JOURNAL OF NEPALESE SOCIETY OF CRITICAL CARE MEDICINE
AN OPEN ACCESS, PEER REVIEWED, OFFICIAL JOURNAL OF NSCCM

CASE REPORT

Intravesical Colistin Bladder Irrigation for Multidrug-Resistant Urinary Tract Infections in Critically Ill Patients with Renal Compromise: A Single-Center Case Series from Nepal

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HOW TO CITE THIS ARTICLE IN VANCOUVER STYLE?

Poudel S, Khadka B, Khanal K, Ghimire A, Regmi A. Intravesical Colistin Bladder Irrigation for Multidrug- Resistant Urinary Tract Infections in Critically Ill Patients with Renal Compromise: A Single-Center Case Series from Nepal. Journal of Nepalese Society of Critical Care Medicine. 2026 Jan;4(1):36-42.

Submitted	:	July 29, 2025
Accepted	:	November 28, 2025
Published Online	:	January 8, 2026
Declarations	:	None
Conflicts of Interest	:	None

ABSTRACT

Multidrug-resistant (MDR) gram-negative infections are increasing, posing a challenge in critically ill patients with renal impairment. Intravesical colistin irrigation offers targeted therapy while minimizing systemic toxicity. We present three critically ill patients with renal compromise and MDR urinary tract infections (UTIs) caused by *Klebsiella pneumoniae*, *Acinetobacter spp.*, and *Pseudomonas aeruginosa*. All patients received intravesical colistimethate sodium (1 million units in 100 ml saline, 8-hourly for 7 days). Clinical resolution occurred within 48 hours, with negative urine cultures post-treatment and no adverse effects. Intravesical colistin irrigation serves as a valuable treatment alternative for managing MDR UTIs in renally compromised patients, minimizing systemic toxicity. Further studies are needed to optimize dosing and assess long-term safety.

Keywords: *Colistimethate sodium, intravesical therapy, urinary tract infection.*

INTRODUCTION

Multidrug-resistant (MDR) gram-negative pathogens, such as *Klebsiella pneumoniae, Acinetobacter baumannii,* and *Pseudomonas aeruginosa*, have emerged as critical threats in intensive care units (ICUs), contributing to infections such as bacteremia, ventilator-associated pneumonia, intra-abdominal infections, and urinary tract infections (UTIs).[1] More than 61% of critically ill patients worldwide have gram-negative sepsis, and the wrong use of broad-spectrum antibiotics is making resistance worse. This is closely related to the spread of MDR microorganisms.[2,3]

The therapeutic options for MDR gram-negative infections remain limited, especially in patients with renal impairment. Polymyxins, an older class of antibiotics previously discontinued because of their kidney and nerve toxicities, are now being reinstituted as an important therapy against MDR bacteria. However, choosing an appropriate antibiotic remains difficult in patients who are immunocompromised or with impaired renal function. However, systemic polymyxins administration poses significant nephrotoxicity, which makes challenging to renal-impaired patients, so the selection of the drug, its dose, and through route of administration is vital.[4-6]

Colistin (polymyxin E) and polymyxin B are cationic polypeptide antibiotics effective against many Gram-negative bacteria including Enterobacterales, *Pseudomonas aeruginosa*, and *Acinetobacter spp*., but not against Proteus, Serratia, or *Burkholderia spp*. They are also intrinsically resistant to Providencia, Morganella, and Neisseria.[7-9] Both polymyxins disrupt the outer membranes of bacterial by binding to lipopolysaccharide, but they differ markedly in pharmacokinetics. Polymyxin B is administered as the active form and has negligible renal excretion, whereas colistimethate sodium (CMS) is a prodrug that undergoes hydrolysis to convert into active colistin. In normal renal function, about 60–70% of CMS is excreted unchanged, while in acute kidney injury (AKI) or chronic kidney disease (CKD), non-renal clearance becomes predominant, leading to increased conversion to active colistin, causing nephrotoxicity in 40–60% of patients.[10-12] This pharmacokinetic alteration makes systemic colistin particularly risky in renally impaired critically ill patients.

