Shaping Tomorrow's Nephrology: Insight-Driven Kidney Care
KSN 2026 SEOUL, KOREA
The 46th Annual Meeting of the Korean Society of Nephrology
THE KOREAN SOCIETY OF NEPHROLOGY
Jun 11 (Thu) – 14 (Sun), 2026
COEX, Seoul, Korea

Abstract Type : Poster exhibition
Abstract Submission No.: A-0810
Abstract Topic : Fluid, Electrolyte and Acid-base Disorder

A Rare Case of Acquired Bartter-Like Tubulopathy in Systemic Lupus Erythematosus With Secondary Sjogren Syndrome

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Case Study : Objective To describe a rare case of acquired Bartter-like tubulopathy in a patient with systemic lupus erythematosus and secondary Sjögren syndrome, and to highlight the nephrologic clues that support this diagnosis. Methods We reviewed the patient's clinical presentation, serum and urine biochemical profile, acid-base status, medication history, and response to therapy. Alternative causes of hypokalemic metabolic alkalosis, including gastrointestinal chloride loss, diuretic exposure, and drug-induced tubulopathy, were assessed clinically and excluded. Results A middle-aged Filipino woman with longstanding systemic lupus erythematosus and secondary Sjögren syndrome on hydroxychloroquine and prednisone presented with generalized weakness, bipedal edema, poor concentration, polyuria, and constipation. She had no vomiting, diarrhea, or diuretic use. Laboratory tests showed hypokalemia (2.80 mmol/L), hypomagnesemia (1.48 mg/dL), and metabolic alkalosis (pH 7.49, pCO2 35.9 mmHg, HCO3 27.4 mmol/L) in a normotensive setting. Serum osmolality was 290 mOsm/kg and urine osmolality was 547 mOsm/kg. Urine studies showed inappropriate renal losses with urine potassium of 41.21 mmol/L and urine chloride of 99.74 mmol/L. Spot urine calcium-creatinine ratio was elevated at 1.08, favoring a Bartter-like phenotype. The patient received intravenous and oral potassium and magnesium replacement and was started on spironolactone, while maintenance autoimmune therapy was continued. Serial monitoring showed correction of electrolyte abnormalities with marked clinical improvement. Conclusions Acquired Bartter-like tubulopathy should be considered in patients with autoimmune overlap disease who present with unexplained hypokalemic metabolic alkalosis and persistent urinary electrolyte wasting. Early nephrologic recognition allows timely electrolyte replacement, potassium-sparing therapy, and optimization of control of the underlying autoimmune condition.

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Table 1. Key Findings Supporting an Acquired Bartter-Like Tubulopathy

Parameter	Patient Data	Nephrology Interpretation
Clinical setting	Normotensive; no vomiting/diarrhea; no diuretic exposure	Points away from extrarenal Cl− loss / diuretic effect
Serum K+	2.80 mmol/L	Hypokalemia
Serum Mg2+	1.48 mg/dL	Hypomagnesemia (can accompany salt-wasting tubulopathy)
ABG	pH 7.49; pCO_2 35.9 mmHg; HCO_3^- 27.4 mmol/L	Metabolic alkalosis
Serum osmolality	290 mOsm/kg	Normal
Urine osmolality	547 mOsm/kg	Concentrated urine despite symptoms of polyuria
Urine K+	41.21 mmol/L	Renal K+ wasting
Urine Cl−	99.74 mmol/L	High urine Cl− supports renal/diuretic-like alkalosis pattern
Spot urine Ca/Cr	1.08	Hypercalciuria pattern → favors Bartter-like phenotype
Response to therapy	K+/Mg2+ repletion + spironolactone; continued HCQ/prednisone	Electrolytes normalized; symptoms improved (per serial monitoring)

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Figure 1. Diagnostic Flow Diagram