Acute Psychosis in Two Patients with Bartter Syndrome

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To The Editor:

BARTTER SYNDROME IS AN INHERITED CONDITION affecting NaCl reabsorption in the loop of Henle (Rodriguez-Soriano 1998). Loss of Na⁺ causes volume depletion, increased renin, and aldosterone release, and K⁺ and H⁺ losses leading to hypokalemia, metabolic alkalosis, and hypotension. There are at least five gene variations associated with Bartter syndrome (Rodriguez-Soriano 1998). Neuropsychological sequelae are not known to be associated with this syndrome currently. Here, we describe two cases of adolescent patients with Bartter syndrome and new-onset psychosis.

Case Reports

Case 1

An 18-year-old male with Bartter syndrome, depression, and a previous episode of delusional ideation and grandiosity was psychiatrically admitted after a 1 week history of disorganized thought process, auditory hallucinations, hyperreligious delusions, suspicions, and decreased sleep. On presentation, Na⁺ was 128 mEq/L, K⁺ was 4.7 mEq/L, CO₂ was 15 mEq/L, blood urea nitrogen (BUN) was 25 mg/dL, and creatinine was 1.35 mg/dL. With increases in his olanzapine and initiation of lorazepam, his catatonic and psychotic symptoms improved. His BUN and creatinine normalized. He remained hyponatremic during the entire hospital stay (126–133 mEq/L). He was discharged on day 15 with notably improved mental status. During outpatient psychiatric follow-up, his antipsychotic was tapered because of medication side effects, which resulted in worsening psychiatric symptoms of anxiety and paranoid delusions.

Case 2

A 15-year-old male with Bartter syndrome and no prior psychiatric history presented with hypokalemia (K⁺ 2.6 mEq/L) after medication refusal. After hypokalemia resolution, on hospital day 4, he reported auditory and visual hallucinations, paranoid delusions, and hyperreligiosity. His mental status examination was notable for a guarded attitude, psychomotor retardation mixed with agitation, tangential thought process, and auditory and visual hallucinations. His psychosis resolved with initiation of risperidone. During his hospital stay, his creatinine and Na⁺ remained normal. He was rehospitalized 4 months later for a similar presentation of psychotic

symptoms in the context of being nonadherent to risperidone because of sedation. K⁺ was 3.3 mEq/L on that admission. Risperidone was discontinued and aripiprazole started, which was better tolerated by the patient and improved his psychotic symptoms.

Discussion

Based on our search of the literature, these are some of the first reports of Bartter syndrome associated with psychosis. In both cases, the patients did not undergo testing to reveal the genetic variant resulting in Bartter syndrome. Psychotic symptoms did not specifically correlate with their metabolic or electrolyte disturbances. Classically, Bartter syndrome results from genetic variations in potassium or chloride channel receptors. Each associated receptor is expressed in multiple brain tissues, including the limbic, frontal, and parietal systems (Allen Institute for Brain Science 2014). Psychosis is a neuropsychological phenomena tied to these brain structures (Oyeboode 2008), and abnormal neuronal ion transport (Seeman et al. 2006). In our patients, chronic electrolyte disturbances may underlie shared pathophysiologic mechanisms between these conditions. Further investigation is needed to elucidate potential mechanisms relating these two disorders.

References

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