that indicates there are renal causes for potassium loss. However, random measurements may be misleading as the urine potassium concentration is determined by both the amount of potassium in the urine and the urine volume [6]. Usually there are many overlapping values of the spot urinary potassium concentration as polyuria is common in patients with hypokalaemia. The polyuria may be due to thirst or defective renal concentration in patients with chronic hypokalaemia which leads to a low value of the urinary potassium concentration even if a significant renal potassium loss is present. Therefore, a more reliable test is required [5].

The trans-tubular potassium concentration gradient (TTKG) is an index reflecting the degree of potassium excretion in the cortical collecting ducts of the kidneys and indirectly assessing mineralocorticoid bioactivity in patients who have hypo- or hyperkalaemia [7,8]. Measurement of the TTKG is considered superior to measurement of urinary potassium alone for assessing the contribution of renal excretion to potassium levels. (9) TTKG are useful in the pathophysiological differential diagnosis of hypokalaemia [1].

The trans-tubular potassium gradient estimates the ratio of potassium in the tubular fluid at the end of the cortical collecting tubule to that in the peritubular capillaries and can be estimated from:

$$TTKG = \frac{(\text{urine potassium} \times \text{Osmolality plasma})}{(\text{plasma potassium} \times \text{Osmolality urine})}$$

For this formula to be accurate, urine osmolality must exceed plasma osmolality and urine sodium should be greater than 25 mmol/L [7,8]. In our patient, the urine osmolality was 381 mOsm/

kg/H₂O which exceed the plasma osmolality of the patient (281 mOsm/kg/H₂O) and the urine sodium was 41 mmol/L (greater than 25 mmol/L).

The normal renal response when hypokalaemia is due to non-renal causes is a trans-tubular potassium gradient less than 2 which indicates intracellular shift of potassium. while a trans-tubular potassium gradient more than 3 indicates hypokalaemia of renal origin (renal potassium wasting) [9]. In our patients there was a TTKG was very high (27.15) which is indicative of increased secretion of K⁺ in the cortical collecting ducts.

However, there are recent studies that advise to shift in emphasis from the TTKG to other variable reflecting potassium excretion e.g. the urinary potassium/creatinine ratio due to intrarenal urea recycling which leads to a higher rate of renal excretion of potassium [10,11].

The other variable reflecting potassium excretion, the urinary potassium/creatinine ratio (U(K/Cr)), has been used to evaluate the cause of hypokalaemia [5]. Measurement of the urinary potassium/creatinine ratio is preferred because it is not influenced by the urine volume. U(K/Cr) may be a valuable marker to distinguish between renal and extra-renal K loss [4,6].

Many studies have shown that the urinary potassium/creatinine ratio can be used to differentiate between hypokalaemia caused by renal potassium loss and hypokalaemia caused by extrarenal potassium depletion, as found in ion channelopathy hypokalemic periodic paralysis, by intracellular shift of serum potassium into skeletal muscle [4,6,12].

The efficacy of the urinary potassium/creatinine ratio was evaluated in a previous study of forty-three patients with severe hypokalaemia associated with paralysis. The urinary potassium/creatinine ratio was able to differentiate between the thirty patients with hypokalemic periodic paralysis (HPP) (whose hypokalaemia was caused by an internal shift of extracellular potassium into the cells) and the thirteen patients with hypokalaemia mostly due to renal potassium wasting (Ten of them had renal tubular acidosis or Gitelman syndrome). The urinary potassium/creatinine ratio was significantly lower in the patients with periodic paralysis. Hypokalaemia and paralysis may be due to short term shift of potassium into cells in HPP or due to a large deficit of potassium in non-HPP [5].

Failure to differentiate between hypokalemic periodic paralysis from non hypokalemic periodic paralysis may lead to improper management. It is critical to distinguish hypokalaemia caused by redistribution, to avoid the risk that these patients may have rebound hyperkalaemia after potassium replacement due to reversal of the shift of extracellular potassium into the cells [5].

