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Case Report

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Acute Kidney Injury Secondary to Rhabdomyolysis in Case with Gitelman Syndrome

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Abstract

Gitelman syndrome (GS) is a genetically transmitted tubulopathy. It is caused by mutation in the thiazide-sensitive Na-Cl cotransporter–coding gene, SLC12A3. In this case study, we have discussed a patient diagnosed with GS and referred with muscular weakness and cramping complaints due to discontinuing potassium replacement in the follow-up. The patient was diagnosed with rhabdomyolysis and acute kidney injury secondary to hypokalemia upon determination of 2.14 mEq/L potassium, 27.610 U/L creatine kinase, and 3.09 mg/dL creatinine in further examination. Therefore, NaCl 100 cc/h isotonic was administered to the patient in addition to oral and intravenous potassium replacement. The dose of given acetazolamide was 2×250 due to the presence of severe metabolic alkalosis. Clinical and laboratory findings were fully restored to normal levels one week following the initiation of treatment.

Keywords: Acute kidney injury, gitelman syndrome, rhabdomyolysis

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INTRODUCTION

Gitelman syndrome (GS) is a renal tubular disease that presents with electrolyte imbalance (1). It is induced by the defect in the thiazide-sensitive Na-Cl cotransporter in distal tubules. It manifests itself with hypokalemia without hypertension, hypomagnesemia, hypercalciuria, and metabolic alkalosis (2). Patients may be commonly referred with muscular weakness, spasm, and cramping and also acute kidney injury (AKI) secondary to rhabdomyolysis in case of long-term severe hypokalemia. Case reports on hypokalemia, rhabdomyolysis, and AKI secondary to GS are limited in the literature. A case followed up with GS diagnosis, referred with muscular weakness and cramping complaints, and determined to have rhabdomyolysis and AKI secondary to hypokalemia has been discussed in the present case report.

CASE PRESENTATION

A 32-year-old male patient was admitted to the emergency service with muscular weakness and cramping for two days. Patient history revealed that he had a diagnosis of GS 12 years previously. Therefore, he was on treatment with potassium citrate which he had discontinued during the previous week, and he was on no other medication. The patient did not smoke cigarettes or drink alcohol. Family history was negative. Physical examination revealed palpation and sensitivity in extremities with no other abnormal examination findings. Vital findings were as follows: fever, 36 °C; blood pressure, 110/60 mmHg; heart rate, 84 bpm; and respiratory rate, 12/min. The laboratory tests of the patient revealed the following: urea, 31 mg/dL; creatinine, 3.09 mg/dL; potassium, 2.14 mEq/L; pH, 7.56; and HCO_3, 59 mmol/L. Patient was cramping and was determined to have 27.610

U/L creatine kinase (CK), 258 IU aspartate aminotransferase, and 240 ng/ml aldolase (reference: 0-10 ng/mL) levels with normal findings from thyroid function tests. Other electrolyte levels including calcium and routine laboratory parameters were determined to be normal. Kidney dimensions and parenchymal echogenicity were determined to be normal without hydronephrosis in the urinary system of the patient as per ultrasound imaging. Renal artery Doppler ultrasound findings were normal. It was learned that kidney function tests of the patient had revealed normal results one month previously. There was no potential cause of dehydration such as nephrotoxic agent intake, contract exposure, diarrhea, and vomiting. Thus, the findings suggested that patient had rhabdomyolysis and AKI secondary to hypokalemia. Therefore, NaCl 100 cc/h isotonic was administered to the patient in addition to oral and intravenous potassium replacement. The dose of given acetazolamide was 2 × 250 due to the presence of severe metabolic alkalosis. In the follow-up one week after the beginning of treatment, test findings were as follows: potassium, 3.42 mEq/L; pH, 7.42; HCO_3, 24.5 mmol/L; CK, 272 U/L; and creatinine, 1.16 mg/dL. Patient's symptoms were fully recovered, so acetazolamide treatment was stopped, potassium citrate treatment was prescribed as 1 × 1, and the patient was discharged with further control to be performed at the outpatient clinic. Written informed consent was obtained from the patient who participated in this study.

DISCUSSION

GS is a rare tubulopathy transmitted as an autosomal recessive trait. It is caused by mutation in the thiazide-sensitive Na-Cl cotransporter-coding gene, *SLC12A3* (3). Patients may be referred with hypokalemia, hypomagnesemia, and metabolic alkalosis-associated muscular weakness and cramping (2). Patients are usually diagnosed with GS during puberty and adulthood.

Rhabdomyolysis is induced by several factors such as muscular trauma, alcohol consumption, infection, enzyme deficiencies, and endocrine disorders. Hypokalemia, which is an electrolyte disorder, is among these reasons (4). Rhabdomyolysis associated with hypokalemia is an expected condition in patients with GS. Literature widely reports hypokalemia and rhabdomyolysis secondary to GS. However, literature review resulted in a number of pediatric cases presented with GS=>hypokalemia=>rhabdomyolysis=>AKI. Kumagai et al. (5) reported that a 13-year-old girl was referred with muscular weakness and cramping. Further examination led to the establishment of diagnosis of GS and hypokalemia=>rhabdomyolysis=>AKI secondary to GS. Patient did not have severe metabolic alkalosis and was administered oral and intravenous potassium and treatment with hydration. Laboratory and clinical findings of the patient were restored to normal levels on the 10th day of treatment. In contrast to the above-mentioned case, our case had already been diagnosed with GS 12 years prior to his referral, and rhabdomyolysis and AKI had developed secondary to severe hypokalemia induced by discontinuing the potassium treatment he was on. While the case reported by Kumagai et al. (5) is in the pediatric age group, our case is in the adult age group. Therefore, our case is the first case to be reported in the adult age group.

It has been demonstrated that the level of CK is correlated to the risk of AKI development in rhabdomyolysis cases. Talving et al. determined in their study that >3000 IU/L CK level is an independent risk factor for the development of AKI (6). Rodríguez et al. determined in their study that >5000 IU/L CK level is associated with AKI (7). CK level was determined as 27.610 U/L in the present study, and it is significant with respect to AKI in accordance with the mentioned studies.

CONCLUSION

Consequently, it should be remembered that rhabdomyolysis secondary to severe hypokalemia which induces AKI may develop in patients with GS.

Informed Consent: Written informed consent was obtained from the patient who participated in this study.

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