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Case Report

Polymyxin B–Induced Bartter-Like Tubulopathy: A Case Report of Severe Potassium and Magnesium Wasting

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ABSTRACT

Bartter syndrome is a rare inherited salt-wasting tubulopathy characterized by hypokalemia, hypomagnesemia, metabolic alkalosis, and hypercalciuria. Although typically genetic, Bartter-like phenotypes can occur secondary to nephrotoxic medications such as polymyxins, which induce tubular dysfunction through oxidative and mitochondrial injury. These acquired presentations are underrecognized in critically ill adults receiving salvage antimicrobial therapy. We report a 77-year-old woman who developed acute electrolyte wasting consistent with a Bartter-like phenotype shortly after initiation of polymyxin B for multidrug-resistant Acinetobacter pneumonia. She developed progressive hypokalemia (2.3–2.9 mmol/L by day 5), hypomagnesemia (1.11 mg/dL by day 6), metabolic alkalosis (bicarbonate 35 mmol/L; chloride 90 mmol/L), and polyuria (4–5.8 L/day). A spot urine sample during hypokalemia (serum potassium 2.6 mmol/L) showed urine potassium 48.9 mmol/L, urine creatinine 31.8 mg/dL, and urine chloride 23 mmol/L, yielding a K/Cr ratio of 154 mmol/g—confirming inappropriate renal potassium wasting. No osmotic diuresis or increased urinary calcium excretion was present. Competing causes, including diuretics, beta-agonists, gastrointestinal losses, aminoglycosides, amphotericin, and refeeding, were absent. Kidney function remained stable (creatinine 0.76–0.98 mg/dL). Despite aggressive electrolyte replacement, abnormalities persisted until polymyxin B was discontinued on day 14, after which potassium and magnesium normalized within 48 hours and polyuria resolved. Polymyxin-associated nephrotoxicity is well described, but Bartter-like electrolyte-wasting phenotypes are less frequently recognized. The earlier-than-typical onset in this patient may reflect advanced age, critical illness, and reduced renal reserve. This case highlights the importance of routine electrolyte surveillance during polymyxin therapy and prompt evaluation for renal wasting when new hypokalemia, hypomagnesemia, or polyuria develops.

1. Introduction

Bartter syndrome refers to a group of rare autosomal recessive salt wasting tubulopathies affecting the thick ascending limb (TAL) of the loop of Henle, characterized by hypokalemia, metabolic alkalosis, and variable disturbances in magnesium handling. Reported prevalence differs across genetic subtypes, reflecting the heterogeneity of the disorder rather than a single uniform incidence [1].

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Bartter-like electrolyte disturbances have also been described in association with nephrotoxic medications, such as antibiotics, including polymyxins, used to treat multidrug-resistant Gram-negative infections such as Pseudomonas aeruginosa, Klebsiella pneumoniae, Enterobacteriaceae, and Acinetobacter baumannii [2, 3]. Polymyxins are internalized by proximal tubular epithelial cells through megalin and PEPT2 mediated endocytosis, leading to oxidative and mitochondrial injury [4, 5]. Although this mechanism primarily injures the proximal tubule, the resulting impairment of sodium and chloride reabsorption, along with activation of tubuloglomerular feedback, can produce downstream functional changes that mimic TAL dysfunction, thereby generating a Bartter-like clinical phenotype.

We describe a 77 year old woman who developed a Bartter-like syndrome shortly after receiving polymyxin B, with complete resolution following discontinuation of the drug.

2. Case Report Methods

Clinical information was obtained through review of the electronic medical record, including physician and nursing documentation,

Table 1: Laboratory test results with corresponding reference ranges

Test	Patient Value	Reference Range (Adults)
Hemoglobin	12.1 g/dL	12.0–16.0 g/dL
WBC	6.7 K/μL	4.0–11.0 K/μL
Platelets	307 K/μL	150–400 K/μL
BUN	26 mg/dL	7–20 mg/dL
Creatinine	0.76 mg/dL	0.6–1.2 mg/dL
Albumin	3.9 g/dL	3.5–5.0 g/dL
Sodium	139 mmol/L	135–145 mmol/L
Potassium	4.1 mmol/L	3.5–5.0 mmol/L
Chloride	105 mmol/L	98–107 mmol/L
Phosphate	4.1 mg/dL	2.5–4.5 mg/dL
Calcium	10.1 mg/dL	8.5–10.5 mg/dL
Magnesium	2.19 mg/dL	1.7–2.2 mg/dL
CO_2	25 mmol/L	20–31 mmol/L
AST	45 U/L	10–40 U/L
ALT	242 U/L	7–56 U/L
Alkaline Phosphatase	357 U/L	44–147 U/L
Procalcitonin	4.89 ng/mL	<0.1 ng/mL
Lactate	1.1 mmol/L	0.5–2.0 mmol/L

