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Recurrent Hypokalemia in Apatient of Gitelman Syndrome: A Case Report

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I. INTRODUCTION:

Gitelman syndrome (GS), also referred to as familial hypokalemia-hypomagnesemia, is characterized by hypokalemic metabolic alkalosis in combination with significant hypomagnesemia and low urinary calcium excretion. The prevalence is estimated at approximately 1:40,000 and accordingly, the prevalence of heterozygotes is approximately 1% in Caucasian populations, making it one of the most frequent inherited renal tubular disorders. In the majority of cases, symptoms do not appear before the age of six years and the disease is usually diagnosed during adolescence or adulthood. Transient periods of muscle weakness and tetany, sometimes accompanied by abdominal pain, vomiting and fever are often seen in GS patients. Paresthesias, especially in the face, frequently occur.[1]

II. CASE REPORT:

A 34 years old male patient was admitted in hospital with chief complaints of gradual weakness of both upper limbs and lower limbs since 2 days with no symptoms of vomitings, diarrhea,palpitations.The same condition occurred in his previous two admissions to hospital,dischargedwith supplementation of potassium as his treatment.Initial laboratory examination revealed Metabolic Alkalosis with Serum Potassium levels of 2.4mEq/L and Magnesium 1.4mg/dL.Further laboratory examination in the ward showed 24hour urine potassium of 44mEq, urinary potassium creatinine ratio of 2.6.

He was given potassium chloride and magnesium infusion and his symptoms gradually improved.He was discharged with potassium and magnesium supplementation and remains symptoms free until now.

III. DISCUSSION:

Gitelman syndrome (GS) is a rare, salt-losing tubulopathycharacterized by hypokalemic metabolic alkalosis with hypomagnesemia and hypocalciuria. The disease is recessively inherited, caused by inactivating mutations in the SLC12A3 gene that encodes the thiazide-sensitive sodium-chloride cotransporter (NCC). GS is usually detected during adolescence or adulthood, either fortuitously or in association with mild or nonspecific symptoms or both. The disease is characterized by high phenotypic variability and a significant reduction in the quality of life.[2]

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The plasma biochemical picture is characterized by hypokalemia, hypomagnesemia, hypocalciuria, metabolic alkalosis and hypereninemichyperaldosteronism.[3]

GS is usually managed by a liberal salt intake together with oral magnesium and potassium supplements.[2]

IV. CONCLUSION:

Gitelmansyndrome is a disease with features of Hypokalemia, Metabolic alkalosis, hypomagnesemia and hypocalciuriathat is often under diagnosed , adequate approach of diagnosis and treatment would prevent the recurrent episode of hospitalisation hence reduce morbidity.

LABORATORY RESULTS OF PATIENT:

Examination	Results	Units	References
Sodium (Na)	130	mEq/L	135-145
Potassium(K)	2.4	mEq/L	3.30-5.40
Chloride (Cl)	96	mEq/L	94-110
Magnesium (Mg)	1.4	mg/dL	1.70-2.55
Calcium (Ca)	7.7	mg/dL	8.4-10.2

Random urinary Calcium	0.1	mg/dL	
Random urinary Creatinine	8.8	mg/dL	Ratio 0.011
Serum osmolarity	265	mOsm/Kg	275-295
Urine osmolarity	61	mOsm/Kg	250-900
24hour urine sodium	60	mEq/24hours	30-220
24hour urine potassium	57	mEq/24hours	25-100
24hour urine chloride	80	mEq/24hours	120-250

REFERENCES

[1]. ANine VAM Knoers, Elena N Levtchenko Orphanet journal of rare diseases 3 (1), 1-6, 2008

[2]. Anne Blanchard, DetlefBockenhauer, Davide Bolignano, Lorenzo A Calo, Etienne Cosyns, Olivier Devuyst, David H Ellison, Fiona E KaretFrankl, Nine VAM Knoers, Martin Konrad, Shih-Hua Lin, Rosa Vargas-PoussouKidney international 91 (1), 24-33, 2017

[3]. Giorgio Graziani, Carlo Fedeli, Luca Moroni, Laura Cosmai, Salvatore Badalamenti, Claudio Ponticell QJM: An International Journal of Medicine 103 (10), 741-748, 2010