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Severe Electrolyte Abnormalities and Rhabdomyolysis in Suspected Gitelman Syndrome (poster)

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Malaz Mohamed [1], Olga Abreu [2], Mohemmed Abbas [3], Shaunique Ferguson [4], Elaina Butt [4], Duc Ly [5], Diya Grover [6], Samer Asous [4], Diksha Pinjani [4]

1. Family Medicine, Mount Sinai Hospital, Chicago, USA 2. Family Medicine, Mount Sinai Hospital, Chicago , USA 3. Family Medicine, Sinai Chicago, chicago , USA 4. Family Medicine, Sinai Chicago, Chicago, USA 5. Medical Student, Ross University School of Medicine, Chicago, USA 6. School of Medicine, Ross University School of Medicine, Chicago, USA

Corresponding author: Mohemmed Abbas, malazmohamed@students.aucmed.edu

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Abstract

Introduction:

Severe hypokalemia and hypomagnesemia can lead to significant neuromuscular and cardiac complications, including rhabdomyolysis and QT interval prolongation. Persistent electrolyte abnormalities should prompt evaluation for underlying renal tubular disorders such as Gitelman syndrome.

Case Description:

A 45-year-old woman with hypertension, chronic hypokalemia, iron deficiency anemia, and alcohol use disorder presented with progressive generalized weakness and myalgias. Laboratory evaluation revealed profound hypokalemia (1.7 mEq/L), hypomagnesemia (1.2 mg/dL), hypocalcemia (6.2 mg/dL), and an elevated creatine kinase (6,549 U/L). Electrocardiogram demonstrated marked QTc prolongation to 670 ms. She was treated with aggressive intravenous and oral electrolyte repletion, isotonic fluids, and continuous telemetry monitoring. Creatine kinase was worsening as electrolytes abnormalities were correcting and it peaked at 9,187 U/L before declining with increased hydration. Electrolytes normalized, and QTc interval improved without arrhythmic events. Although alcohol use likely contributed, the severity and persistence of hypokalemia with concurrent hypomagnesemia raised strong suspicion for Gitelman syndrome. Nephrology recommended further outpatient evaluation to establish a definitive diagnosis and genetic testing.

Discussion:

Severe hypokalemia can precipitate rhabdomyolysis through impaired muscle membrane stability and reduced skeletal muscle perfusion. Concomitant hypomagnesemia exacerbates renal potassium wasting and increases arrhythmic risk, particularly in the setting of marked QT prolongation. Gitelman syndrome, a hereditary renal salt-wasting tubulopathy characterized by hypokalemia and hypomagnesemia, should be considered when electrolyte abnormalities are persistent or disproportionate to the clinical context.

Conclusion:

Profound electrolyte disturbances may unmask an underlying renal tubular disorder rather than reflect isolated nutritional deficiency. Early recognition of suspected Gitelman syndrome is essential to guide long-term management and prevent recurrent neuromuscular and cardiac complications.