BUN, blood urea nitrogen; WBC, white blood cell count; AST, aspartate aminotransferase; ALT, alanine aminotransferase.

laboratory data, imaging studies, and the medication administration record. Electrolyte replacement was quantified by daily potassium(40-160 meq/day) and magnesium(1-3 grams/day) doses administered via intravenous and enteral routes. No diuretics, laxatives, beta agonists, insulin infusions, corticosteroids, amphotericin, aminoglycosides, or nutrition-related factors (including changes in tube feed formulation) were present during the period of interest unless otherwise specified. Institutional review board (IRB) approval was waived for this single-patient case report, and consent was obtained from the patient in accordance with institutional policy.

3. Case Presentation

A 77-year-old female patient with a past medical history of COPD, hypotension, and respiratory failure status post tracheostomy and PEG tube placement was nonverbal but responsive to noxious stimuli. She was sent to the ED from a nursing home after findings on a HIDA scan. Nursing home records indicated elevated liver function tests (LFTs) and a HIDA scan showing cystic duct obstruction.

On initial presentation, the patient was afebrile (temperature 98.2°F), with a heart rate of 83 bpm, blood pressure of 148/75 mmHg, and oxygen saturation of 100% on mechanical ventilation (MV) settings of 18/400/40/5. Physical examination was unremarkable; the abdomen was non-tender and had a PEG tube in place. The initial laboratory values are shown in **(Table 1)**.

Gastroenterology and General Surgery teams evaluated the patient. HIDA Scan images were reviewed, and it suggested chronic cholecystitis versus cystic duct obstruction due to a stone. The next day, the patient became septic with a BP in the 80/50 mm Hg, HR: 108 bpm, and temperature: 95.5°F. Septic workup was sent, and the patient was

Table 2: Daily potassium and magnesium levels along with replacement amounts

Day	Polymyxin B	Serum K (mmol/L)	Serum Mg (mg/dL)	K Replacement (mEq/day)	Mg Replacement (g/day)
1	Y (Start day)	4.1	1.95	-	-
2	Y	4.2	2.00	-	-
3	Y	3.4	1.79	-	-
4	Y	4.2	1.95	-	-
5	Y	2.9	1.50	100	1
6	Y	3.2	1.11	80	2
7	Y	3.0	1.26	80	2
8	Y	2.6	1.23	120	2
9	Y	2.2	0.88	120	3
10	Y	2.8	1.17	120	2
11	Y	1.7	1.46	160	2
12	Y	2.5	1.07	120	2
13	Y	2.6	1.03	120	3
14	Y (final day)	2.3	1.25	120	2
15	N	2.4	1.35	120	2
16	N	3.0	1.46	100	1
17	N	3.5	1.77	40	1
18	N	3.9	1.63	-	1
19	N	4.3	1.78	-	-

K, potassium; Mg, magnesium; Y, yes; N, no; mEq, milliequivalents; g, grams.

started on norepinephrine after receiving 3 liters of IV fluids. Chest X-ray **(Figure 1)** showed bilateral multifocal patchy opacities, left greater than right, in the lower lobe. The infectious disease team was consulted, and the patient was started on broad-spectrum antibiotics with meropenem 1 g IV q8h and vancomycin 750 mg IV q12h.

A few days later, sputum culture grew Acinetobacter and Stenotrophomonas, sensitive to Bactrim, which was added (5 mg/kg IV q8h). Urine culture grew E. coli and Proteus, both of which were sensitive to meropenem. Two days later, due to worsening clinical status, meropenem was changed to ampicillin/sulbactam 3 G IV q6h and polymyxin B (2 million units IV once, then 1 million units IV q12h), patient's weight was 106kg and Polymyxin B dosing was selected according to standard weight based recommendations, and no renal function based adjustment was required as the patient's kidney function remained stable throughout therapy. At that time, potassium was 4.2 mmol/L, and magnesium was 2.0 mg/dL. Five days after initiation of polymyxin, serum potassium decreased to 2.9 mmol/L, and by the sixth day, magnesium had fallen to 1.11 mg/dL, necessitating replacement.