Therapeutic options for MDR gram-negative infections are limited in patients with AKI or CKD, as systemic antibiotics often fail due to either intrinsic/ or extrinsic antibiotic resistance or inability to achieve therapeutic urinary concentrations without causing further renal injury.[13] Intravesical administration of CMS offers an effective alternative by achieving high urinary concentrations (>1000 mg/L) with minimal systemic absorption (<1%), avoiding nephron-or neurotoxicity.[14-16] Several case series and reports have demonstrated rapid eradication of MDR pathogens like *Acinetobacter baumannii*, carbapenem-resistant *Klebsiella pneumoniae*, and *Pseudomonas aeruginosa*, even when parenteral therapy fails.[14,17,18] We prefer using CMS rather than polymyxin B because of its water solubility, non-irritating properties, and sterile availability.

Patient with CKD are especially vulnerable to complicated UTIs because of immune dysfunction, urinary stasis, frequent catheterization, and impaired drug clearance.[19,20] Traditional systemic antibiotics frequently fail in this population due to resistance or dose-limiting toxicity. Despite growing international experience with intravesical colistin, data from low- and middle-income settings with high MDR burden remain scarce.

This is the first reported case series from Nepal describing the successful use of intravesical colistimethate sodium irrigation for MDR UTIs in critically ill patients with renal compromise. This single-center case series involved three patients with CKD received intravesical colistin irrigation as a treatment for MDR UTIs. The diagnosis of UTIs was considered in the presence of ≥12 pus cells/high-power field (hpf), and trace nitrite levels.[21] Patients with suspected UTIs but negative urine cultures were excluded from the study.

The intravesical colistin protocol was standardized. All patients received intravesical colistimethate sodium (1 million units dissolved in 100ml 0.9% normal saline) and administered via a two-way Foley catheter, clamped for 90 minutes, administered every eight-hours intervals for seven days. Systemic antibiotics were physician-dependent and adjusted for renal function. Antimicrobial identification and susceptibility testing, including for polymyxins, were performed using broth microdilution according to Clinical and Laboratory Standards Institute (CLSI) guidelines.

Regarding the dosing regimen, the systemic doses of CMS typically range from 6-9 million international units (MIU), but with intravesical administration, optimal bladder concentrations can likely be achieved with lower doses than those used systemically. In aqueous environments, CMS hydrolyzes to colistin, enabling local bactericidal activity. The selection of 1 MIU every 8 hours was based on prior literature demonstrating efficacy with similar or lower doses.[14,17] We opted for 1 MIU to ensure robust local exposure in critically ill patients with potentially variable bladder volumes and urine pH, which could influence conversion rates, while avoiding excessive dosing that might increase risks of irritation or systemic absorption. Although CMS pharmacokinetics exhibit interpatient variability in systemic administration, intravesical use limits systemic exposure, reducing the impact of such variability on efficacy. The fixed 7-day course of treatment was empirically chosen based on successful eradication in previous case reports confirming culture negativity and clinical resolution, with patient monitoring confirming sterility by day 7.[14,17,18] Further pharmacokinetic studies on intravesical CMS conversion and optimal dosing are warranted. Patient demographics and clinical characteristics are summarized in Table 1.

Table 1. Demographic and clinical characteristics of patients treated with intravesical colistin for multidrug-resistant urinary tract infections.

Parameters	Case 1	Case 2	Case 3
Age (Years)	81	40	35
Sex	Female	Male	Male
Creatinine (mg/dL)	2.3	6.6	2.4
Culture Source	Urine	Urine	Urine
Organism isolated	*Klebsiella pneumonia*	*Acinetobacter spp.*	*Pseudomonas aeruginosa*
Diagnosis	CKD, Urosepsis	CKD, Urosepsis	CKD, Urosepsis, Polytrauma (RTA)
Sensitivity to Polymyxins	Sensitive	Sensitive	Sensitive
Antibiotics used	Piperacillin/Tazobactam, Meropenem, Intravesical Colistin	Imipenem/Cilastatin, Intravesical Colistin	Meropenem, Tigecycline, Intravesical Colistin
Treatment Duration	7 days completed No growth	7 days completed No growth	7 days completed No growth
Outcome	Resolution, no relapse	Resolution, no adverse effect	Resolution, no adverse effect

CASE 1

An 81-year-old female presented to our emergency room with oliguria, fever (102°F), hypoxia, altered sensorium, and a Glasgow Coma Scale (GCS) score of 5/15. She had recurrent UTIs over the past four months. Her medical history included type 2 diabetes mellitus (DM) managed with glargine insulin and chronic kidney disease requiring maintenance hemodialysis. She was transferred to the ICU for mechanical ventilation support and hemodynamic monitoring.

