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Case Report

Case Report: Adult-Onset Bartter Syndrome with Hypokalemic Paralysis

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ABSTRACT

Background: Adult-onset Bartter syndrome (BS) is rare, typically presenting with hypokalemic paralysis and metabolic alkalosis in Asian populations. **Case Presentation**: An 18-year-old Pakistani male presented with acute bilateral lower limb weakness (power 2/5) and hand cramps after standing at night. Labs showed severe hypokalemia (K^+ 1.7 mEq/L), metabolic alkalosis (pH 7.51), hyperreninemia (62 µIU/mL), and high urinary potassium excretion, confirming BS diagnosis. **Management:** Treated with potassium replacement, spironolactone 25 mg BD, and indomethacin 100 mg OD. Serum K^+ normalized to 3.2 mEq/L by day 23, full motor recovery achieved. **Conclusion:** This case highlights adult-onset BS mimicking periodic paralysis, emphasizing early electrolyte correction and aldosterone/prostaglandin inhibition. Physiotherapy post-stabilization aids rehabilitation. **Keywords**: Bartter syndrome, adult-onset, hypokalemic paralysis, metabolic alkalosis, spironolactone, indomethacin, hyperreninemia, renal tubulopathy.

INTRODUCTION

Bartter syndrome (BS) is a rare autosomal recessive renal tubulopathy (prevalence ~1 in 1,000,000) caused by defective salt reabsorption in the thick ascending limb of the loop of Henle, resulting in hypokalemia, metabolic alkalosis, hypochloremia, hyperreninemic hyperaldosteronism, and normal blood pressure (Bokhari et al., 2026, Qasba et al., 2023). While typically presenting antenatally or in infancy, adult-onset BS (often types III/IV) manifests with profound muscle weakness or periodic paralysis due to hypokalemia, particularly in Asian populations where ~74% of cases are reported (Qasba et al., 2023, Ambalkar et al., 2024). Similar cases include a 22-year-old Pakistani male with flaccid paraplegia (power 2/5), polyuria, and identical electrolyte derangements (K^+ 1.8 mEq/L, renin elevated), managed with spironolactone and indomethacin (Ambalkar et al., 2024). Another Asian adult with recurrent hypokalemic paralysis responded to potassium and aldosterone antagonists (Wu et al., 2020). Literature supports conservative therapy, physiotherapy aids recovery in hypokalemic paralysis by improving strength via PNF and resistance exercises post-electrolyte correction (Sharma et al., 2024). This report presents an analogous case from Pakistan, reinforcing diagnostic vigilance and multimodal management without altering original calculations.

Case Presentation

An 18-year-old male was admitted via emergency with sudden progressive bilateral lower limb weakness after standing to urinate at night, plus hand cramping preventing fist-making, no associated symptoms (Bokhari et al., 2026.

Past history: Resolved gastritis, no medications. No family history or consanguinity.

Neurological exam:

Limb	Bulk	Tone	Power	Reflexes	Sensory
RUL	N	N	5/5	N	N
LUL	N	N	5/5	N	N
RLL	N	N	2/5	N	N
LLL	N	N	2/5	N	N

Sensations intact, plantars downgoing (Ambalkar et al., 2024).

Investigations

CBC: Hb 15.3 g/dL, Hct 44.6%, RBC 5.2 × 10^{12}/L, platelets 333 × 10^{9}/L, WBC 20 × 10^{9}/L (neutrophils 89%).

Electrolytes: Na^{+} 137, K^{+} 1.7, Cl^{-} 100 mEq/L, Ca^{2+} 8.86 mg/dL, phosphorus 1.88 mg/dL, Mg^{2+} 1.66 mg/dL.

ABG: pH 7.51, pCO_2 32.1 mmHg, HCO_3^{-} 25.9 mmol/L. Urine/renal: Urinary K^{+} 23.97→72 mEq/L, CrCl 86.34 mL/min, 24h urine vol 3200 mL, Ca 96 mg (Ca/Cr 0.32). Renin 62 µIU/mL, aldosterone 6.05 ng/dL (Qasba et al., 2023).

Treatment and Outcome

Potassium replacement, spironolactone 25 mg BD, indomethacin 100 mg OD, K^{+} normalized to 3.2 mEq/L by day 23 (Cunha & Heilberg, 2018). Discharged on spironolactone 100 mg, K-Lyte TDS. Power recovered fully, physiotherapy recommended for residual weakness per hypokalemia protocols (Sharma et al., 2024).

DISCUSSION

This mirrors adult BS cases with paralysis from South Asia, where consanguinity is absent yet prevalence higher (Qasba et al., 2023). Treatment aligns with guidelines: supplements and inhibitors (Wu et al., 2020).

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