It is important to make the diagnosis promptly because different therapies are required for each type. The differential diagnosis can be challenging as the clinical features of HPP and non-HPP are almost indistinguishable. Many patients with non-HPP have been misdiagnosed as having HPP [5].

The diagnostic cut off value for the potassium/creatinine ratio was approximately 1.5 mmol/mmol (some authors say 2.5 mmol/mmol or 22 mmol/g). In addition, patients with periodic paralysis

had normal acid-base balance, while patients without periodic paralysis often had metabolic alkalosis or metabolic acidosis [5].

In our patient there was severe hypokalaemia associated with limb weakness (lower limb power was 2/5). On calculation of the potassium-creatinine ratio, it was very high (42 mmol/mmol) (368 mmol/g) which is indicative of severe renal potassium loss.

Measurement of urinary indices such as random urine (K/Cr) concentrations along with blood acid-base parameters have been employed in the pathophysiological diagnosis [1].

A random urinary potassium/creatinine ratio (K/Cr) less than 1.5 (mmol/mmol) suggests a transcellular potassium shift into cells, poor potassium intake or gastrointestinal potassium loss. If hypokalaemia is associated with paralysis, we should consider familial, sporadic or thyrotoxic periodic paralysis. Metabolic acidosis with urinary K/Cr ratio less than 1.5 suggests lower gastrointestinal loss due to diarrhoea, enteric fistula or laxative abuse. However, some patients with diarrhoea due to laxative abuse develop metabolic alkalosis [3,13,14].

While metabolic acidosis with urinary K/Cr ratio more than 1.5 is often due to diabetic ketoacidosis or distal renal tubular acidosis type 1 or type 2 [3].

Our patient had persistent severe hypokalaemia due to renal potassium loss (high K/Cr ratio more than 1.5) associated with metabolic alkalosis and normal arterial blood pressure and thyroid function, then our differential diagnosis is narrowing. By measurement of urinary chloride level, we can able to differentiate the causes of hypokalaemia due to renal loss with metabolic alkalosis. Patients with hypokalaemia due to renal loss (high K/Cr ratio) with metabolic alkalosis can be divided into two groups based on their urinary chloride excretion. The possible causes of low urine chloride level (<10 mmol/L) are vomiting, post hypercapnic alkalosis, nasogastric suction and congenital chloride diarrhoea [13,14].

Patients with high urine chloride level (>20 mmol/L) can be separated into two groups based on their arterial blood pressure. Patients with high urinary chloride excretion (>20 mmol/L) and high arterial blood pressure include primary and secondary hyperaldosteronism, Cushing syndrome, renin secreting tumour, adrenal carcinoma, congenital adrenal hyperplasia, renal artery stenosis, malignant hypertension, diuretic therapy in patients with essential hypertension, apparent mineralocorticoid excess, or Liddle syndrome [13].

While patients with high urinary chloride excretion (>20 mmol/L) and normal arterial blood pressure are caused by diuretics, Gitelman syndrome and Bartter syndrome. Diuretic therapy is the most common cause of hypokalaemia which can be excluded by history. Diuretic induced hypokalaemia is usually but not always associated with a mild to moderate metabolic alkalosis [13]. In our patient, there was severe urinary chloride excretion (>150 mmol/L) associated with metabolic alkalosis and normal arterial blood pressure.

The primary defect in both Gitelman syndrome and Bartter syndrome is an impairment in one of the transporters involved in sodium chloride reabsorption in the distal tubule and Henle's

loop, respectively. The tubular defects in sodium chloride transport produce a clinical disorder that appears similar to that seen with chronic ingestion of a loop diuretic (mimicking Bartter syndrome) or a thiazide diuretic (mimicking Gitelman syndrome) [15].

