

Colistin-induced Acquired Bartter-like Syndrome: A Rare Cause of Difficult Weaning

AJ Mahendran¹, Nitesh Gupta², Sumita Agrawal³, Pranav Ish⁴

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Sir,

In the late 20th century, polymyxin group of drugs, especially colistin, reemerged after 4 decades to curb the emerging pandemic of highly resistant bacteria. Although acute kidney injury (AKI), proteinuria, and tubulopathy are all known complications, disorders involving the tubulo-interstitium that lead to electrolyte and acid–base disorders are increasingly recognized.¹ The occurrence of such disturbances in the intensive care unit set up causes undesired complications, deviating from the natural course and expected outcome of the primary disease.

A 65-year-old male patient, known case of hypertension, COPD, and Parkinson's disease, on irregular treatment and follow-up, presented with history of progressive worsening of respiratory distress and productive cough for 1 week. On presentation, he had features suggestive of hypercapnic respiratory failure leading to altered sensorium with leukocytosis suggestive of infective exacerbation of COPD. He was intubated and put on invasive mechanical ventilation. On day 9, he developed ventilator-associated pneumonia, endotracheal aspirate isolating *Acinetobacter baumannii*. Antibiotics were upgraded to cefoperazone/sulbactam (2 g 12 hourly) and colistin (2 million units 8 hourly) based on culture sensitivity. There was significant fall in procalcitonin levels. But spontaneous breath trial (SBT) failed after 30 minutes with worsening respiratory acidosis, desaturation, and hypotension. Evaluation showed hypovolemic hypotension that was fluid responsive, despite persistent polyuria. Investigations revealed he had hypokalemia, hyponatremia, hypocalcemia, and hypomagnesaemia. Excess renal loss of the electrolytes evidenced by high fractional excretion of sodium (FeNa) and transtubular potassium gradient (TTKG). There was concomitant polyuria, despite being hypovolemic. Despite the parenteral correction of the electrolytes, there was recurrence and weaning was difficult. On stopping colistin on day 9, the parameters started to reverse after 3 days and on day 4 of stopping the drug, the patient was extubated. Unfortunately, he developed grade III bed sores during the course of illness, which was infected and cultured *Klebsiella* spp., sensitive to colistin only. In view of worsening general parameters secondary to sepsis with rising procalcitonin levels, he was restarted on colistin again on day 27 of admission. The electrolyte abnormalities and polyuria with hypovolemic hypotension recurred, which persisted until stopping colistin. Despite corrections given repeatedly, there was an episode of supraventricular tachycardia (SVT) which was reverted with adenosine. During the episode, very low level of serum magnesium was documented. Magnesium replacement

^{1,2,4}Department of Pulmonary, Critical Care and Sleep Medicine, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, India

³Department of Pulmonary, Critical Care and Sleep Medicine, Jodhpur, Rajasthan, India

Corresponding Author: Nitesh Gupta, Department of Pulmonary, Critical Care and Sleep Medicine, Vardhaman Mahavir Medical College and Safdarjung Hospital, New Delhi, India, Phone: +91 9873096364, e-mail: niteshgupta2107@gmail.com

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was done, and there was no recurrence. A course of 10 day colistin was given with repeated electrolyte corrections and volume replacements, and the patient was successfully discharged on day 42 after resolution of the abnormalities.

The diagnosis of Bartter syndrome is a diagnosis of exclusion. It is a differential for metabolic alkalosis with hypokalemia and normal blood pressure more commonly seen in surreptitious vomiting and diuretic use.² Both these were not present in our patient. Metabolic alkalosis with hypokalemia is a common occurrence in hyperaldosteronism which invariably is associated with hypertension and normovolemic state. In our case, the patient had hypovolemic hypotension with polyuria, which excluded the possibility.

Reviewing the literature, previous case reports are found which shows similar presentation.^{3–5} The duration of use of the drug appears to have little impact. Nevertheless, in all cases, the disorder was completely reversible. None of the cases had any underlying renal dysfunction.

There is increased and inevitable use of colistin in the ongoing battle against drug-resistant bacteria. As the patient stay increases in the hospital, there is increasing contributing factors for difficult weaning. Apart from the primary disease process and comorbidities, polypharmacy and antibiotic-induced adverse reactions contribute significantly as evidenced by hypokalemia due to Bartter-like syndrome leading to difficult weaning in our case. A high index of suspicion for Bartter-like syndrome while using colistin can help in early diagnosis and appropriate treatment decisions.

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