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Case Report

Reversible neurotoxicity associated with polymyxin B: a case report

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ABSTRACT

Polymyxin B is a potent antibiotic used against multidrug-resistant Gram-negative infections such as Acinetobacter baumannii and Pseudomonas aeruginosa. It acts by disrupting the bacterial outer membrane, leading to cell death. Despite its effectiveness, its use is limited by a narrow safety margin and notable toxicities. The most common adverse effects are kidney injury and neurotoxicity, while symptoms such as fever, rash, headache, or chest tightness occur less often. Neurotoxic effects can range from mild paresthesia to severe neuromuscular impairment. This case describes a 60-year-old patient who developed perioral numbness, chest tightness, and headache shortly after receiving polymyxin B, highlighting the need for close monitoring even at therapeutic doses. This case is distinctive because the constellation of perioral numbness, chest tightness, and headache occurred in the absence of renal dysfunction, and symptoms resolved promptly with discontinuation of polymyxin B and supportive diuretic therapy. Such a presentation has not been widely described in prior reports, underscoring the need for vigilance even when dosing is within therapeutic ranges. Polymyxin B remains an important therapeutic agent for multidrug-resistant Gram-negative infections; however, its neurotoxic potential necessitates close observation during treatment. In our case, the patient developed transient neurological symptoms that improved following discontinuation of polymyxin B and supportive management with diuretics, including furosemide (Lasix). This highlights the importance of early recognition and prompt intervention to prevent serious toxicity while maintaining therapeutic efficacy.

Keywords: Polymyxin B, Neurotoxicity, Adverse drug reaction, Multidrug-resistant Gram-negative bacteria, Reversible neurological symptoms

INTRODUCTION

Polymyxin B is a well-established antimicrobial agent effective against Gram-negative bacilli, including *Acinetobacter baumannii* and *Pseudomonas aeruginosa*.[1] Among the five identified polymyxins—A, B, C, D, and E—only polymyxins B and E are currently approved for therapeutic use. Notably, polymyxin B exhibits stronger antibacterial potency compared to polymyxin E.[2]

As a cationic lipopeptide antibiotic, polymyxin B acts by binding to lipopolysaccharides on the bacterial outer membrane, leading to membrane disruption, increased permeability, and subsequent cell death.[3] Despite its potent bactericidal properties, its clinical use is limited by a narrow therapeutic margin and significant toxicity risks.

Common adverse effects of polymyxin B sulfate include nephrotoxicity and neurotoxicity; less frequently observed events include fever, eosinophilia, rash, headache, and chest tightness.[4] Neurotoxic manifestations are often dose-related and may arise from interference with neuromuscular transmission. In this context, our case report describes an uncommon instance of polymyxin B-induced adverse reaction, wherein a 60-year-old patient developed perioral numbness, chest tightness, and headache shortly after administration—emphasizing the need for close monitoring even at therapeutic doses.

CASE REPORT

A 60-year-old male presented to the emergency department with progressive shortness of breath for two days. He had a known history of chronic obstructive pulmonary disease (COPD), diagnosed three years earlier. On evaluation, he was found to have an acute exacerbation of COPD and was admitted for further management. His oxygen saturation was 86%, which was corrected using a nasal cannula delivering 4 l/min of oxygen. Nebulized salbutamol (short-acting β2-agonist) and intravenous methylprednisolone were initiated. Sputum culture and sensitivity revealed growth of multidrug-resistant Gram-negative organisms, including *Pseudomonas aeruginosa*, *Acinetobacter baumannii*, and *Klebsiella pneumoniae*. Based on the sensitivity profile, polymyxin B was identified as the effective agent. Although the patient's condition stabilized following control of the exacerbation, targeted antimicrobial therapy was commenced to eradicate the infection. In this 80-kg adult, intravenous polymyxin B was administered at a dose of 600,000 to 1,000,000 units every 12 hours (total daily dose 1.2–2.0 million units), guided by culture sensitivity results confirming multidrug-resistant Gram-negative pathogens.

One day following initiation of polymyxin B therapy, the patient developed an acute allergic reaction characterized by chest tightness, perioral numbness, and headache. These findings were consistent with an adverse drug reaction in response to polymyxin. Alternative explanations such as electrolyte imbalance, metabolic derangements, cerebrovascular events, or concomitant neurotoxic medications were excluded based on laboratory values, clinical examination, and medication history, strengthening the likelihood of polymyxin B as the causative agent. Using the Naranjo adverse drug reaction probability scale, the event scored 9, consistent with a definite adverse drug reaction. This strengthens the causal association between polymyxin B and the observed neurotoxic manifestations. The following criteria and scores contributed to the total: temporal relationship (+2), improvement on drug withdrawal (dechallenge) (+1), reappearance of symptoms on re-exposure (rechallenge) (+2), no alternative causes identified (+2), previous conclusive reports of similar reactions (+1), and objective evidence supporting the reaction (+1). Total score was 9 (definite adverse drug reaction).