For this patient, drug induced context, "Bartter-like syndrome" was defined as the presence of: Hypokalemia, Hypomagnesemia, Metabolic alkalosis, Polyuria in the absence of osmotic diuresis, and inappropriately high urinary potassium excretion despite hypokalemia. Serial electrolytes during the period of interest showed stable serum sodium (136 – 140 mmol/L) and chloride (98 – 102

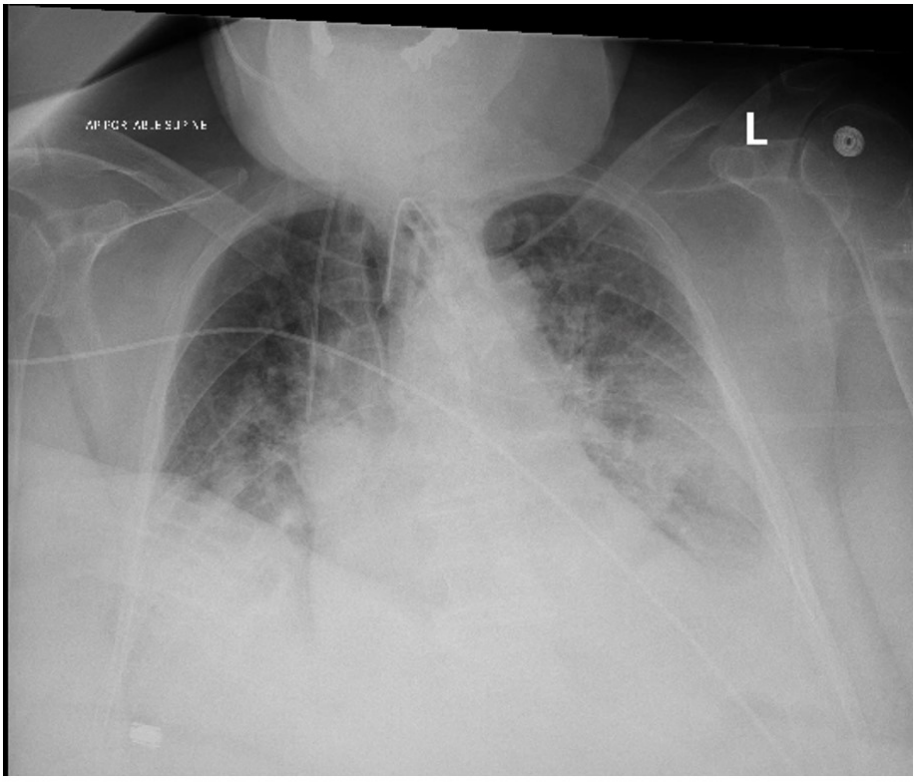

Figure 1: CXR showing bilateral patchy opacities.

mmol/L), but metabolic alkalosis developed, with bicarbonate rising to 35 mmol/L (baseline 22 mmol/L) and serum chloride decreasing to 90 mmol/L. The patient developed polyuria of 3 – 4 mL/kg/h, corresponding to a daily urine output of 4-5.8 L/day. Fluid balance was otherwise neutral, and vasopressor requirements were stable. Serum glucose, urea, and osmolar gap were not elevated, excluding osmotic diuresis. Kidney function remained stable throughout polymyxin B therapy, with creatinine ranging from 0.76 to 0.98 mg/dL (baseline 0.74 mg/dL), and no evidence of acute kidney injury.

This hypokalemia and hypomagnesemia worsened during polymyxin B therapy **(Figs. 2 and 3)**. A spot urine sample obtained during documented hypokalemia (serum potassium 2.6 mmol/L) showed a urine potassium of 48.9 mmol/L, urine creatinine of 31.8 mg/dL, and urine chloride of 23 mmol/L. The calculated urine K/Cr ratio was 154 mmol/g, consistent with inappropriate renal potassium wasting in the setting of metabolic alkalosis. All antibiotics were discontinued upon resolution of sepsis. Urinary potassium significantly decreased after polymyxin B was discontinued on day 14. Baseline serum potassium was 2.3 – 3.0 mmol/L during the course of polymyxin B therapy and rose to 3.9 mmol/L after discontinuation. Daily potassium and magnesium levels, replacement amounts, polymyxin exposure dates, and electrolyte nadirs are summarized in **(Table 2)**.

48 hours after stopping polymyxin B, serum potassium improved significantly, and polyuria resolved. After electrolyte levels returned to baseline, the patient was discharged to a short-term rehabilitation facility with daily potassium and magnesium supplementation.