Bartter syndrome versus Gitelman syndrome

Bartter syndrome (BS) is a hereditary condition transmitted as an autosomal recessive (Bartter type 1 to 4) or dominant trait (Bartter type 5). There is an inherited defect in the renal tubules that causes low potassium levels, low chloride levels, which in turn causes metabolic alkalosis with varying degrees of hypercalciuria [16,17]. It is a consequence of abnormal function of the kidney, which become unable to properly regulate the volume and composition of body fluids due to mutations in renal tubular transporters in the thick ascending limb of loop of Henle which lead to defective reabsorption of sodium and chloride. A first consequence of the tubular defect in Bartter Syndrome is polyuria [16]. These patients appear similar to that seen with patients of chronic ingestion of a loop diuretic. Bartter Syndrome is characterized by hypokalaemia of renal origin with renal potassium and salt wasting, metabolic alkalosis, hypercalciuria, normal arterial blood pressure, high plasma renin and aldosterone levels [13].

Type 3 Bartter syndrome patients have the mildest presentation [17,18]. The classical form or type 3 Bartter syndrome is associated with hypomagnesemia in 20% of cases and normal or increased calciuria. This form is related to mutations of CLCNKB gene encoding for a chloride channel in Henle's loop [15,19]. Clinically, Bartter Syndrome type 3 and 5 appear to be less severe than types 1 and 2, despite similar biochemical indices [16].

Patients with Bartter syndrome types 1, 2 and 4 presents at a younger age than classic Bartter syndrome type 3. Patients with classic Bartter syndrome type 3 present later in life and may be sporadically asymptomatic or mild symptomatic [16,18].

Most patients of Bartter Syndrome are usually symptomatic early in life, they present with symptoms often severe in the neonatal period, although occasional patients, mostly Bartter Syndrome type 3 and rare patients with Bartter Syndrome type 4 and 5, may continue into adulthood with few if any manifestations [16,20].

In contrast, Gitelman Syndrome (GS) always presents in adult life. It is a much more common disease and less severe than Bartter Syndrome [6]. These patients appear similar to that seen with patients of chronic ingestion of thiazide diuretics. It is due to a defect in the gene encoding for the thiazide sensitive sodium/chloride cotransporter and it is associated with hypocalciuria and hypomagnesemia [13]. The degree of hypokalaemia is usually mild to moderate in Gitelman Syndrome. Recently, however, more patients with Gitelman Syndrome have been found with a degree of hypokalaemia that is similar to that in patients with Bartter syndrome. Many patients of Gitelman Syndrome in Asia present with paralysis related to severe hypokalaemia [5].

The diagnosis of Gitelman Syndrome is based on the following criteria: Hypokalaemia due to renal potassium loss, hypomagnesemia of renal origin, and hypocalciuria with a urine

calcium/creatinine ratio less than 0.1 mmol/mmol (0.2 mg/mg) [5]. No evidence supports the need for 24 hours urine collection, spot urine samples are usually sufficient to establish the diagnosis [21].

Urinary calcium excretion is important because it differentiate between the two syndromes. In contrast to the hypocalciuria of Gitelman syndrome, Bartter syndrome patients are often documented to have hypercalciuria [18].

Although 24 hours collection of urine calcium is best, random urine calcium measurement in relation to creatinine can be performed. The normal reference for the urine calcium (mg/dl): Urine creatinine (mg/dl) ratio is less than 0.14. Values exceeding 0.2 are found in patients with hypercalciuria [22].

In our patient, the urine calcium (mg/dl): Urine creatinine (mg/dl) ratio was 0.26 which was indicative of hypercalciuria along with severe urinary chloride excretion (150 mmol/L) and persisting severe hypokalaemia due to renal potassium wasting. The main differential diagnosis is with Gitelman Syndrome, also known as the hypocalciuric variant of Bartter Syndrome, but due to hypercalciuria the clinical diagnosis of Bartter syndrome was made.

