



## Gitelman's Syndrome: A Differential Diagnosis of Normocalcemic Tetany

Sir,

Gitelman's syndrome is a renal tubular disorder, characterized by hypokalemia, hypomagnesemia, metabolic alkalosis and hypocalciuria due to mutation in the thiazide-sensitive Na-Cl co-transporter gene.<sup>1</sup> Episodes of muscle weakness, accompanied by abdominal pain and vomiting, are the predominant presentation.<sup>2</sup> Tetany may occur during a febrile illness.<sup>2</sup> We describe a young lady with this syndrome who presented with tetanic spasms and normal serum calcium.

A 31 year old female, mother of a four month old, was admitted with recurrent bouts of painful carpo-pedal spasm over last four days, preceded by tingling and numbness of distal extremities for 15 days. There was no history of weakness in any limb. Two weeks ago, the patient was treated for urinary tract infection with a course of oral antibiotic (Ciprofloxacin 500 mg twice daily). Her past, personal and family history was non-contributory.

On examination, a blood pressure (BP) of 90/58 mm Hg was recorded. Examination of the nervous system revealed presence of carpo-pedal spasm, but no weakness, sensory changes, autonomic features, cranial nerve involvement or cerebellar signs. Routine hemogram, renal and hepatic biochemical profile was normal. Serum electrolytes showed normal serum sodium (138 mEq/L) and calcium (8.3 mg/dL, normal 8.3-10.6 mEq/L), along with hypokalemia (1.8 mEq/L, normal 3.3-5.5 mEq/L) and metabolic alkalosis (pH 7.48, p<sub>a</sub>CO<sub>2</sub> 53 mm Hg). Electrocardiogram revealed presence of ST segment depression in leads V<sub>2</sub>, V<sub>3</sub>, V<sub>4</sub>. Intravenous potassium chloride was infused and serum potassium brought to normal. However, tetanic attacks continued. Serum magnesium was sent and a low value was detected (1.02 mg/dl; normal 2-4 mg/dl). Repeat blood gas analysis revealed persistent alkalosis. In absence of other causes of hypokalemic, hypomagnesemic alkalosis (like vomiting, diarrhea, diuretic/laxative use), the possibility of a renal tubular disorder was strongly considered. Estimation of daily calcium excretion established hypocalciuria (below 90 mg, normal 100-400 mg/ 24 hours). Serum aldosterone level was high (85.30 ng/dL; reference 4-31 ng/dL) and plasma renin activity raised (6.98 ng/ml/hr in supine position with normal sodium diet). Based on the presence of hypokalemia, metabolic alkalosis, low normal BP, hypomagnesemia, hypocalciuria, and elevated renin and aldosterone, a diagnosis of Gitelman's syndrome was made. The patient was treated with oral magnesium sulfate and spironolactone, and her symptoms improved gradually. Screening of her parents and siblings did not reveal any abnormality.

Gitelman's syndrome is a renal tubular disorder characterized by hypokalemia, hypomagnesemia, and hypocalciuria and is often mistaken for Bartter's syndrome (which is due to defect in Na and Cl reabsorption in the thick ascending limb).<sup>13</sup> Age of onset, tetanic manifestations, absence of growth retardation, hypermagnesuria despite hypomagnesemia, and hypocalciuria not improved by furosemide favor the diagnosis of Gitelman's syndrome rather than Bartter's syndrome.<sup>3</sup> Prognosis is generally good.<sup>2</sup>

Following a febrile illness, our patient of previously

asymptomatic Gitelman's syndrome presented with tetany that remained poorly controlled even after adequate potassium replacement. Ultimately hypomagnesemia was documented whose correction led to reversal of tetanic spasms. In contrast to the usual cases of Gitelman's syndrome, the patient did not have any motor weakness and her disease first manifested at 31 years of age.

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## References

1. Sabath E, Meade P, Berkman J, et al. Pathophysiology of functional mutations of the thiazide-sensitive Na-Cl cotransporter in Gitelman disease. *Am J Physiol Renal Physiol* 2004;287:195-203.
2. Schepkens H, Lameire N. Gitelman's syndrome: an overlooked cause of chronic hypokalemia and hypomagnesemia in adults. *Acta Clin Belg* 2001;56:248-54.
3. Zimmermann J, Reincke M, Schramm L, et al. The Gitelman syndrome-a differential diagnosis of Bartter syndrome. *Med Klin (Munich)* 1994;89:640-4.