4. Discussion

Bartter syndrome is classically an inherited renal tubular disorder characterized by defective sodium – chloride reabsorption in the thick ascending limb (TAL), leading to renal salt wasting, hypokalemia, hypomagnesemia, and metabolic alkalosis. In contrast, “Bartter-like syndrome” in adults refers not to a true TAL channelopathy but to a phenocopy of these electrolyte and acid – base abnormalities arising from secondary tubular injury. Several nephrotoxic agents, including aminoglycosides and polymyxins, have been implicated in producing this phenotype through proximal tubular toxicity rather than primary TAL dysfunction [2, 3].

Polymyxin B is filtered at the glomerulus and taken up by proximal tubular epithelial cells via megalin- and PEPT2-mediated endocytosis, triggering oxidative and mitochondrial injury [4]. Although this mechanism primarily affects the proximal tubule, downstream impairment of sodium and chloride handling, activation of tubuloglomerular feedback, and secondary alterations in distal potassium and magnesium transport can collectively mimic the electrolyte pattern of Bartter syndrome. Thus, the term “Bartter-like” in this case reflects a functional resemblance rather than a mechanistic equivalence.

Our patient developed progressive hypokalemia, hypomagnesemia, metabolic alkalosis, and polyuria shortly after initiation of polymyxin B, despite previously stable electrolytes. The early onset on day 5, earlier than the median 10 day onset reported in the literature (range 7 – 18 days) [2], may be explained by several susceptibility factors: advanced age, critical illness with sepsis, baseline hypotension, chronic ventilator dependence, and cumulative exposure to other nephrotoxic stressors (including vasopressors and broad spectrum antibiotics). Reduced renal reserve in older adults may also accelerate tubular accumulation of polymyxin B, precipitating earlier toxicity.

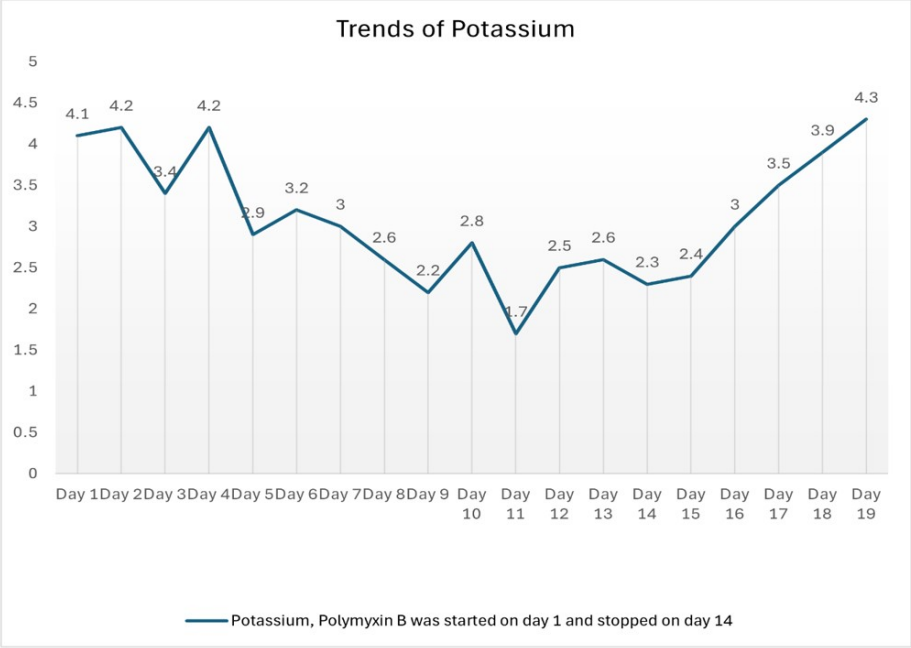

Figure 2: Trends of Potassium.