This UTI was classified as healthcare-associated, given her recent exposure to healthcare settings through maintenance hemodialysis sessions and history of recurrent UTIs. Risk factors for UTI in this patient included advanced age (>65 years), female gender, type 2 diabetes mellitus, chronic kidney disease with impaired immune function and drug clearance, recurrent UTIs, and frequent catheterization (a Foley catheter was in place for urinary management and hemodialysis-related procedures). Risk factors for multidrug-resistant organism (MDRO) infection included prior antibiotic exposure for her recurrent UTIs in the past four months (including broad-spectrum agents), prolonged healthcare contact for hemodialysis, and immunosuppression due to diabetes and CKD.

Empiric broad-spectrum treatment with renal-adjusted intravenous piperacillin/tazobactam was initiated. Laboratory analysis showed an elevated total leucocyte count (TLC) of 22,000/mm^3, procalcitonin of 7.2 ng/ml, C-reactive protein (CRP) of 67 mg/dl, creatinine of 2.3 mg/dl, and blood urea nitrogen (BUN) of 102 mg/dl. A chest X-ray did not reveal any abnormalities or signs of infection. Cultures were obtained from blood, sputum, and urine.

After 48 hours in the ICU, the patient's fever did not subside, and urinalysis revealed leukocytosis (16-18/hpf) with trace nitrie. The antibiotic regimen was modified to carbapenem (meropenem) at a renal-appropriate dose. Urine culture revealed MDR *Klebsiella pneumoniae*, susceptible only to Polymyxins (polymyxin-B and colistin). Blood and sputum cultures were negative.

Considering the patient's compromised renal function (creatinine: 2.1 mg/dL, BUN: 110 mg/dl), we opted for intravesical administration of CMS instead of the parenteral route to minimize systemic toxicity. Intravenous meropenem was continued alongside intravesical colistin to provide ongoing systemic coverage given the initial presentation suggestive of sepsis (high grade fever, oliguria, and altered sensorium). Within 48 hours of commencing intravesical colistin irrigation, the patient's fever subsided, and urinary symptoms resolved. No adverse effects were reported. Urine cultures performed on the 7th day revealed no bacterial growth. The patient was successfully liberated from mechanical ventilation after symptoms resolve and was later transferred out of the ICU to a general ward.

CASE 2

A 40-year-old male with CKD on hemodialysis presented with fever, chills, hypotension (80/60mmHg), tachycardia, and tachypnea at the Emergency room. The urinary tract infection (UTI) was present at the time of initial presentation, as evidenced by urinalysis showing leukocytosis (14-18 leukocytes per high-power field) and trace nitrite, along with subsequent urine culture positive for MDR Acinetobacter species. The patient's symptoms were consistent with urosepsis secondary to the UTI. Risk factors for UTI included underlying CKD on maintenance hemodialysis, which increases susceptibility due to immune dysfunction, impaired drug clearance, and frequent invasive procedures such as catheterization. An indwelling Foley catheter was in place for urine output monitoring, a common practice in such patients. Additional risk factors for MDR organism infection encompass repeated healthcare interactions through dialysis sessions, potential exposure to contaminated environments or equipment, and ICU admission with empiric broad-spectrum antibiotics.

He was then transferred to the ICU for inotropic and ventilatory support. Laboratory findings included elevated TLC of 18,290/mm^3, lactate of 5.8 mmol/l, creatinine of 6.6 mg/dl, BUN of 188 mg/dl, elevated liver enzymes (AST: 202, ALT: 104), and procalcitonin of 2.2 ng/ml. Urinalysis showed leukocytes (14-18/hpf) and trace nitrite. Chest X-ray was normal. Blood, urine, and sputum cultures were obtained and sent for microbiologic evaluation.

Empiric treatment with renal-adjusted broad-spectrum antibiotics (imipenem/cilastatin) was started. Hemodialysis was continued and intensified due to worsening renal function (creatinine: 7.3mg/dl, BUN: 245 mg/dl). MDR Acinetobacter species was isolated from the urine culture, susceptible to colistin (polymyxin-E) and polymyxin-B. Blood and sputum cultures were negative.

Intravesical colistimethate sodium administration was initiated for seven days. The fever subsided, and urinary symptoms disappeared. Urine cultures taken on the seventh day revealed no bacterial growth, and no adverse effects reported.

CASE 3

A 35-year-old male patient was brought in intubated to our center following a road traffic accident (RTA) with pan-facial fractures. At the time of presentation, the patient was hypotensive (70/50 mmHg), and tachycardic (120 bpm). The patient had a history of chronic kidney disease, requiring hemodialysis. The patient was referred to our hospital after being treated for 3 days in another hospital. A Foley catheter was present upon admission from an outside hospital.

