CASE REPORT

A Case of Persistent Hypokalemia

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ABSTRACT

Case of 51-year-old female patient having persistent features of hypokalemia which on further evaluation came to diagnose as Gittelmen syndrome.

Keywords: Hypokalemia , Electrolyte, Gitelman syndrome (GS)

INTRODUCTION

Hypokalemia is characterized by a decline in plasma K+ concentration of <3.5 millimole. It can be driven on by K+ loss from the kidneys and gastrointestinal system, as well as by K+ redistribution between cells and the extended credit facility. Drugs like diuretics are a very common cause of hypokalemia.[1]

Electrolyte disturbances, such as hypokalemia, are frequently encountered in hospitalized patients. Persistent hypokalemia typically results from gastrointestinal potassium loss, renal potassium ion loss, or inadequate or lacking replenishment. Channelopathies such as Gitelman, Liddle syndrome, and Barter syndrome are more prevalent among the reasons of urinary loss of potassium in RTA. Gitelman syndrome (GS) is an Autosomal Recessive disorder. It manifests as salt-wasting renal tubulopathy that results in marked hypocalcemia, hypomagnesemia, persistent hypokalemia, and metabolic alkalosis.

CASE REPORT

A 51-year-old female patient came with the main complaints of significant loss of appetite for 3 days prior admission and weakness in all four limbs whose onset is acute and progressive associated with inability to walk or move her legs, no association to any urinary complaints or any pain and lacked any aggravating or reliving elements.[2] There was no medical history of any trauma, posture discomfort, radiating pain, headache, diarrhea, nausea, or vomiting or of symptoms that would wax and wane. On examination, patient was conscious but visibly distressed and anxious; however, she is still cooperative and oriented to time, place, and person and was vitally stable. On neurological examination, hypotonia presents in all four limbs with the power of 2/5 in all of them. Furthermore, Deep tendon reflexes were diminished in all four limbs too.

Plantar reflex was B/L equivocal. Sensory examination revealed impaired vibration, pain, and temperature sensations in lower limbs. Cranial nerve examination was found to be normal.

Electrocardiogram was suggestive of T wave flattening and inversion, prominent U waves in all chests leads suggestive of hypokalemia [Figure 1].

On Investigations

Sr. sodium	142 mEq/l
Sr. potassium	1.7 mEq/l
Sr. chloride	122 mEq/l
Urinary potassium	29.80 mmol
Sr. magnesium	1.7 mEq/l
Sr. calcium	6 mg/dl
Urine calcium	10.8 mmol
Urine creatinine	7.06 mmol

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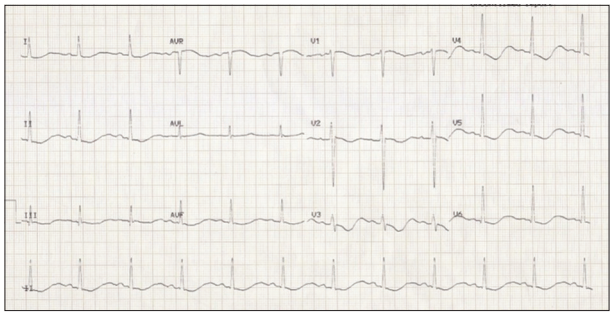

Figure 1: Electrocardiogram showing hypokalemia changes

Hypokalemia, hypocalcaemia, and hypomagnesemia were present.

On urine examination, hyperkalemia with raised TTKG (16) is seen.

ABG metabolic alkalosis is present. As urine chloride levels are more than 20 mEq/l, urine calcium creatinine ratio was calculated which came <0.15 s/o GS. Chest X-ray showed no abnormal findings.[3]

DIAGNOSIS

Clinical findings	In our patient
Weakness, cramps	+
Constipation	+
Abdominal pain	-
Polyuria	-
Growth retardation	-

Biochemical findings	In our patient
Metabolic alkalosis	+
Hypokalemia	+
Hypomagnesemia	+
Hypocalcemia	+

DISCUSSION

GS belongs to an autosomal recessive category. It is a salt-wasting tubulopathy which is characterized by renal potassium wasting, hypokalemia, hypocalciuria, hypomagnesemia, and hyper reninemic hyperaldosteronism along with metabolic alkalosis. It is also known as familial hypokalemia-hypomagnesemia. It has prevalence of 1–10/40,000. Usually GS considered as a benign tubulopathy which is mostly presenting during adolescence or adulthood. Often, the condition may remain asymptomatic or present with mild and non-specific symptoms. While if someone presented with chondrocalcinosis, tetany, seizures, and ventricular arrythmia which is early in onset (before age of six), all such patients considered as severe category and need to be evaluated on time.[4] Treatment of GS is symptomatic. Potassium supplementation is given either through oral route in the form of potassium chloride salts or by intravenous route, according to patients condition and acceptance. Furthermore, magnesium supplementation is crucial in correcting hypokalemia.[5]

CONCLUSION

Every patient presenting with features of hypotonia and weakness must be evealuated for dyselectromia. In the above case repeated follow up of patient's enlighten the persistent hypokalemia features alongwith metabolic abnormalities which leads to diagnosis of Gittelman syndrome.

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