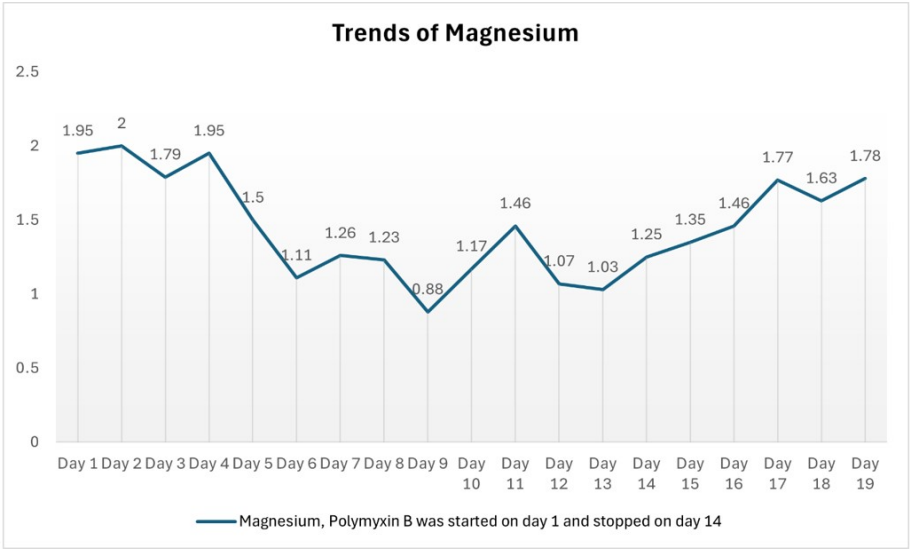

Figure 3: Trends of Magnesium.

A structured adverse drug reaction assessment further supports the assessment of causality. Using the Naranjo algorithm, the event scored 8, consistent with a "probable" adverse drug reaction. Supporting elements included:

- A clear temporal relationship between drug initiation and onset of electrolyte wasting
- Lack of alternative explanations (no diuretics, no osmotic diuresis, no other tubular toxins)
- Objective improvement within 48 hours of drug discontinuation (positive dechallenge)
- No rechallenge, appropriately avoided in this context

This structured approach strengthens the attribution beyond temporal association alone.

Polymyxin-associated nephrotoxicity is reported in 30 – 60% of treated patients, depending on cohort characteristics and the AKI definition used [6]. Importantly, these estimates primarily refer to serum creatinine – based AKI, whereas electrolyte-wasting phenotypes – such as Bartter-like presentations – may occur with or without overt AKI. Our patient maintained stable creatinine throughout therapy, underscoring that tubular electrolyte loss can be an isolated manifestation of polymyxin toxicity.

Differentiating Bartter-like syndrome from other drug induced tubulopathies is essential. Gitelman like features (hypocalciuria,

hypomagnesemia with metabolic alkalosis) and Fanconi type proximal tubular dysfunction (phosphaturia, glycosuria, aminoaciduria, bicarbonate wasting) were not present. Serum phosphate remained stable, there was no glycosuria or proteinuria, and bicarbonate was elevated rather than low, arguing against Fanconi syndrome. Urine chloride was appropriately elevated, supporting a chloride resistant metabolic alkalosis consistent with a Bartter-like pattern.

Across published cases, electrolyte abnormalities typically resolve within days of discontinuing polymyxin therapy [7, 8]. Our patient demonstrated rapid correction of potassium and magnesium levels and resolution of polyuria within 48 hours of drug withdrawal, mirroring prior reports and reinforcing the diagnosis.

Inherited Bartter syndromes typically present in infancy or childhood and may include hypercalciuria and prostaglandin mediated symptoms [9, 10]. In contrast, acquired Bartter-like syndromes emerge abruptly after a clear exposure and resolve with removal of the offending agent, as seen here. Given the rarity of inherited Bartter syndrome, a drug-induced etiology should remain high on the differential in adults with new-onset salt wasting.

5. Conclusion

Bartter-like syndrome with electrolyte wasting can develop rapidly and resolve with timely discontinuation of the drug. Early monitoring of electrolytes, including magnesium, from day 1 of therapy is essential. Development of persistent hypokalemia, hypomagnesemia, metabolic alkalosis, or polyuria should prompt nephrology involvement and consideration of alternative antimicrobials when feasible. Proactive surveillance can prevent complications and support safer antibiotic use.

Conflicts of Interest

The authors declare no competing interests that could have influenced the objectivity or outcome of this research.

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Informed consent

Written informed consent for publication of this case was obtained from the patient.

Large Language Model

None.

Author Contributions

FN contributed to conceptualization, data collection, drafting the original manuscript, review, and preparation of the tables and figures. ZH provided supervision, editing, manuscript review, drafting support, validation of diagnostic reasoning, critical revision, and final approval of the version to be published. AHB contributed to the literature review, manuscript review, and visualization. FA performed a detailed literature review and contributed to the discussion section. MZM contributed to the literature review and writing of the introduction section. AR contributed to manuscript review, table drafting, and editing. SA contributed to editing, reviewing, and literature review. FB contributed to the literature review and drafting of the manuscript. FS contributed to the literature review, manuscript drafting, and editing. MR contributed to drafting the original manuscript and secondary literature review. MA contributed to revision of the draft and secondary literature review.

Data Availability

No new datasets were generated or publicly deposited for this case report. The clinical data used in this report are not publicly available due to patient privacy and confidentiality considerations. De-identified information may be available from the corresponding author upon reasonable request and subject to institutional policies.

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