Treatment was initiated promptly with IV fluid, inotropic support (nor-adrenaline), intravenous antibiotics (meropenem), intravenous analgesics, and proton pump inhibitors for stress ulcer prophylaxis. Bilateral chest tubes were placed for hemopneumothorax, and the patient was transferred to the intensive care unit for close hemodynamic monitoring and ventilator support.

Laboratory examination showed TLC of 7,370/mm^3, hemoglobin of 6.0 g/dl, creatinine of 2.4 mg/dl, BUN of 106 mg/dl, international normalized ratio (INR) of 1.64, elevated liver enzymes (AST: 471, ALT: 109), and CPK total of 9,318 U/L. Chest X-ray showed a collapse of the right upper lobe of the lung.

On the second day, the patient developed a fever, TLC increased to 12590/mm^3, and procalcitonin was 5.02 ng/ml. His urine analysis revealed leukocytosis (16-18/hpf), trace nitrite, and turbid urine. Urine culture was sent, and Foley's catheter was changed. MDR Pseudomonas aeruginosa was isolated in the urine culture and Acinetobacter spp. from both the bronchoalveolar lavage and endotracheal aspirate, both susceptible to Polymyxins (polymyxin-B and colistin) and Tigecycline. Blood cultures returned negative, and the patient's antibiotic therapy was upgraded to intravenous Tigecycline and intravesical Colistin.

The risk factors for MDRO UTI are preexisting CKD, immune dysfunction, impaired drug clearance. Additional risk factors include trauma-related catheterization, ICU admission, mechanical ventilation, and hemodynamic instability, which collectively increase susceptibility to nosocomial pathogens like *Pseudomonas aeruginosa*. Although uncommon in community-acquired UTIs, *P. aeruginosa* is a frequent cause of hospital-acquired UTIs in catheterized, hospitalized patients, particularly those with comorbidities such as CKD. The presence of pyuria, turbid urine, and systemic signs of infection (fever, leukocytosis, elevated procalcitonin), coupled with clinical resolution following targeted intravesical therapy, supports the diagnosis of a true UTI rather than mere colonization, in line with IDSA guidelines for catheter-associated UTI (CAUTI) diagnosis in critically ill patients. Concurrent isolation of Acinetobacter spp. from respiratory samples indicated a separate, likely ventilator-associated pulmonary infection, managed with systemic tigecycline. Negative blood cultures suggest no bacteremia linking the sites, indicating coexisting but distinct infections in the urinary and respiratory tracts.

Considering the patient's compromised renal function, CMS intravesical was initiated and continued for seven days, which improved the patient's condition clinically. Urine cultures performed on the 7th day revealed no bacterial growth, and no adverse effects reported.

DISCUSSION

Urinary tract infections (UTIs) remain a global health burden. They affect approximately 150 million patients annually.[19] Various factors, such as age, diabetes mellitus, genetic predisposition, and sexual activity, contribute to the risk of

UTIs. In this particular patient group, urosepsis due to UTI may result in high mortality rates of 25% to 60%.[20] CKD itself heightens UTI susceptibility, ranging from mild cystitis to life-threatening infections and multiorgan failure. About a quarter of adult sepsis cases post-UTI are classified as urosepsis.[15]

Gram-negative rods predominate as causative agents, accounting for 75-85% of cases. Gram-positive organisms are less common, at about 15%.[15] *Escherichia coli* is the predominant pathogen causing UTIs, although other bacteria like *Proteus mirabilis, Pseudomonas aeruginosa, Klebsiella pneumoniae, Enterobacter spp.*, Staphylococci, and Enterococci are also implicated.[13,21]

A study conducted in Kenya found that 55.6% of infections caused by MDR gram-negative bacteria were mainly UTIs. The most frequently identified bacteria were *Escherichia coli* (33.3%), *Klebsiella pneumoniae* (31.1%), Pseudomonas species (14.4%), and *A. baumannii* (5.6%).[16]

Diagnosis of UTI in CKD relies on standard clinical and laboratory criteria, including pyuria (≥10 leukocytes/μl), oliguria, and bacterial colony counts. Treatment strategies for UTIs in CKD patients generally follow the same principles as those with normal renal function. However, adjustments are necessary for drugs cleared by the kidney or dialysis membranes to avoid systemic toxicity and nephrotoxicity.[21]

