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Case Report

Adult-Onset Bartter Syndrome Presenting as an Unprovoked Seizure - A Rare case report

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Abstract:

Bartter syndrome is a rare inherited kidney tubule disorder that is often diagnosed in infancy or early childhood. Seizures as the initial presentation are extremely rare, especially in adult-onset cases. A 59-year-old woman who was previously in good health presented with an unprovoked generalised tonic-clonic seizure. A comprehensive assessment found persistent low potassium levels, elevated blood pH, normal blood pressure, low calcium levels, low magnesium levels and excessive calcium in the urine—results characteristic of Bartter syndrome. This case highlights the necessity to take into account uncommon renal tubular disorders when diagnosing unexplained seizures in adults.

Key words: bartter syndrome; adult-onset; hypokalemia; unprovoked seizure; metabolic alkalosis; renal tubular disorder

Introduction

Bartter syndrome is an autosomal recessive disorder that affects the thick ascending limb of the loop of Henle, resulting in impaired reabsorption of sodium, chloride, and potassium. The condition is predominantly identified in youngsters but can occasionally manifest in adults. The classic features comprise hypokalemia, metabolic alkalosis, normotension, hyperreninemia, and hyperaldosteronism, with normal to low blood pressure values. Seizures are uncommon neurological symptoms, typically resulting from severe disturbances in electrolyte levels.

Adults experiencing unprovoked seizures require comprehensive evaluations to rule out structural, metabolic, infectious, and idiopathic factors. Electrolyte disorders, especially hyponatremia or hypocalcemia, are more frequently the cause than hypokalemia. In cases of unexplained hypokalemia accompanied by neurological symptoms, a renal tubular defect should be taken into consideration.

A case report documents a rare manifestation of adult-onset Bartter syndrome presenting as a generalized seizure in a previously healthy 59-year-old woman.

Case Presentation

A 59-year-old woman with no prior medical history was rushed to the emergency department after experiencing a witnessed seizure.Family members reported that the event lasted around five minutes, after which there was a period of post-ictal confusion lasting 15 minutes. Before developing this condition, she had experienced polyuria, polydipsia, and intermittent muscle weakness; there was no prior history of seizures, trauma, recent illness, or medication use. She was a non-smoker, did not consume alcohol and had no family history of epilepsy or renal disease.

Upon examination, the patient was alert but disoriented. Vital signs showed a blood pressure of 90/60 mmHg, a heart rate of 84 beats per minute, a temperature of 36.8 degrees Celsius, and an oxygen saturation of 98% on room air. A normal neurological examination was found after the seizure. Cardiovascular, respiratory, and abdominal examinations revealed no abnormalities.

Biochemical Investigations

Investigation	value	Reference range
Sodium- mmol/L	130	136-145
Potassium- mmol/L	2.1	3.5-5.1
Serum chloride - mmol/L	88	96-106
ABG		
PH	7.5	7.35 -7.45
PO2- mmHg	96	75-100
PCO2- mmHg	43	35-45

Lactate- mmol/L HCO3- mmol/L	0.8 42	0-2 22-26
WBC -10^3/uL	7.3	4.00-11.00
HGB- g/dl	11.3	11.0-15.0
PLT- 10^3 uL	230	150-450
AST- U/L	34	15-37
ALT – U/L	20	12-78
Alkaline Phosphatase- U/L	61	46-116
Blood Urea – mmol/L	5.6	1.8-6.3
Creatinine – ummol/L	98	62-115
C Reactive Protein – mg/L	5	0-5
Magnesium – mmol/L	0.3	0.8-1.1
Calcium – mmol/L	2.01	2.1-2.5
ESR – mm/1st hr	22	
Serum Osmolality-mosm/Kg	271	
Urine osmolality-mOsm/Kg	263	
Urine chloride- mmol/l	101	
Urine Potassium- mmol/l	56	
TTKG	20	

Table 1: Hematological, biochemical, imaging investigations.

NCCT bran - **Normal**
EEG - **Normal**
9 am cortisol - **147(80-510)**
TSH - **0.44**
FT4 - **25.1**
SST – Basal - **36**
30min -**82**
ECG - **hypokalemic changes/U waves**
2D Echo - **normal**
US Abdomen/KUB - **No supra renal masses**
24 hours urinary calcium Level - **350mg/24**
Mantoux - **negative**
AFB sputum - **negative**
Urine creatinine - **1149ummol**
Urine Calcium - **1.08mmol/l**
Urinary Calcium/Creatine - **0.9**
CBS - **98**

These findings indicated renal potassium wasting in the context of normotension and metabolic alkalosis. A provisional diagnosis of Bartter syndrome was made. Genetic testing for mutations in SLC12A1, KCNJ1, and CLCNKB genes was offered but declined due to financial limitations.

Discussion

Bartter syndrome is a group of autosomal recessive renal tubular disorders that lead to impaired salt reabsorption in the thick ascending limb of the loop of Henle. Five subtypes have been classified according to genetic mutations, exhibiting diverse clinical manifestations. Typically, Classic Bartter syndrome presents in early childhood, but milder variations may appear later.

The diagnosis in our patient was difficult to make because there was no history of childhood electrolyte imbalances or growth delays. The initial seizure was a result of profound hypokalemia, a rare but acknowledged complication. Hypokalemia may decrease the seizure threshold by impacting membrane stability and resulting in neuronal hyperexcitability, even though seizures are more often linked with hyponatremia or hypocalcemia.

The differential diagnoses examined were diuretic abuse (ruled out due to negative toxicology results), Gitelman syndrome (characterised by low magnesium and calcium levels in the urine), and concealed vomiting (no prior history and negative urine test results). The combination of normotension, metabolic alkalosis, high urinary potassium and chloride levels, low urinary calcium, and elevated renin and aldosterone levels supported the diagnosis of classic Bartter syndrome.

Bartter syndrome occurring in adults is extremely uncommon, with few documented cases in the literature. The majority of adult diagnoses stem from late manifestations of genetically mild variants or previously undiagnosed childhood cases. Chronic low potassium levels can result in problems like kidney stone formation, muscle weakness, abnormal heart rhythms, and in uncommon instances, seizures.

Management involves potassium supplements, potassium-sparing diuretics such as amiloride, NSAIDs to reduce prostaglandin-mediated renal salt loss, and sufficient fluid intake. Our patient received intravenous potassium chloride, magnesium and calcium then took potassium supplements orally, along with amiloride. The decision was made not to start seizure prevention treatment due to the belief that the event was caused by a metabolic issue.

Follow-Up and Outcome

The patient's serum potassium levels returned to normal within three days of the treatment. Her mental condition completely recovered, and she did not experience any further seizures while in the hospital. A cardiac review revealed no arrhythmias or structural anomalies. An ultrasound of the kidneys revealed no signs of nephrocalcinosis. She was discharged on oral potassium chloride (40 Amiloride 5mg morning dosage and was advised

to have regular follow-up with both nephrology and neurology departments.

At three-month follow-up, she continued to be seizure-free with stable potassium levels ranging from 3.6–3.8 mmol/L. Genetic testing was put off once more, despite genetic counseling being offered. Regular checks on kidney function and electrolyte levels were suggested for the long term.

Conclusion

This case illustrates a rare presentation of adult-onset Bartter syndrome with a generalized seizure due to severe hypokalemia. Although primarily a pediatric condition, clinicians should remain vigilant for atypical adult presentations, especially in patients with unexplained hypokalemia, metabolic alkalosis, and normotension. Prompt recognition and treatment can prevent life-threatening complications, including seizures and cardiac arrhythmias.

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