Hypercalciuria predisposes Bartter Syndrome patients for nephrolithiasis and nephrocalcinosis (calcification of the renal tissue) [16]. A renal echography of our case showed two adjacent small stones in left renal calyx with mild pelvicalyceal dilatation. Thus, increased urine calcium excretion may be considered as the possible risk factor for stone formation in our patient.

The persistent steady form of hypokalaemia in BS and GS may suddenly become life threatening under certain aggravating conditions. Physicians need to be cognizant of such renal tubular disorders and promptly treat patients at risk [18].

Distinguishing between BS and GS is not always straightforward due to phenotypic variance. The genetic diagnosis is now possible but there are several limitations including unavailability and cost. As mentioned before, GS is typically associated with hypocalciuria and BS with hypercalciuria, although some patients with type 3 Bartter syndrome have normal urinary calcium excretion [18,20,23].

At the present, Bartter Syndrome can't be cured, and treatment is mainly directed for correction of dehydration and electrolytes disturbance, i.e. hypokalaemia and possible hypomagnesemia. Simply increasing the intake of single electrolytes has a little benefit, since urinary excretion increases proportionally [16].

The focus of chronic therapy of BS and GS patients includes electrolytes replacement as well as inhibition of secondary increases of prostaglandin production and/or the renin-angiotensin-aldosterone axis, which exacerbate the urinary electrolyte loss. Hypokalaemia also induces prostaglandin production [18].

Indomethacin is a nonsteroidal anti-inflammatory drug which being used in Bartter syndrome to help improve growth in children and decrease urinary potassium excretion. Indomethacin has been shown to abolish vasopressor insensitivity to angiotensin II

and to lower plasma renin activity and aldosterone concentration in a patient with Bartter's syndrome [24]. Indomethacin has been shown to inhibit prostaglandin synthetase, which support the hypothesis that prostaglandin excess is a basic pathogenic mechanism in Bartter's syndrome [25].

Indomethacin significantly reduced eGFR, plasma renin concentration and increases plasma potassium concentration. Indomethacin also decrease the rate of urinary excretion of sodium and inorganic phosphate [25,26].

Indomethacin can lead to improvement in laboratory parameters typically seen in BS patients. The dose of indomethacin can be as high as 2-3 mg/kg/day. Long term gastrointestinal side effects of indomethacin including chronic gastritis and gastric ulcers can be dose limited. Cyclooxygenase 2 selective inhibitors may be considered as an alternative option. The same response to indomethacin is not seen with GS because patients with GS do not typically show the increased prostaglandin production seen in patients with BS [18].

Our patient had a resistant severe hypokalaemia in spite of large potassium replacement. Hypokalaemia was marked improved after administration of indomethacin in dose of 50 mg twice daily. Our patient showed marked increase of serum potassium after administration of indomethacin.

This patient is an unusual, possibly extreme case of Bartter syndrome. His diagnosis remained unsuspected until adult life, being 30 years old at the time of diagnosis, he is by far the oldest patient so far described with Bartter syndrome. His age and late symptom presentation would have suggested Gitelman disease, but hypercalciuria suggested Bartter syndrome.

In summary, in our case report, there was a persisting severe hypokalaemia due to renal potassium wasting and to establish a proper diagnosis and management in this medical emergency, urinary potassium-creatinine ratio is used as a simple appropriate index of potassium excretion. Hypercalciuria along with high urinary chloride excretion in conjunction with plasma acid-base values help for the clinical diagnosis of Bartter syndrome (BS). Our case suggests that BS3 may be presenting in adult life, as in G.S. Hypercalciuria and clearance data should allow differentiating it from the more common Gitelman disease.

Conclusion

Calculating the urinary potassium/creatinine ratio, urinary chloride excretion and urinary calcium/creatinine ratio in conjunction with plasma acid/base values provided simple and reliable tests to distinguish Bartter syndrome from other differential diagnosis of hypokalaemia and to provide a proper management in the intensive care unit until further advanced investigations to be done.

Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

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