Polymyxins, discovered in 1947, are not in use today due to their neurotoxicity and nephrotoxicity. However, they are making a comeback to combat the resistance posed by MDR gram-negative bacteria. These antibiotics disrupt bacterial cell membranes by binding to lipopolysaccharides and phospholipids, leading to cell death.[6] Colistin methanesulfonate (CMS) is a prodrug of colistin and is excreted primarily by renal mechanisms through renal tubular secretion. After intravenous administration, CMS shows its direct linear dose-dependent PK properties, and around 60% of the portion is cleared through the kidneys.[11]

Intravesical CMS has shown efficacy against MDR *A. baumannii* in prior studies. Volkow-Fernández et al. (2012) demonstrated that a continuous intravesical administration of CMS at a dosage of 3.5 mg/kg over 7 days effectively eliminated MDR Acinetobacter baumannii.[14] Giua et al. (2014) found that 100,000 IU during a 90-minute irrigations period achieved optimal bladder concentration, with successfully treating three critically ill patients with MDR Acinetobacter (durations: 7 days for two, 2 days for one)[17] Garcés-Jimeno et al. employed continuous bladder instillation with 3 million IU over 7 days.[18] Thus, based on these studies, we used an equivalent colistin dosage for bladder irrigation, which represents an ideal dosage for bladder irrigation.

Historical uses of intravesical antibiotics include aminoglycosides and polymyxins for preventing bacteriuria post-catheterization. In 1962, Martin and Bookrajian delayed bacteriuria onset with continuous neomycin-polymyxin B irrigations.[22] A 1979 study by Pearman halved bacteriuria incidence in neurogenic bladder in 47 in-patients using 150mg kanamycin plus 30mg colistin instillations, though resistance emerged.[23] However, success was limited, and serious concerns were raised regarding the safety of intravesical neomycin, specifically in end-stage renal disease patients.[24] Paterson et al. reduced postoperative UTI risk from 70% to 13.5% with 0.02% chlorhexidine irrigations in women post-gynecologic surgery.[25] A 2018 systematic review by Pietropaolo et al. analyzed intravesical gentamicin, neomycin/polymyxin, neomycin, and colistin. It reported an 88% average infection reduction. The authors deemed it safe and effective for short-term use when conventional treatments fail.[26]

In our cases, intravesical CMS eradicated MDR *A. baumannii*, yielding negative urine cultures by day 7. Symptoms resolved early, aligning with Volkow-Fernández et al. (resolution by day 2, negative culture by day 10) but with faster clearance possibly due to our intermittent protocol for better drug retention.[14] Like Giua et al., our 7-day regimen ensured success across MDR pathogens.[17]

Intravesical colistin is primarily appropriate for lower UTIs, such as catheter-associated cystitis. It achieves high local concentrations in the bladder with minimal systemic absorption due to the low permeability of the urothelial barrier. This route minimizes the risk of nephrotoxicity from systemic administration, making it ideal for patients with renal compromise. However, its applicability to infections involving the upper urinary tracts, such as pyelonephritis, is limited, as intravesical therapy targets the bladder and does not reach effective therapeutic levels in the kidneys or ureters unless vesicoureteral reflux is present.[13] Pharmacokinetic and pharmacodynamic studies specifically evaluating intravesical colistin are extremely limited, with most literature consisting of case reports and small series rather than dedicated PK/PD studies. General principles from studies of intravesical antibiotics suggest negligible systemic bioavailability supporting localized efficacy but underscoring the need for systemic therapy in upper tract involvement.[13,26] Further PK/PD could potentially provide support for minimal absorption in specific clinical scenarios.

CONCLUSION

Intravesical colistimethate sodium effectively eradicates an MDR gram-negative bacteria in critically ill patients admitted to the ICU with UTIs. Using colistimethate sodium through bladder irrigation could be a good choice for certain patients, especially when parenteral administration could be toxic. Further research and clinical trials are warranted to optimize dosing regimens and assess long-term efficacy and safety.

ACKNOWLEDGEMENT

We sincerely thank the patients and the Critical Care Department for their support.

DISCLOSURE

A preprint has previously been published on Authorea (DOI: 10.22541/au.172216973.38607830/v1). It has not been peer-reviewed.[27]

CONSENT

Written informed consent was obtained from all patients or their legal representatives.

FUNDING INFORMATION

The authors received no financial support for the research

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