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A Rare Cause of Hypokalemia in the Emergency Department: Gitelman Syndrome

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Abstract

Gitelman's syndrome is an autosomal recessively inherited disease with a wide clinical spectrum usually seen in adolescents and adults. Patients have hypokalemia, metabolic alkalosis, hypomagnesemia and hypocalciuria together with normal blood pressure. Effect of GS on pregnancy is not known well. In this study, we presented a pregnant woman who experienced seizure due to electrolyte imbalance and diagnosed with Gitelman's syndrome.

Introduction

Gitelman Syndrome (GS) is an autosomal recessively inherited disease with a wide clinical spectrum, usually seen in adolescents and adults. It is reported that function loss develops in the sodium chloride (NaCl) cotransporter system in the distal renal tubule as the result of SLC12A3 gene mutation [1]. Patients have hypokalemia, metabolic alkalosis, hypomagnesemia, and hypocalciuria together with normal blood pressure. Serum calcium (Ca), Phosphorous (P), Parathormone (PTH), and vitamin D3 levels are normal despite hypocalciuria [2]. Urinary NaCl loss and thereby hyperreninemia and hyperaldosteronism are seen [2]. Most of the patients are clinically asymptomatic, but some patients experience seizures, muscle weakness, cramps, episodic tetany, and paresthesia, and these complaints are reported to affect delivery in pregnant women [3,4].

The diagnosis is usually made based on clinical features and laboratory and renal function tests in adulthood as the disease continues with recurrent episodes. Treatment is correction of electrolyte imbalance [5].

In this report, we present a 38-year-old pregnant woman who experienced seizure due to electrolyte imbalance and was subsequently diagnosed with GS.

Case

The pregnant patient with gestational age of 19 weeks was admitted to the emergency room with the complaint of seizure. On her physical examination, her general condition was moderate and she demonstrated confusion. Her abdominal examination revealed that her uterine dimensions were consistent with approximately the 20th week of gestation. Her blood pressure was 110/70 mmHg, heart rate 84 bpm, and respiratory rate 20/min. Other system findings were normal. She had used carbamazepine for epilepsy for 15 years and did not have any history of diuretic or laxative drug use. She had no complaints like vomiting or nausea. This was the patient's 8th pregnancy: the 7 previous pregnancies resulted in 4 live births and 3 intrauterine losses. On obstetric ultrasonography, a live pregnancy of 19 weeks' gestation was detected. Electrocardiography revealed normal sinus rhythm. Laboratory test results are presented in table 1. Intravenous Potassium Chloride (KCl) replacement therapy was started in the emergency room. No pathologies were detected on radiologic tests performed for differential diagnosis (abdominal ultrasonography, abdominal and cranial magnetic resonance imaging, echocardiography, and renal Doppler ultrasonography). The patient was diagnosed with GS based on these findings. Her general condition improved upon normalization of her electrolyte values. She was discharged with oral therapy (magnesium [Mg] b.i.d., Ca b.i.d., K once a day).

Discussion

Hypokalemia and hypomagnesemia may develop due to many reasons and may result in severe outcomes [6]. Maternal hypokalemia and hypomagnesemia are seen in a vast majority of pregnancies and usually result from gastrointestinal (vomiting, diarrhea, ulcerative colitis, malabsorption syndromes, malnutrition) and renal (diabetes mellitus, ketoacidosis, parenteral fluid treatment, diuretics, fasting, alcoholism, pyelonephritis) disorders. In addition, GS and Bartter Syndrome (BS) are shown among the causes of hypokalemia [7-9]. BS causes hypokalemia, hypocalcemia, hypercalciuria, and hypomagnesemia, and is inherited autosomal recessively, similar to GS. However, in contrast to GS, BS is usually diagnosed in the first year

	Value	Normal range
Potassium	2.1 mmol/L	3.5-5.1
Magnesium	1.24 mg/dl	1.58-2.55
Chlorine	100 mmol/L	98-110
Calcium	8 mg/dl	8.4-10.3
Urine calcium	1.17 mg/dl	6.7-21.3
pH	7.52	7.35-7.45
Plasma renin activity	16 ng/mL/saat	0,2-3,6
Aldosterone	260 pg/mL	20 -240
24-hour urine sodium	100 mmol/gün	30-300
24 hour urine potassium	27,5 mmol/gün	25-125
Serum cortisol	14 mcg/dl	5-25
Parathormone	24,5 pg/mL	11-67
Vitamin D3	20 mcg/L	10-60
ACTH*	30 pg/mL	5-46
Albumin	3.1 g	3.2-5.5
Globulin	3.4 g	2.6-3.9
Phosphorus	4.3 mg/dl	2.7-4.5

*Adrenocorticotropic hormone

Table 1: Laboratory characteristics of case.

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of life and leads to hypocalciuria [9-11]. In the presented case, absence of drug use, any known chronic diseases, diarrhea, and vomiting, and normal serum P, PTH and vitamin D3 levels despite hypocalciuria and normal blood pressure were suggestive of GS.

The effects of GS on pregnancy are not well known [11]. McCarthy et al. [1] reported that GS increased the risk of intrauterine growth retardation, oligohydramnios and abortion together with maternal morbidity, and thus stressed the importance of close follow-up of pregnant women with GS. Jones and Dorrell [12] published a 35-year-old case with GS who produced two normal births after two successive abortions. They suggested that controlling electrolyte levels within desired ranges is difficult in pregnant women with GS, and that electrolyte monitoring is not necessary for obstetric and neonatal outcomes [12]. Daskalakis et al. [7] reported that GS has no negative effects on pregnancy. Basu et al. [13] described a GS case in whom maintenance of physiologic K and Mg levels was difficult, but who nevertheless produced three successive normal pregnancies. Lakhi et al. [11] reported a pregnant GS case that resulted in intrauterine fetal death. In the study of Cruz et al. [4] evaluating GS cases, they reported that 20 of 35 patients had children, and of the 20 patients, 7 required intravenous fluid and electrolyte support, and complications like abortion and early delivery developed. In our study, the patient was gravida 8, and 3 previous pregnancies had resulted in intrauterine fetal loss. This subject was diagnosed with GS in her last pregnancy.

Conclusion

In the presented case, although the exact cause of the intrauterine losses is not fully known, it cannot be claimed that they are not associated with the GS-related electrolyte imbalance. Thus, close monitoring of pregnant women with GS for electrolyte imbalance and electrolyte replacement when needed seem important for both maternal and fetal health. Nevertheless, further comprehensive studies are needed to investigate the effects of GS on pregnancy.

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