Polymyxin B was discontinued, and intravenous furosemide (Lasix) was administered, which alleviated the sudden adverse reaction. The episode was subsequently diagnosed as a polymyxin B–induced allergic drug reaction. Based on the extended sensitivity profile, intravenous tigecycline was initiated as an alternative agent, providing coverage against *Acinetobacter baumannii* and *Klebsiella pneumoniae*. For multidrug-resistant *Pseudomonas aeruginosa*, intravenous cefiderocol was added according to susceptibility testing. The patient was closely monitored, and clinical improvement was noted with stabilization of respiratory status and resolution of infectious symptoms following seven days of admission, after which he was discharged in a stable condition.

The flow chart illustrates the timeline and sequence of events observed in the case report (Figure 1).

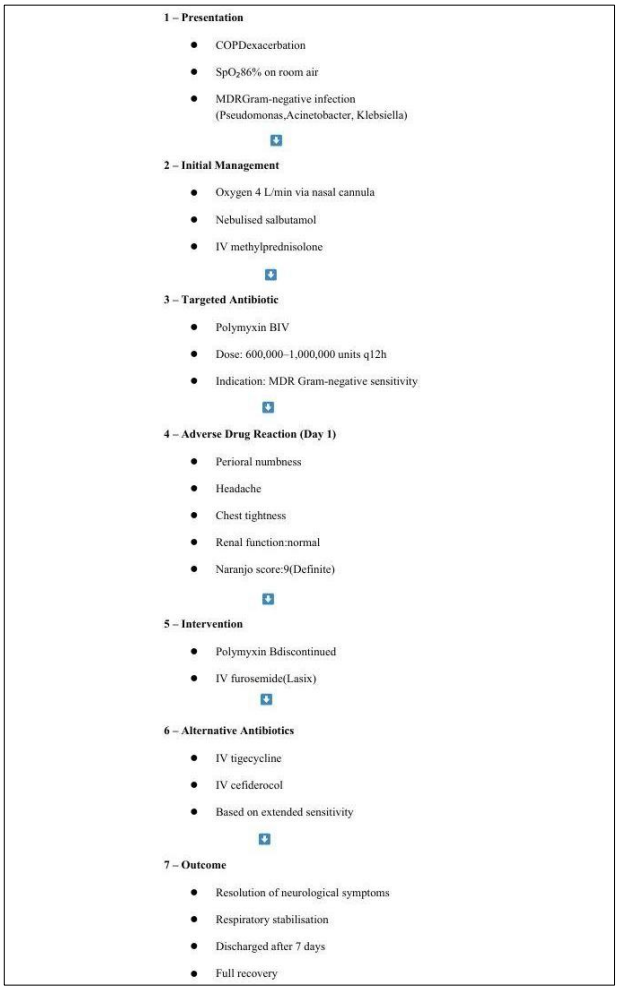

Figure 1: Timeline showing the sequence of clinical events, interventions, and outcome.

DISCUSSION

Polymyxin B has re-emerged as a critical treatment option against multidrug-resistant (MDR) Gram-negative bacteria such as *Acinetobacter baumannii* and *Pseudomonas aeruginosa*, particularly in settings where carbapenem resistance limits alternatives.[2] Despite its renewed clinical use, polymyxin B carries substantial risks of nephrotoxicity and neurotoxicity, both of which are dose-dependent and influenced by patient factors such as age, renal function, and concurrent nephrotoxic or neurotoxic drugs.[5]

Neurotoxicity associated with polymyxin B is less common than nephrotoxicity but has been well documented in clinical and experimental settings. Reported manifestations include dizziness, perioral or

limb paresthesia, headache, ataxia, visual disturbances, and in severe cases, neuromuscular blockade or apnea.[6] The mechanism is believed to involve interference with acetylcholine release at the neuromuscular junction and increased neuronal membrane permeability to cations.[7] These effects are usually reversible upon discontinuation of the drug and supportive management.

In our case, the patient developed perioral numbness, headache, and chest tightness, one day following administration of polymyxin B, without evidence of renal impairment. Symptom resolution occurred after discontinuation of polymyxin B and supportive therapy with furosemide (Lasix). The use of loop diuretics may aid in drug clearance by enhancing urinary excretion and reducing tissue accumulation, although evidence remains limited.[8] This case expands the clinical spectrum of polymyxin B neurotoxicity by documenting a rare triad of symptoms in a patient without renal impairment.

Previous studies have reported similar transient neurotoxic reactions, often arising within hours to days of treatment initiation and resolving spontaneously or with dose reduction.[9] Given the narrow therapeutic index of polymyxin B, such adverse effects highlight the importance of individualized dosing, therapeutic drug monitoring, and avoidance of concomitant neurotoxic medications.

CONCLUSION

This case emphasizes the need for clinicians to recognize early neurological symptoms, even mild manifestations such as perioral numbness or headache, as potential indicators of polymyxin B-related neurotoxicity. Prompt identification and discontinuation of the offending agent remain essential in management.

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