Original Research Paper

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Medicine

GITELMAN SYNDROME PRESENTING AS ACUTE RECURRENT HYPOKALEMIA

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ABSTRACT Gitelman Syndrome is also referred to as familial hypokalemia hypomagnesemia.It is characterised by Hypokalemic metabolic alkalosis in combination with hypomagnesemia and low urinary calcium excretion.Gitelman syndrome is transferred as an autosomal recessive trait.Most cases are diagnosed during adolescence or adulthood.We presented the case of 23year old male with no co-morbidities who presented to casualty with persistent recurrent hypokalemia. There was no history of drugs taken (even potassium supplements).

KEYWORDS :

Complementary Evaluation Revealed-
- Hypokalemia- 2.1meq/l
- Hypomagnesemia-1.4meq/l
- Metabolic alkalosis-7.45
- Bicarbonate-28mmol/l
- Increased urine chloride excretion-117.8mmol/l
- Increased urine sodium excretion-109mmol/l
- Decreased urine calcium excretion-7.84mg/dl

Renal function and renal ultrasound was normal.Thus,a diagnosis of Gitelman was established.The aim of our article is to remind that Gitelman syndrome is a differential diagnosis of Persistent Hypokalemia.

Background-
Hypokalemia is a frequent electrolyte disturbance, particularly in hospitalized patients.In most cases of hypokalemia,the cause is pretty straight forward, mostly results from gastrointestinal losses or urinary losses.Gitelman syndrome is autosomal recessive salt losing renal tubulopathy causing hypokalemia and metabolic alkalosis.

Case presentation-
We presented the case of 23year old male with no co-morbidities presenting with recurrent persistent hypokalemia who came to MGM casualty.He had presented to MGM with same complaints 3months back.He came with complaints of-
- Weakness in bilateral upper limbs and lower limbs since 7days
- Inability to get up from sitting position since 7days
- Inability to walk since 7days
- Tingling numbness in bilateral upper limb and lower limb since 7days

No history of any drugs taken

No history of diabetes mellitus/hypertension/hypothyroidism

No significant past medical history

No significant familial history

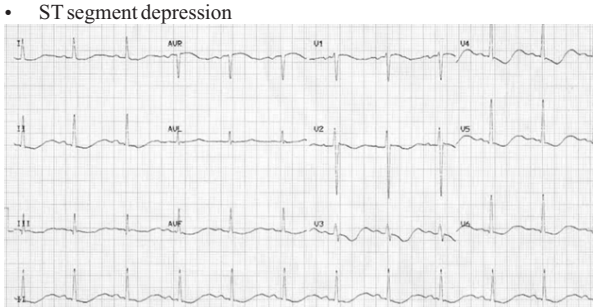
On physical examination,

He had a Blood Pressure of 130/80mmHg and regular pulse rate of 82beats/min.There were mo signs of peripheral oedema.On systemic examination, Cardiopulmonary examination was normal.The rest of the physical examination was also normal.

On arterial blood gas analysis,he presented with metabolic acidosis(ph, bicarbonate,PCO2)

Biochemical Analysis revealed-
- Serum potassium-2.1meq/l
- Serum Magnesium-1.4meq/l
- Serum Creatinine-0.7mg/dl
- Blood Urea-37mg/dl
- Urine chloride excretion-117.8 mmol/l
- Urine sodium excretion-109 mmol/l
- Urine calcium excretion-7.84mg/dl

ECG shows-
- Presence of U waves
- Long PR interval
- ST segment depression



Renal Ultrasound was normal
Based on the findings of Hypomagnesemia, Hypokalemia, Metabolic alkalosis and Hypocalcuria,the diagnosis of Gitelman Syndrome can be established.

Treatment-The patient was given IV Inj Kesol supplementation with Syrup Kesol 10ml TDS with Tab Magvion 400mg 1/2BD.

Outcome and FollowUp
1month later he was asymptomatic and with Serum potassium of 4.2meq/l and Serum magnesium of 1.8meq/l.

DISCUSSION-
Chronic Hypokalemia is common clinical problem with life threatening manifestations.Our patient had history of persistent hypokalemia with hypomagnesemia and metabolic alkalosis.

Vomiting and use of Diuretics are excluded by the high urine chloride levels obtained and negative history of Diuretic use respectively.

The remaining differential diagnosis were the genetic disorders of Gitelman and Barter's syndrome.Barter's was unlikely because it has early onset with urinary calcium excretion increased and normal or mildly reduced serum magnesium levels.

Hence ,our final diagnosis was Gitelman syndrome,an autosomal recessive salt losing renal tubulopathy. In vast majority of the cases, Disease is due to inactivating mutations in the gene that encodes renal thiazide sensitive sodium chloride co-transporter(NCC) present in epithelial cells of distal convoluted tubule. This is characterised by Hypomagnesemia, Hypocalciuria and metabolic alkalosis. Clinical

manifestations are similar to the prolonged administration of thiazide diuretics. The diagnosis of Gitelman syndrome is based on the clinical symptoms and biochemical abnormalities which include hypomagnesemia, metabolic alkalosis, hypocacuria.DNA mutation analysis for the gene responsible for Gitelman Syndrome may confirm the diagnosis.Progression to renal insufficiency is extremely rare.

Concerning treatment, supplementation with magnesium along with high sodium and high potassium diet.If symptomatic hypokalemia is not corrected,it can be the associated drugs that antagonise aldosterone activity or block sodium eNaC in collecting duct.An option is combination of amilorode,spironolactone or eplerenone and potassium chloride.

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